Fitness variance and sex

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Doctor of Philosophy
University of Edinburgh
2008

Declaration

I declare that this thesis was composed by myself and that the work contained therein is my own, except where explicitly stated otherwise in the text.

(*Philipp L. Wesche*)

Abstract

Interest in sex expression theory has continued and grown since some of the most radical recent findings were made in the 1970s. The most influential such work has been Trivers and Willard's hypothesis of condition-dependent sex allocation. Condition-dependent sex allocation occurs when the two sexes differ in fitness variance, and when fitness depends either on the phenotype or genotype of either or both of the parents, or the habitat of either the offspring or either or both parents.

Here, I present a thorough investigation of the meaning of fitness variance in the context of condition-dependent sex allocation, followed by a comprehensive analysis of a weak maternal effects model of condition-dependent sex allocation. This includes a consideration of the effects of different sex expression strategies on the population genetics and conservation ecology of species.

I focused on three questions: What is the meaning of fitness variance in the context of condition-dependent sex allocation? Under what conditions can condition-dependent sex allocation evolve? How can we detect condition-dependent sex allocation in natural populations, given new findings on its population genetics and the significance of fitness variance?

Findings include the following:

1. Fitness variance is not a useful metric to detect condition-dependent sex allocation.

2. Condition-dependent sex allocation evolves when selection is greater on one sex than the other; this holds true for a wide range of parameter values. It corroborates existing theory.

3. In the first known model including sexual selection and condition-dependent sex allocation, this combination is shown to produce faster adaptation than sexual selection alone.

4. Condition-dependent sex allocation has the potential to increase population longevity, and may additionally spread through clade (or group) selection.

5. Condition-dependent sex allocation can be reliably detected using a combination of allele frequency trajectories through time and cross-sectional

allele frequency data from polymorphic loci; this combination is necessary to avoid false positives from loci under balancing selection.

Acknowledgements

I thank Nick Barton, Chris Jiggins and Stu West for supervision of this shape-shifting thesis. Nick Barton, in particular, has repeatedly encouraged me to express ideas algebraically, and has had great patience with me. Nick also specifically helped with simplifying the algebra of the second model in the sweeps chapter. For discussion, I am further indebted to Toby Johnson, Laurence Loewe, Andy Gardner, J Taylor, Steve D. Simpson, Dieter Kadelka, Denis Roze, Jonathan Coe, and Alex Kalinka. Kathi Foerster and Josephine Pemberton made suggestions on applying and testing theory with real data. Dan Gaffney and Peter Keightley were a joy to collaborate with for my first published paper. (The idea for the paper was Peter's.) The Charlesworths - Deborah and Brian - have occasionally recommended reading, and allowed me to socialise with their lab group. Andrew Tuson and Tim Taylor were generous with their time and advice when I was interested in genetic algorithms. My external examiner, Ben Sheldon, has suggested very appropriate, necessary, and useful revisions to the thesis. Thanks also to Sean Nee, my internal examiner, for organising and chairing the viva voce, keeping spirits high, and for additional suggestions.

I would like to thank Angus Buckling, John Pannell, David de Lorenzo, and Stuart Baird and other alumni of the Barton empire for their encouragement; Milan Mijajlovic, Alexie Papanicolaou, Alex Phythian-Adams, Sven Kreiss, and many budding technology entrepreneurs for intellectual stimulation, much of it outside biology, and less often than I wished.

I also have to thank numerous colleagues who have sent, or offered to send, reprints of their publications free of charge (in no particular order): N. V. Joshi, Michael Rosenzweig, Marc Mangel, Lee Cronk, William Stubblefield, Marco Festa-Bianchet, Ido Pen and Kathleen Miglia. Due to my rather broad interests, some papers have not related directly to this work, and hence are not cited.

Finally, I would like to thank and congratulate all who have been bold enough to publish their work for coming generations to criticise.

Any omissions from the above lists are accidental and regretted.

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Chapter 1

Introduction

1.1 Motivations of interest in the sex ratio

It may be reasonably conjectured that people have been interested in sex ratios ever since the first animals were domesticated, and the two sexes found to be of different utility; males, for instance, produce neither eggs nor milk in any domesticated species, and the rate of growth of an animal herd is usually limited by the number of females; not only is each male birth a loss of potential growth, but it also creates an animal that, unless killed, consumes additional food. Slaughtering creates ethical conflicts that need to be mitigated at some additional cost (which could range from devising methods or facilities to conceal the slaughter to creating rituals to appease the spirits, or founding storytelling traditions that justify the killing). For animals whose value is not in their meat products, producing a female-biased sex ratio could have provided an ethical and thus cultural benefit.

Interest in the sex ratio has by no means been restricted to animals; it has been desirable, although not yet possible, for humans to manipulate the birth sex ratio in cultures where only one of the sexes required a dowry in order to get married, and in post-war situations, where many women were widowed or destined to remain single. The converse may be true of raids, where women may have been removed from a local population. Finally, the boundless curiosity of 19th century gentlemen also included the sex ratio and what factors might contribute to sex ratios within particular families (e.g. Darwin 1871, 1874, Starkweather 1883).

Furthermore, many species have easily distinguishable sexes (Darwin 1871, pp. 253-255), allowing relevant data to be easily obtained. It has also been known since Darwin's time (Darwin 1871 chpt. VIII) that in many species, females exert strong selection on males - stronger usually than the sexual selection experienced by females. This is simply another way of saying that male success is more variable than female success - some males sire many progeny while others remain bachelors, as Darwin called them. Statistically, we should distinguish two kinds of variance in fitness. The first arises through the sampling procedure by which (in most species) the vast majority of female gametes are fertilised (i.e. very little variance), while the gametes of some males may fertilise more eggs than those of others. This constitutes genetic drift (or a reduced effective population size), is discussed in more detail in chapter 2 and, while inconsequential to the sex ratio adjustments here discussed, is relevant to the question of whether fitness variance can be a useful metric. Secondly, though, there are situations where some males are systematically more successful due to some trait that they possess. This is known as sexual selection.

1.2 Academic history

Sex ratios have been of interest since it was discovered that they vary considerably. They are easy to study, since, as Darwin (1871, pp. 253-255) points out, the two sexes often show considerable morphological differences as adults, at least in their external genitalia, so the sex can usually be determined once the animal has been caught (see Andersson 1994 for a more recent review).[1]

Evolutionary biologists also have a growing interest in sex allocation theory due the close match between predicted and observed ratios, particularly in parasitoid and fig wasps, which serves as one of the best demonstrations of the power of natural selection (Godfray 1994, Hamilton 2005 p. 132). In fact, Godfray has suggested that the accuracy of quantitative predictions generated from sex ratio theory has been compared to those of astronomy and physics (Godfray 1994), although this mostly applies to the more primitive species commonly studied in the laboratory (West *et al.* 2005).

Darwin (1871) discusses sex ratios first in the context of sexual selection (pp. 261, 263-265), and then in their own right (pp. 300-318). He cites a large number of statistics, including a considerable number of ratios from human beings in different countries, and observes certain trends. For instance, he has figures to show that Jewish communities have a more male-biased sex ratio at birth than Christian ones, that males are more often stillborn (indicating an even larger bias at conception), and that illegitimate births are more female-biased than legitimate ones. He makes the link between the lower mortality of the smaller, female infant at birth and in early childhood and both the greater likelihood of illicit children

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[1] Alternatively, if sex determination is genetic, the sex ratio can be estimated from pools of DNA samples representative of the composition of the population (e.g. Barcellos *et al.* 1997, Johnson 2005).

being born to young mothers, and (although not explicitly in these words) the stress caused by carrying an illicit child (he refers more to the physical than the psychological).

Darwin gives a rationale for sex ratio evolution that, as several authors have pointed out (Seger and Stubblefield 2002 and references therein), comes very close to our current understanding:

> *Let us now take the case of a species producing from the unknown causes just alluded to, an excess of one sex-we will say of males-these being superfluous and useless, or nearly useless. Could the sexes be equalised through natural selection? We may feel sure, from all characters being variable, that certain pairs would produce a somewhat less excess of males over females than other pairs. The former, supposing the actual number of the offspring to remain constant, would necessarily produce more females, and would therefore be more productive. On the doctrine of chances a greater number of the offspring of the more productive pairs would survive; and these would inherit a tendency to procreate fewer males and more females. Thus a tendency towards the equalisation of the sexes would be brought about. But our supposed species would by this process be rendered, as just remarked, more productive; and this would in many cases be far from an advantage; for whenever the limit to the numbers which exist, depends, not on destruction by enemies, but on the amount of food, increased fertility will lead to severer competition and to most of the survivors being badly fed. In this case, if the sexes were equalised by an increase in the number of the females, a simultaneous decrease in the total number of the offspring would be beneficial, or even necessary, for the existence of the species; and this, I believe, could be effected through natural selection in the manner hereafter to be described. The same train of reasoning is applicable in the above, as well as in the following case, if we assume that females instead of males are produced in excess, for such females from not uniting with males would be superfluous and useless.*

By the 1879 edition of his book, however, Darwin seems to have lost the clarity of his thought, and replaces this section with a discussion of differential infanticide. In that edition, the discussion of sex ratios closes with the suggestion that the *"problem is so intricate that it is safer to leave its solution for the future"*.

As early as 1883, Düsing, Düsing gives a superbly clear exposition of the idea, by noting[2],

> *All the males taken together have just as many offspring as all the females have (namely the same ones).*

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[2] I use here the translation by Edwards 2000 as I have not seen either of the original papers, or the book, myself

and proposed the first mathematical rendering, the basics of which can be gleaned from Seger and Stubblefield (2002). It is a simple algebraic model that may be the first mathematical model in evolutionary biology (Seger and Stubblefield 2002).

The famous statistician and population geneticist Sir Ronald Fisher probably deserves a mention here, as he is more widely credited and was for many decades more widely read than Düsing, for formulating a model that could not add much to the latter. In his 1930 book, *The Genetical Theory of Natural Selection*, he writes,

> *each sex must supply half the ancestry of all future generations of the species. [...] It follows that the sex ratio will so adjust itself [...] that the total parental expenditure [for] each sex, shall be equal.*

It is interesting, however, that Fisher already gives a verbal equivalent of a modifier model:

> *[...] those parents, the* ***innate tendencies of which*** *caused them to produce males in excess, would, for the same expenditure, produce a greater amount of reproductive value; and in consequence would be the progenitors of a larger fraction of future generations [...]*

(emphasis mine)

If we substitute "allele" for "innate tendencies", we are left with a description in terms of modifier loci as you might find in a peer-reviewed article of our day.

Note that in the first quotation, Fisher speaks of equality of *expenditure*, not numbers - an important insight. This parental investment might include prenatal nourishment, time and effort invested in parental care and future fitness detriment through competition with offspring, e.g. sharing of a parent's territory with an offspring. It turns out that being able to accurately measure parental investment is both a crucial and non-trivial step in predicting sex ratios, and we shall revisit it later.

Fisher's idea thus includes the possibility that some species may have larger males, and that these would be produced in smaller numbers to make the overall investment in the two sexes equal. In the same chapter, Fisher addresses issues of sexual selection, and from his omission of any argument to the contrary, it seems he believed the sex ratio would be influenced only indirectly, following sexual selection for a larger investment in individual males or females.

Stubblefield (1980) has given a good bullet-point summary of Fisher's argument:

1. Each parent has only a limited amount of resources to convert into offspring. This may include costs necessary to obtain mating opportunities.
2. Every individual has a mother and a father, each contributing (approximately) half of its genetic composition. (Note that this does not apply to haplodiploid species, where one sex comes from unfertilised eggs.) Therefore, the sum of all females' fitness equals the sum of all males'.
3. Assuming that the number of recruits into the next generation is fixed (that is, selection is "soft"), females are competing for their contribution only with other females, males only with other males. (This has nothing to do with actual competition for resources, where individual females may well compete with individual males.)

Originally, sex allocation theory was being developed to explain why some species deviate from a 1:1 ratio, or to predict the most adaptive time-point for a sex-changing individual to transform. However, as it started being used to make predictions for species of given genetics and behaviour, examples have been found where sex ratios did not vary as predicted by theory (e.g. Hamilton 1967). Both cases where deviations are observed, and those where it is expected but not observed, must be explained in terms of selection pressures or evolutionary constraint.

From the early 1960s through to the early 1980s, the field was dominated by publications relying on optimality arguments rather than explicitly genetic models (e.g. Bodmer and Edwards 1960, Kolman 1960, MacArthur 1965). The first modifier allele studies of sex ratios were Spieth (1974) and Eshel (1975) studying modifiers of parental investment in offspring of the two sexes.

Having discussed how clear and well-supported some of the early sex ratio theory is, I have to concede that sex ratio theory has also produced theories with more equivocal evidence. This specifically applies to what I shall discuss in broader terms as condition-dependent sex allocation, but which is known in various narrow and broad senses as the Trivers-Willard hypothesis, theory, model, or theorem (most authors)[3], conditional sex expression (CSE) (Frank and Swingland 1988, Frank 1990)), generalised Trivers Willard hypothesis (gTWH) (Kanazawa 2005, 2006, 2007), and, finally, condition-dependent sex allocation (Miller and Aviles 2000) - the term I favour for its descriptive nature and recent

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[3] Clutton-Brock (1991) has argued that condition-dependent sex allocation has been previously, but tersely, described by Willson and Pianka (1963). Furthermore, it is just one special case of the same theory also underlying host size and sex change, interest in both of whom pre-dates 1973.

popularity (Miller and Aviles 2000, Thuman *et al.* 2003, Schwanz *et al.* 2006, Wild and West 2007). Condition-dependent sex allocation is a theory that since its inception has been claimed both proven and refuted, often in the same study species (Festa-Bianchet 1996). It is this theory that is the focus of this publication, and which I shall return to after some brief excursions into the wider background.

1.3 The tale of two sexes

The male and female sexes differ in a number of interesting ways that have had major consequences for natural history. As I shall explain in more detail in the remainder of this introductory chapter, males are typically much less limited in their reproductive success, typically producing amounts of sperm or pollen that would be sufficient for fertilising many females; and in most species, males are much less tied to the fate of their offspring (as females are whenever fertilisation takes place inside the female). On the other hand, some DNA, such as mitochondrial DNA in animals, does not transmit through sperm (or only in exceptional cases, e.g. Schwartz and Vissing 2002), that is, males are only vectors of nuclear DNA. More importantly perhaps, males have little influence on the initial size and nutritional fate of an egg.

And yet there are other species in whom males are not more variable in their reproductive success, or even less so, as well as species where males do most of the investing and looking after.

When the two mating types differentiated in size (anisogamy) and became proper sexes - in itself probably an inevitable development (for a review, see Hurst 1996) - they set in motion a chain of events. As one sex made the main investment in offspring, it could afford to be choosy (Johnstone *et al.* 1996), which increased the variance in reproductive success among members of the opposite sex beyond a value that would have been elevated through random sampling already (Sutherland 1985).

This became one of a number of causes for sex ratios deviating from 1:1, which typically also included a change in the size of one of the sexes, besides other specialisations that may arise by the coevolution inherent in sexual selection and sexual conflict. If the size difference is sufficiently large, the two sexes may use their environment in a different way: they occupy different niches. (This may also happen if one sex has a particularly cumbersome adaptation for sexual selection or conflict.) Each sex will have to compete with other species incumbent on its niche, and so there may be selection pressure for the two sexes to diverge morphologically above what is dictated by their sexual coevolution. This requires two phenotypes to be generated from one genome, which may be difficult, but plays a crucial role in the evolution of sex determining systems. One sex may simply have more genes than the other; we observe this in species with chromosomal sex determination, such as mammals, birds, snakes, and Lepidoptera (butterflies and moths) (see Bull 1983, Vallender and Lahn 2006 for more details), or one sex may have more copies, as in species with haplodiploidy, such as Hymenoptera (wasps, ants and bees). DNA can also be chemically modified to change the expression level of genes; this is known as imprinting. Species may also try to mitigate the effects of such divergent selection. For instance, species could have a joint larval stage from which they emerge as adults earlier or later depending on sex (i.e. sex is determined by laying date). The adult stage could be short, obviating the need for competing with other species for food in two different niches. An adult life that is short by design would also at least initially avoid predation (although predators will, in the long term, adapt).

There is a connection between sex determination and sex ratio evolution. For instance, species that have selected for stable 1:1 sex ratios (I will explain how in a moment) will favour the evolution of a sex determining system that ensures this, regardless of external influences - they could evolve sex chromosomes, for instance. Reciprocally, sex-determining systems that do not allow for deviations from 1:1 without significant costs could constrain the species to a particular sex ratio and the behavioural systems this favours.

Much work has been produced on the evolution of sex determining system, the early parts of which are summarised and extended in Bull's 1983 monograph. Most relevant to this thesis is the question of genetic vs. environmental sex determination. First of all, the genetic vs. environmental question raises memories of an important debate in evolutionary biology - the nature vs. nurture debate. One of the important lessons from that debate tells us that even when nurture has a large influence, this is only by permission of the underlying nature (genes). In the case of human intellect, nature (genes) provides the flexibility that nurture (learning) needs. Where does this lead? When discussing environmental sex determining systems it is important that these, too, are selected at a genetic level, in order to enable the organism to respond to the correct stimuli in its environment.

What, then, are the accepted results in this particular subject area? In a first exploratory paper by Charnov and Bull in 1977, the authors propose a number of conditions under which sex would depend on environmental cues, that is (as they explain), in which sex determination would be delayed until more information is available on the life time environment of the individual whose sex is to be determined. So in species in whom the success of offspring of a given sex would depend on environmental factors that cannot be known at conception, environmental sex determination would be favoured. This is just one example that illustrates the interaction between life history and sex determination, and how this is used to fill a sex ratio need.

1.4 Sexual selection causes fitness variance

Female choice can come about in two ways. Firstly, if a female mates with a male that other females have a genetically determined or otherwise inherited preference for, her sons may inherit genetic attributes that make them attractive to females of the next generation. This is the sexy sons hypothesis (Weatherhead and Robertson 1979). Note that it relies on arbitrary tags - whatever makes a male sexy to the majority of the population, is further selected. In particular, it is believed that males could exploit existing sensory responses of the female - for instance, by imitating the sensory characteristics of food items (e.g. Rodd *et al.* 2001).

However, it may be the case that some males convey benefits beyond simple attractiveness, enhancing the success of their offspring not through preferred mating cues, but through enhanced survival, longevity or fertility. Daughters of preferred fathers showing increased fecundity or survival would be strong evidence for such "good genes" being the determinant of mate choice (for the good genes hypothesis, see Fisher 1915, Maynard Smith 1956, Williams 1966, Trivers 1972).

Note that when sexual selection is caused by female choice, the increased variance in male reproductive success actually results from biases in female perception. The better females are at judging the attractiveness of males, the greater male variance will be. Therefore, the more discerning the female, the greater the variance in male reproductive success will be. Whether this will lead to the "lek paradox" of females being such efficient selectors that little genetic variance is actually left for them to judge, depends on details of the system - whether females are restricted to choosing among a small number of local males, whether males disperse widely between breeding seasons, and whether female sensory abilities are sufficiently general to select among new and cryptic variation, for instance.

However, there are known to be non-linear effects as well. For one thing, females are known to prefer rare male genotypes - an effect that is strongly present in *Drosophila* spp. (Ribo *et al.* 1989, Curtsinger and Sheen 1991), but not, for instance, in house flies (Childress and McDonald 1973). This will always conflict with, and limit, the effectiveness of sexual selection in any given direction. Secondly, while it has been long understood that male capacity for fertilisation is not unlimited (Bergerud 1974, Haigh and Hudson 1993) and that males are to some extent choosy (Johnstone *et al.* 1996), it has recently become clear that females of some species will delay mating when preferred males are not present (Mysterud *et al.* 2002). Which is the choosy sex can be determined on the basis of post-mating latency (Hubbell and Johnson 1987) as well as the more traditional limiting resource view.

Two of the general predictions of condition-dependent sex allocation are that the less variable sex will be produced when conditions are worse, and the more variable sex will be the less abundant sex. The former I take as given to arrive at the solution, but the latter arises as a prediction of the model presented here. We can write expressions for the fitness of the two sexes in any given population. Female fitness is simply equal to her fecundity (note that we are ignoring density dependence). Male fitness is measured relative to other males (Charnov 1979a), that is,

$$w_{i,m} = d_{m,i} \frac{\sum_{k|female} d_{f,k}}{\sum_{j|male} d_{m,j}} \tag{1.1}$$

This can reflect situations where

1. females accept males after courtship time proportional to $\frac{1}{d_{m,i}}$
2. males move at a rate proportional to $d_{m,i}$ and mate all females they encounter
3. males produce an ejaculate of size $d_{m,i}$ and ejaculate size determines fertilisation probability
4. females use one ejaculate, but the probability that it is from a given male i is $\frac{d_{m,i}}{\sum d_{m,j}}$ where i is one of the j.

To my knowledge, it is the most general way of representing differential male success, and shall be used for the remainder of this thesis. Likewise, except where stated otherwise, I will assume that males are the more variable and/or more costly sex.

In many species, males do not have certainty over paternity, whereas females can usually be sure of their eggs being fertilised, and, where maternal care is observed, can usually identify their direct descendants.

We can thus identify several questions that are particularly relevant to the question of what is the optimal sex ratio from the point of view of the father, mother, or offspring:

- Who has control over the sex ratio? Does it matter?
- By what mechanism?
- Which sex, if any, is heterogametic?
- Who has certainty of parenthood?
- Whose condition does offspring condition depend on?
- Which sex is making the larger investment?
- Is the investment made in offspring of both sexes equally?

1.5 Sex allocation should follow fitness benefits

Trivers and Willard (1973) had observed several assumed trends in the empirical literature on mammals:

1. The condition of young at the end of parental investment tends to be positively correlated with the condition of the mother during the period of parental investment. This is less true of species with large, variable brood sizes.
2. Differences in condition of young are preserved into adulthood.
3. Adult males will disproportionately gain in reproductive success through small advantages in condition.

As a corollary to 3, Trivers and Willard suggested that, especially in species with negligible parental investment by the father, male reproductive success (RS) would vary much more than female RS.

Trivers and Willard went on to predict that if males were more reproductively variable than females, mothers in some favourable state (such as nutritional condition or social status) would produce sons more often than daughters.

Essentially, Trivers and Willard (1973) formulated a model that assumed that males and females had some traits in common, the possession of which would enhance male reproductive success much more than female. This being the case, prospective mothers would gain by having sons if they can produce an offspring

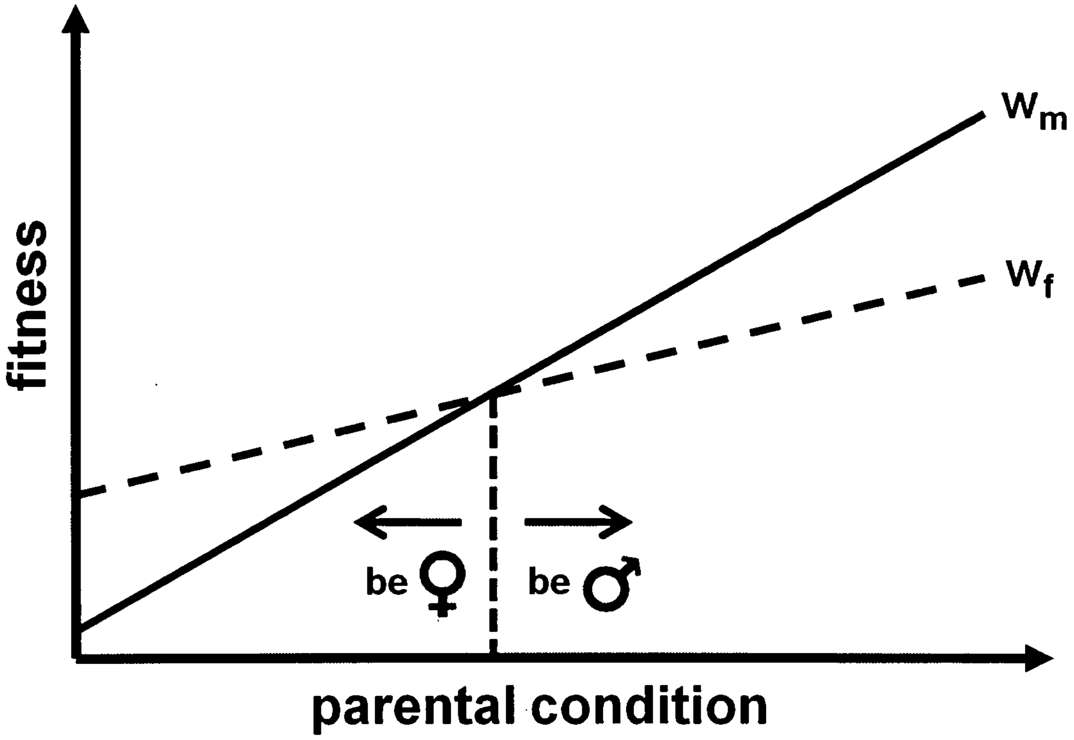

Figure 1.1: The principal idea of the Trivers-Willard hypothesis is that individuals of given trait value may have different fitness depending on which sex they become, and that they will become whichever sex is fitter. When the ratio of male to female fitness is monotonically increasing or decreasing with trait value, there exists a threshold at which the preferred sex changes. After Charnov (1993).

that has a higher value in any such trait. Their original paper suggested that body size was a trait that transmitted in this way, through what we now call maternal effects.

Trivers' and Willard's theory was poorly understood when first published. Even G. C. Williams (1979) posits that the theory applies when the costs imposed by sons and daughters are different, and goes on to summarise Trivers and Willard as having stated that sex differences in fitness variance bias optimum expenditure. Retrospectively, it is easy to point out that trivially, when some females cannot produce say, sons, because they do not have the necessary investment at their disposal, then they will not. The correct way to phrase Trivers and Willard is to say that the sex that benefits more from increased investment will be produced when *more* investment is available. In fact, allowing individuals to manipulate the investment they make while allocating some of it to other purposes, such as additional offspring in the current or future seasons, or additional investment in particular offspring, introduces further complications that Trivers' and Willard's model was not equipped to deal with. In fact, Williams goes on to attempt a discussion of these complications, but he introduces the unwarranted assumption that there is an ideal amount of investment for each sex produced, when in reality, the availability of resources may determine the optimum, and fitness may vary linearly with investment for each sex over some larger range of parameters.

The evidence Trivers and Willard cited from the empirical literature (for references, see their paper) was criticised by Myers (1978), mostly on the grounds of statistical nonsignificance and showing opposite trends to those predicted by the theory. However, Trivers' and Willard's contentions were later vindicated by a review paper by Clutton-Brock and Albon (1982) presenting more solid evidence from a large range of sources and taxa.

Extending from the model based on maternal condition, it is in fact true to say that whenever fitness variation in one sex is more strongly affected by parental investment, there will be consequences for optimal sex allocation strategies (Trivers and Willard 1973) and/or sex ratios (Bull 1981, Charnov 1982, Frank and Swingland 1988). I will review these early theoretical developments in detail in the theory section of this chapter.

1.6 Host size hypothesis

One example where the condition of various offspring differs is when a parasitic species encounters hosts of different size. This is invariably the case in parasitic species because hosts may not have completed growth when infected, and are likewise likely to have encountered environments of varying quality as a result of stochastic factors.

A good example of this is Charnov's (1982, p. 39) example of sex ratio adjustment in a parasitic wasp. Parasitoid wasp females lay a single egg in each host, but hosts will differ in size, and hence the amount of nutrient available to the offspring. However, one sex may benefit more from being larger than the other. In Charnov's example, a "large" female will lay ten times more eggs than a "small" female, while for the same nutrient available, a "large" male will only inseminate three times as many females as a "small" male. (In fact, males beyond a certain size may be less agile and hence less successful.) Since wasps have haplodiploid sex determination, it is easy to see that mothers could be selected to control the sex ratio by choosing which eggs to fertilise. Knowing that sexes differ in their success based on host size, we may expect her to base her sex decision (the *sex allocation*) on host size, too.

Charnov, in the same volume, considers a number of empirical examples of host size effects from the earlier literature.

Parasitoid Hymenopteran species have become the most important experimental system for demonstrating host size effects. Chewyreuv published the first data in 1913, anticipating what is now recognised as the root of theoretical developments, by 60 years. Chewyreuv (1913) offered *Pimpla* wasps either one of two sets of pupae each containing a larger and smaller species, and found that sex ratios were always more female-biased on the larger host species. Brunson (1937), using the Japanese beetle, *Popillia japonica*, and its parasitoid Hymenopteran parasite, *Tiphia popilliavora* for his experiments, found that female *Tiphia* prefer to parasitise third instar over second instar larvae of the host, and produce strongly male-biased sex ratios when offered second instar larvae, versus slightly female-biased when given third instars. By a cross-fostering setup, Brunson also showed that mothers had control over the sex determination of their offspring, which therefore had to occur prior to oviposition (Brunson 1937). Note that primacy over the finding that parasitoid mothers lay female eggs in large hosts and vice versa is sometimes erroneously attributed in the literature to Clausen (1939) (e.g. Flanders 1965, but c.f. Flanders 1956).

The observed bias makes sense for a species in which male reproductive success is predominantly determined by encounter rates rather than contests between males. Thus it benefits females to be producing small males, each of which can fly equally well if not better, than a larger male, and larger females, in whom body size limits the number of eggs they can lay, and hence, fitness. In fact, *Lariophagus* males even emerged sooner than females, regardless of host size (e.g. Charnov *et al.* 1981). Coincidentally, male birds tend to arrive at breeding grounds before females (Berthold *et al.* 2001, p. 52).

The method by which parasitoid wasps are able to influence offspring sex ratio so radically, consists of a spermathecal gland whose secretions can switch the sperm stored in the female reproductive tract from a suppressed to an active state (Flanders 1965).

S. A. West (draft of an unpublished monograph) has commented that the literature on parasitoids is so plentiful that it allows three publication epochs to be clearly distinguished by journal focus, namely entomological journals prior to 1980, "high impact interdisciplinary and evolution journals" during the 1980s, which West attributes to the influence of Charnov's monograph of 1982, and finally, "applied (biocontrol) and entomological journals" since the 1980s.

Other early evidence for the host size hypothesis is in a paper from 1929

(Christie 1929). In the nematode investigated by Christie, it is males that are disproportionately produced when many parasite worms are present in the host. Charnov (1982) has a longer review of the evidence in nematodes, and also points to parasitoid Diptera as a further source of taxonomically independent corroboration. However, he does note that those species may be constrained by genetic sex determination. Indeed, the dominant male-determining factor, M, is believed to be widespread among Diptera, and possibly ancestral to a wider range of metazoans (Shearman 2002).

The main conclusion from the existing evidence is that there exists a very well-documented pattern of one sex being more often produced from large hosts, that this is not due to differential mortality, but rather, to deliberate sex manipulation by the egg-laying female, and at least the anatomical mechanisms are well documented and understood, with the physiological and especially neurological basis remaining as a subject of further research.

1.7 Allocation to male and female functions in the same individual

Many species of plant, fish, and shrimp, some marine mollusks and a genus of annelid, all devote energy to male and female functions within one individual's lifespan. It can be shown that the same theory that applies to host size also applies here (Leigh *et al.* 1976).

Plants have always been noted for their variety of sexual habits (e.g. Linnaeus 1753, E. Darwin 1791, C. R. Darwin 1877), usually one of the three main habits hermaphroditism (bisexual flowers), monoecy (unisexual flowers of two different sexes on the same plant) and dioecy (male and female plants are separate). Some hermaphrodites are able to self-fertilise, others prevent this.[4]

There is mounting evidence for condition-dependent sex allocation in plants. For instance, two well established facts are that among hermaphrodite plants, reproductive success through male and female functions varies depending on whether a site is favourable or not (references in Freeman *et al.* 1981), and that in monoecious plants, production of male flowers is favoured on xeric sites,

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[4] Apart from this, there are many other variants, such as populations that contain male, female and hermaphrodite plants, and plants that have not only bisexual flowers, but also unisexual ones of either or both sexes. Some insect-pollinated plants add further complexity to this by having more than one type of bisexual flower, typically distinguished by the position of male and female parts (anthers and stamens, respectively) so that each type of flower can only fertilise, and be fertilised by, a specific other type of flower. Often, unisexual derivatives of these flowers are also found.

with female flowers predominating on moist sites (Freeman *et al.* 1976, 1981). This in itself may reflect nothing more than a case of environmental sex determination. However, Freeman *et al.* (1994) subsequently demonstrated that in spinach (*Spinacea oleracea*[5]), plants emerging from large seeds have a male-biased sex ratio, while plants from small seeds are female-biased. Thompson *et al.* (2004) found that cucumber plants (*Cucumis sativus*[6]) respond to herbivory by producing fewer male flowers, in analogy to ungulates and humans, who in some studies were shown to produce fewer males when stressed (see other sections in this chapter). In general, low soil fertility, low temperatures, high stand density, and low light intensity lead to increased allocation towards males (references in Glawe and de Jong 2005). However, as with animals, sweeping generalisms also fail with plants, as high levels of nitrogenous nutrients lead to increased female allocation (Heslop-Harrison 1957). Clearly, more research is needed for plants to catch up with only one of the many groups of animals of whom we have a complex, replicable picture.

In a paper published in 1969, Ghiselin explores several hypotheses that could account for the evolution of hermaphroditism in animals, among them the "size advantage hypothesis", which is the basis of our current thinking on sex change. For most purposes, this is the second origin of condition-dependent sex allocation theory (if we ignore Willson and Pianka 1963 who did not gain wider attention), after the host size hypothesis for parasitoids. The sex-changing strategy is found, for example, in fish such as wrasses (Labridae), who are female first (protogynous), and at least some species of clownfish (Amphiprioninae) who are male first (protandrous). The fact that some wrasses are born as males (Warner and Hoffman 1980) suggests that there may be two layers of condition-dependent sex allocation in these species.

Again, we can note that a flexible strategy such as changing sex at an opportunistic time, may have costs (Leigh *et al.* 1976, Charnov 1982). Specifically, these costs involve the time and energy taken to undergo various hormonal, other physiological, and anatomical changes, as well as learning, if the mating behaviour of the new sex is different. A general intuition is that higher costs will delay the age at which sex is changed. One can think of this as being due to the risk of death during transformation, which favours gaining further fitness as the current sex over going through a period of nonreproduction at the end of one's life, as it were. Having said that, sex change in many protogynous species can be directly

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[5] a plant with morphs that release their pollen either before or after their stigmata are receptive, or are completely male or female (Miglia and Freeman 1996)

[6] a monoecious species

triggered by the removal of the dominant male of the breeding group (Warner 1982, Allsop and West 2004).

More recent developments in the field include the finding that sex changing in fish occurs at the same relative size across all the taxonomic groups containing sex changers (theory by Charnov and Skuladottir 2000, empirical support by Allsop and West 2003a[7]). An additional finding, also reported in Allsop and West (2003a), that relative age at sex change is also invariant, used a smaller sample, and the resulting graph showed two clusters, one with protandrous species, and the other protogynous, with statistical significance a possible artefact of the distance between the two clusters, rather than within-cluster patterns (protandrous species were shorter-lived). It is interesting that there have been no reports of invariant properties of host size or maternal condition.

As part of the analysis, Allsop and West (2003a) noted that whether early-maturing males were present in the otherwise protogynous species significantly affected the relative size at sex change. This difference was followed up in a subsequent paper (Allsop and West 2004), which confirmed that sex ratio is always biased towards the first sex (confirming Fisher's model), and that this bias is less extreme in protogynous species with some proportion of early-maturing males. Allsop and West (2004) also found that sex ratio was more biased in protogynous than protandrous species, following a prediction by Charnov (1982, p. 141).

Simultaneous hermaphroditism is perhaps the least understood among the genetic or mating systems discussed in this section, and Charnov (1982) has pointed to barnacles as a rewarding study system for simultaneous hermaphroditism in animals.

In conclusion, the discussion of sequential hermaphrodites features strong quantitative data on pandalid shrimps and coral reef fishes, but few data available for other sex changers, such as marine molluscs (Patella, Crepidula) and an annelid worm, the polychaete Ophryotrocha, all of whom have been suggested as study systems by Charnov (1982). The finding of an invariant relationship between maximum size and size at sex change, apparently across all sex-changing fish, may be the strongest hint here of the explanatory power of condition-dependent sex allocation theory, and evolutionary theory in general.

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[7] An extension of the survey to other sex-changing groups including echinoderms, crustaceans, mollusks and polychaete worms Allsop and West 2003b did not test for the effect of removing outliers from the analysis, and thus would have to be treated with more caution. Also see Nee *et al.* (2005), de Jong (2005) for a recent critique of these kinds of studies.

1.8 Detecting condition-dependent sex allocation

There are two principal routes to establishing that condition-dependent sex allocation plays a role in a given population. The first is to determine that there are particular correlations of quality and sex (e.g. maternal dominance (Ostner *et al.* 2005), timing of conception (Holand *et al.* 2006), food availability (Holand *et al.* 2006)).

The second is to obtain data on reproductive variance of the two sexes, as is possible with pedigree data that is available for an increasing number of populations[8]. However, in doing so, we must correct for the inflation in variance in the under-represented sex (caused by the frequency-dependent nature of fitness with two sexes).

Let us imagine that there is a study that has demonstrated that sex ratio is adjusted in response to some characteristic of the mother or father, and we would like to determine at which point in the life cycle the adjustment occurs. Our test should include several criteria:

The first test might include showing that the mother has lost more weight, has a shorter residual lifespan, or invests less energy in subsequent reproductive events (as indicated by number or sex of offspring, although the latter on its own makes for a circular argument, and is not sufficient). However, we must also reasonably show that neither are there other benefits to the mother during the time of investment, such as mate fidelity in species tending towards monogamy, nor do mothers with, say, low life expectancy always produce sons. More importantly, we must accept the caveat that perceived fitness benefits in one generation could always cancel out in subsequent generations, albeit by some more complex causality not favoured by Occam's razor.

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[8] e.g. red deer (*Cervus elaphus*) (Clutton-Brock *et al.* 1986), Soay sheep (*Ovis aries*) (Paterson and Pemberton 1997), bighorn sheep (*Ovis canadensis*) (Coltman *et al.* 2002), North American thinhorn sheep (*Ovis dalli*) (Worley *et al.* 2004), collared flycatchers (*Ficedula albicollis*) (Kruuk *et al.* 2002), blue tits (*Parus caeruleus*) (Kempenaers *et al.* 1996), great tits (*Parus major*) (Garant *et al.* 2004), mute swans (*Cygnus olor*) (Quinn *et al.* 2006). Pedigrees will be at different completeness and accuracy - I am not aware of an agreed definition of what constitutes a finished pedigree, and have thus included studies in whom the pedigree was implicit.

1.9 When fitness varies with an individual's condition

The standard condition-dependent sex allocation model with a) complete positive correlation between prospective male and female fitness of an individual, and b) full information for the agent determining sex (i.e. usually mother or offspring, rarely the genetic or social father), has a few interesting properties. First of all, it predicts a threshold behaviour, according to which any individuals exceeding a certain trait value are (assuming male reproductive variance greater), male, and all others female. The very basis of the theory thus has interesting implications in that the variance that can actually be measured (i.e. variance in the realised fitness of males and females) is actually not the relevant variance to determine the behaviour of the system, and whether it is a Trivers-Willard case at all. The relevant variance is the fitness variance if all individuals in the population *were* male or female - a hypothetical case since fitness is always zero unless both males and females are present. However, knowing for each individual what its fitness would be as either sex is crucial to the solution of this optimality game.

It is trivial to obtain this distribution in simulated data, but seems near impossible to do so in a wild population. Assuming that the fitness of individuals relative to other individuals in the population is not affected by the abundance of the two sexes, a hypothetical experiment to determine the fitness distributions of the two sexes in the wild would be direct manipulation of one population to be all-male, and a replicate population to be all-female (e.g. by hormonal treatment). The fitnesses of individuals could then be measured to deduce the original fitness distribution of a single population for both sexes. However, it is not clear whether some genotypes would perform better in different competition experiments. N. Barton *(pers. comm.)* has argued that one would be more likely to accurately represent natural variation by measuring marginal fitnesses, that is, changing the sex of only one individual at a time, in a large population. However, this proposal is hardly practical as an experiment without other additional assumptions (such as a monotonic function relating male and female fitness of given individuals).

In order to experimentally exclude the action of Trivers-Willard dynamics in a population, it may not be sufficient to establish that the fitness variances of the two sexes do not differ. Thorough studies would demonstrate correlations between offspring sex and fitness and paternal trait values, as in studies by Ostner *et al.* (2005) and Holand *et al.* (2006).

A different approach to the problem is to measure realised fitness of males and females separately, and fit statistical distributions to the data, using a Kruskal-Wallis or similar test to check that the observed tail fits the suggested distribution (and it would be nice to develop a more exact and less ad hoc estimation procedure). However, this technique is further hampered in any realistic dataset, in which females have incomplete information, adding heterogeneity such that the realised sex allocation is sub-optimal and does not conform to predictions of the standard Trivers-Willard model. To understand such cases, it is necessary to understand the predictions arising from various degrees of incomplete information, and the skew this effects on the fitness distributions of males and females in a population.

Of the three main assumptions of the model by Trivers and Willard (mother's condition passed on to offspring, offspring's relative condition preserved into adulthood, males benefiting more from increments in condition), it is the effects on fitness, or, usually, lifetime reproductive success, that have been most elusive (Bereczkei and Dunbar 1997, Hewison and Gaillard 1999).

1.10 Are females able to assess their fitness relative to the population?

It is interesting, if not at all unexpected, that in some parasitoid species, females will consistently lay the same sex ratio in hosts of the same size (e.g. *Heterospilus prosopidis* in Charnov *et al.* 1981), even across generations (*H. prosopidis* in Jones 1982) and geographic distances (*Mesopolobus* spp. in Hails 1989), whereas other species will assess the size of available hosts, and adjust their sex ratio response accordingly (e.g. *Coccygomimus turionella* in Sandlan 1979, *Lariophagus distinguendus* in Charnov *et al.* 1981).

However, some of the models that I will present will assume that females can assess the condition of their peers relative to their own. Is this assumption warranted give current literature, or merely convenient *en route* to developing models that include uncertainty?

Kakapo (Clout *et al.* 2002) and water voles (Moorhouse and McDonald 2005) overproduce one sex when external conditions change. Similarly, Brothers Island Tuatara *(Sphenodon guntheri)*, a species with partially temperature-dependent sex determination (Cree *et al.* 1995), are producing a population sex ratio of 0.63, possibly as a consequence of global warming (Nelson *et al.* 2002).

For plants, it has been demonstrated that they are able in principle to assess the population sex ratio, and that indeed *Begonia gracilis* do so (López and

Domínguez 2003). Perhaps further investigation will demonstrate more sophisticated sensory abilities in plants, too.

Clearly then, female animals are able to assess their own condition relative to a historic background, but this might be entirely hard-wired rather than a flexible behaviour that assesses others in the population, and compares that to self. Human data, on the other hand, has some more convincing evidence of relative assessment.

1.11 Do individuals choose investment or sex?

It is safe to say that the original formulation by Trivers and Willard (1973) did not explicitly suggest that males and females had different costs. Their treatment specifically dealt with the effect a mother's nutritional status would have on offspring fitness.

Six years after Trivers' and Willard's, a paper by George C. Williams discussed how sexes would be chosen for a whole litter, given that the mother had a set amount of investment to make (Williams 1979). However, assuming that the more variable sex is produced at the minimum cost at which it can be competitive eliminates the reason why, according to Trivers and Willard, that sex would be originally chosen - it eliminates the very fitness-condition correlation that makes such biased allocation adaptive. In essence, Williams was assuming that the game would reach a stable point - at which the investment made in the more variable sex would not increase further. Williams in fact introduces the Trivers and Willard idea as applying when the costs imposed by daughters and sons are different - a statement that distorts the theory at best. It is not clear whether Williams believed that these costs were somehow physiologically imposed, or whether his argument is in terms of competitiveness among members of the same sex in the offspring generation.

However, Williams did introduce an important question, namely of whether (supposing for a moment the mother to be the sex-determining agent) a female takes a decision more along the lines of "I want a son, so I have to invest more", or "I can only afford a daughter, so that's what I'm going to make", and to what extent females can and do retain investment for subsequent seasons or offspring. Clearly, the answer is unlikely to be found at either extreme. Theory can address this problem, but it remains empirically unsolved.

1.12 Differential provisioning hypothesis

Another example that introduces several crucial ideas is a paper by Burley (1981). Burley showed that in zebra finches *(Poephila guttata)*, attractive individuals tended to have more offspring that were of their own sex (Burley 1981). In that paper she presented sound evidence of two important notions; first, that it is adaptive for individuals to bias the sex of their offspring based on how attractive their partner is to the rest of the population (as a surrogate measure for how attractive they would be in a future population), and that indeed such biases are observable. I shall return to that idea later. The second idea is that sex ratio bias can be brought about by giving more parental care to the preferred sex, at the risk that offspring of the less preferred sex become more likely to die of malnutrition, hypothermia, or other consequences of receiving less attention by parents.

Burley explained that since expression of the leg banding trait that mate selection acts on in this species is sex limited, biasing sex allocation in this way increases the proportion of one's offspring that display the attractive trait. But how do parents achieve this in a species that is constrained by chromosomal sex determination and Mendelian ratios? Burley suggests that parents can recognise their offsprings' sexes soon after hatching and preferentially tend to the offspring of the desired sex, biasing the ratio as a result. A second study by Burley showed that indeed the biased ratio was due to rejection of young with the wrong sex within six days of hatching (Burley 1986a). At the same time, leg band colour also influences mortality rates (Burley 1985, 1986b), suggesting a "good genes" origin of the mating preference.

While the choosy sex may have only limited opportunity to influence the genetic endowment of its offspring, the provisioning sex (to my knowledge invariably the same sex) can devote more or less of its resources to certain offspring or broods.

It is worth noting here that many recent studies point to the importance of Burley's findings within the context of sex allocation; in fact, it merits wondering whether differential provisioning is the main mechanism by which birds and mammals bring about sex ratio adjustments. Specifically, when mating with more attractive males, females of some species feed offspring at higher rates (de Lope and Moller 1993), lay larger eggs (Cunningham and Russell 2000), more eggs per clutch (Petrie and Williams 1993, Balzer and Williams 1998, Uller *et al.* 2005), and endow eggs with more immunoglobulins (Saino *et al.* 2002) and androgens (Gil *et al.* 1999, 2004). Growth and survival of chicks were improved (Petrie

1994) - evidence of either increased parental investment or "good genes". Petrie went to some effort to control for any increased investment, and concludes that the differences observed are likely due to better genes. Note that none of these studies demonstrate sex ratio adjustments; the hypothesis they would seem to support is that the fitness of offspring of attractive males is increased more by small increments in investment than offspring of unattractive males - regardless of the sex of offspring (and the reverse in the case of data presented by Michl *et al.* and Navara *et al.*).

On the other hand, females of other species contributed more to offspring fathered by less attractive males (Michl *et al.* 2005, Navara *et al.* 2006a,b). This makes sense if you consider that a female should invest so as to increase her inclusive fitness. If genetically poor offspring benefit more from the extra resources, she should preferentially provision those. It all comes down to the relationship between investment and fitness, which can be graphically illustrated (Fig. 1.2) and may be different for different offspring. Therefore seeing whether more investment is given to offspring fathered by attractive or unattractive males can potentially tell us something about the relationship between investment and fitness. However, Navara notes that the difference can be explained by the study species' (house finch, *Carpodacus mexicanus*) short life expectancy urging females to pursue a compensatory strategy in order to have all her current brood survive (Navara *et al.* 2006b).

The mechanism for such adjustments is much clearer now than even five years ago. In a series of studies in the same species, the house finch, Badyaev and coauthors showed that male oocytes grew up to five times faster than oocytes producing females, hence reached ovulation size sooner, and hence overlapped less with their potential siblings, and hence were exposed to less competition for nutrients(Young and Badyaev 2004). Oocytes also clustered by sex within the ovary, male oocytes usually being found close to their younger brothers, but not their sisters (Badyaev *et al.* 2006).

One has to be careful, especially in the context of parental investment, not to assume that the objective of evolution is to maximise the number of offspring in the next generation. In many organisms, parents can choose how large an investment to make in each offspring, and hence, how many offspring to produce (Lack 1947, Sinervo and Licht 1991). However, poorly provisioned offspring may have low viability or reproductive success, so a compromise is often found (Lack 1947, 1954, Williams 1966, Wilbur 1977, Sinervo and Licht 1991). In essence, organisms maximise the geometric mean of offspring produced in consecutive

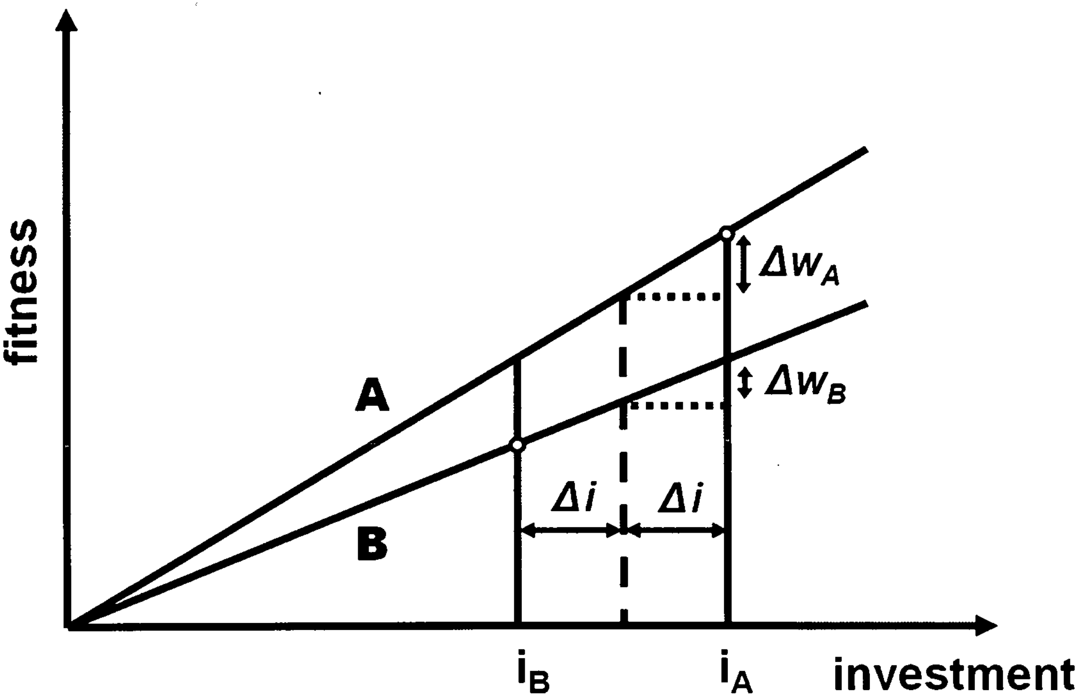

Figure 1.2: When two offspring, A and B, are present, and one offspring benefits consistently more from investment, it pays parents to entirely neglect one offspring and invest completely in the other, as indicated here by Δw_A being greater than Δw_B. However, parents may have a limited ability or incentive to adjust investment, with Δi the maximum possible change in investment. In that case, investment will be adjusted to i_B for B and i_A for A, as additionally indicated by the open circles.

generations, the geometric mean being an exact model of the multiplication of offspring down a lineage (Andy Gardner, pers. comm.).

1.13 Sex expression by attractiveness

Trivers and Willard had suggested that sometimes one sex may benefit more from small increments in investment than the other, and therefore that sex would be preferred when more resources are available. The concept of investment, however, can encompass genetic investment, as would occur, for instance, if a female got a rare opportunity to mate with one of the most desirable males in the population. Burley (1981) was first to express this idea, based on data she had obtained showing that in zebra finches *(Poephila guttata)*, attractive individuals tended to have more offspring that were of their own sex (Burley 1981). Burley suggests that parents can recognise their offsprings' sexes soon after hatching and preferentially tend to the offspring of the desired sex, biasing the ratio as a result. A second study by Burley showed that indeed the biased ratio was due to rejection of young with the wrong sex within six days of hatching (Burley 1986a).

Realising that the aforementioned infant mortality derives from a preferential provisioning of some offspring over others, I shall refer to this as the differential investment hypothesis. It has been previously known as the differential *allocation* hypothesis, but this term is confusing because sex ratio adjustments in a broader sense are also referred to as *sex allocation*. To recap then, Burley introduced both the idea that investment could be genetic, and that sex ratio adjustment and investment could be through differential investment after hatching or birth.

Following these findings, Owens and Thompson (1994) refined Trivers' and Willard's theory by adding the effect of variation in mate quality to the model. They argued that individuals would strive for an ideal trade-off between mate quality and reproductive rate. I have already mentioned the example of zebra finches *(Poephila guttata)* having more offspring of their own sex if they (either parent) are attractive (Burley 1981). The hypothesis put forward by Owens and Thompson was first empirically confirmed in blue tits, who were found to overproduce sons when mated to males with high survival prospects (Svensson and Nilsson 1996). In a corroborating study by Sheldon *et al.* (1999), ultraviolet reflectance was identified as a correlate of male survival probability, and females adjusted the sex ratios of their offspring in response to this variable, with males who survived to the next season having a sex ratio of approximately 0.7 among their offspring. The original blue tit result was also replicated in fairy-wrens

(Rathburn and Montgomerie 2005). Polyandrous female side-blotched lizards *(Uta stansburiana)* produce more sons when mated by large males, and similarly, more daughters when mated to small males (Calsbeek and Sinervo 2004). In addition, males had higher survival probability if sired by large males, and daughters when sired by small males (Calsbeek and Sinervo 2004).

1.14 Operational sex ratio

Trivers (1972) noted that when there are differences in parental investment, the higher investing sex is "limiting" and becomes choosy. Reciprocally, the non-limiting sex will compete for opportunities to mate with the limiting one, and be less choosy (Trivers 1972). This has been recast in the definition of the operational sex ratio, which is the ratio of sexually receptive males to females (Emlen and Oring 1977). Pipefish are a species in which only males have brood pouches that are the limiting factor of the species' reproduction. Berglund (1994) showed that in this species, the operational sex ratio can vary from female-biased to male-biased, and when ratios are female-biased, males become choosy, whereas when ratios are male-biased, they mate indiscriminately and more quickly, leading to higher rates of reproduction when ratios are male biased. Further evidence comes from female fireflies who respond more readily to male signals when males are rare (Cratsley and Lewis 2005), suggesting that they, too, become less choosy as males get rare.

Similar behaviour can be found in some ungulate species, where females, in the absence of large, older males, will refuse to mate with smaller males for some time - presumably in the anticipation that larger males may arrive in the interim (Mysterud *et al.* 2002).

Restating the Trivers-Willard hypothesis from earlier, when males have greater fitness variance than females, those parents, or indeed offspring, who produce or become males when they have high fitness potential, will spread their genes more quickly than others. That is, the operational sex ratio becomes skewed. This means that selection on male traits is further strengthened compared to selection on traits in females. Even in species that are acknowledged to engage in condition-dependent sex allocation, such as red deer, bachelor males are still fairly common (dataset of red deer on the Isle of Rum), which suggests that sex ratios may be tending to yet further extremes - or that environmental variance is high. At first sight, this has the appearance of a runaway process, in which males become successively rarer and more favoured by selection, but it is definitely held in check. Consider that limitations in gamete production can be overcome by selection; limitations in travel distance necessary to meet all the females are much more difficult to overcome.

1.15 Lack of strong sex ratio deviations in mammals and birds

As many authors have admonished, the evidence for condition-dependent sex allocation, particularly in mammals, is less clear (e.g. Hewison and Gaillard 1999, Sheldon and West 2004, Cameron 2004).

There are two genetic sex determination mechanisms that could give one parent complete control over the sex of an offspring. The first is haplodiploidy, and the second would include the ZW chromosomal and XY systems (with their multilocus variants giving progressively less control) (Bull 1983).[9] However, haplodiploidy also results in great sexual dimorphism, in which the role of the male is typically that of a fertilisation instrument. This may be unpractical in "higher" animals such as mammals, in whom males perform more varied roles, frequently including parental care, and where mate choice is a more complex affair that seems to assess the characteristics and abilities of a candidate genome in greater detail than in many insects, whose nervous systems are more limited. Again, it's not clear whether cognitive abilities are set in stone and sex ratio strategies evolve in response, or vice versa.

This argument about the sophistication of cognition is the chicken-and-egg nestmate of West *et al.*'s 2005 hypothesis about greater predictability of a typical insect offspring's environment. The critical issue here is the fast life cycle of most insects, which allows the environment to be more predictable (because the intervening time is short), but also leaves no time for brain morphology to change in response to the requirements of different life cycle stages. The avenues for testing such a hypothesis are therefore limited.

There is some evidence from theoretical studies that sex determination mechanisms can change quickly (Kozielska *et al.* 2006), and in Japan, four regions of Japanese wrinkled frog (*Rana rugosa*) populations can be distinguished on the

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[9] It can be argued that heterogametic systems such as the XY (ZW) give control not only to the male (female), but also to the female (male) when fertilisation happens inside their body, as they could refuse to accept sperm carrying the "wrong" sex chromosome. However, males (females) could refuse to supply sperm (ova) with the "correct" chromosome, forcing their partner to remate until they can achieve the desired sex. Optimality theory predicts that females (males) will eventually give up and accept sperm (ova) of the "wrong" sex. Additionally, circumvention mechanisms would likely evolve, as they do in other examples of sexual conflict.

basis of which is the heterogametic sex (two regions) and whether the Y/W is degenerate (Ogata *et al.* 2003). In addition, significant deviations from a 1:1 sex ratio have been observed even in species with chromosomal sex determination (e.g. Madsen and Shine 1992b, Komdeur *et al.* 1997). Essentially then, different sex determination systems may simply impose different costs on individuals preferring a different ratio (reviewed in Krackow 2002).

A question of particular interest is the extent of variation in offspring sex between different individuals - are there individuals who consistently produce only sons, or only daughters? West and Sheldon (2002) have examined this question in a meta-analysis of studies that focused either on mate attractiveness, or on related individuals helping at the nest. While the studies were consistent with predictions from theory, and effect sizes significantly different from zero, West and Sheldon also commented that the necessary sample size to detect an effect with reasonable certainty was used in only two of 17 studies, thus making it likely that other studies of similar sample size may have been unable to detect these modest effects. While the sex ratio skew was greater for helpers at the nest than for mate attractiveness studies, this relation was roughly the same as between parasitoid wasps that kill or do not kill their offspring's host at oviposition. West and Sheldon (2002) note that this provides support for the environmental predictability hypothesis (first discussed for mammals by Leimar (1996), and birds by Sheldon (1998)). Other differences between Ichneumonid wasps and higher vertebrates include that birds and mammals typically have overlapping generations, with several cohorts living together, and smaller litters. In a paper in which Schwanz *et al.* (2006) introduce a model of condition-dependent sex allocation with overlapping generations, they suggest that some of extensions of such a model "might help elucidate the inconsistent sex ratios of mammals". In the ensuing discussion, they refer to inconsistencies in which sex is produced when conditions are good. However, it is possible such models will also help explain why effect sizes are typically small in mammals and birds. For instance, the longevity of existing dominant males in the population may not be known to a female, and so the lifetime reproductive success of her own sons is uncertain. On the other hand, for many species it has not been reported to what extent individuals compete for sexual partners within, or outside, their cohort, and whether experimental manipulation of the current population sex ratio leads to adaptive shifts in the sex ratio of newborns. Of continuing interest is the extent of sex ratio variation within litters (MacArthur 1965, Williams 1979, Krackow 1997), a rough indication of the degree of control mothers have over offspring sex ratios, and the certainty they attach to their sex ratio decisions.

1.16 Heterogamety - a barrier to allocation?

It is perhaps unfortunate that the idea of condition-dependent sex allocation was first developed for mammals, most of whom have X-Y chromosomal sex determination, which usually entails an increased mortality rate in the heterogametic sex - the males in this case. This can result in a fitness variance increase in males that may explain the origin (as opposed to maintenance) of skewed sex ratios, and contributes to males being the sex more often expressed in favourable condition. However, the heterogamety and resulting higher mortality of males also drew the criticism (e.g. Myers 1978) that the observed condition-dependent allocation could be entirely explained by the heterogametic sex being more vulnerable to stress. While this explanation is unsatisfactory in requiring an apparently maladaptive feature, X-Y sex determination, to have evolved (although some maladaptive traits are favoured by natural selection, such as meiotic driving genes), it has been extensively discussed, not only by Trivers and Willard in the original 1973 paper, but also by Myers (1978) and by West *et al.* (2005) and references therein; earlier examples are cited in Eshel (1975).

However, note that mate quality is sometimes mentioned as influencing sex allocation (Owens and Thompson 1994, Svensson and Nilsson 1996, Dowling and Mulder 2006), and an alternative hypothesis exists according to which males may be overproduced in such pairings because they are the heterogametic sex (in mammals) (Trivers and Willard 1973, Myers 1978, Olsson *et al.* 2005). This controversy could be solved by comparing a large number of heterogametic species with independent evolutionary origins; this would include mammals, birds, and heterogametic insects such as butterflies. There should be several control groups, notably species with non-genetic sex determination, haplodiploid species, and polyfactorial ones. This, however, is not the focus of this thesis.

It is also worth noting that while the condition-dependent sex allocation and heterogamety hypotheses have traditionally been presented as mutually exclusive alternatives (Trivers and Willard 1973, Myers 1978, Olsson *et al.* 2005), it may be that the predictions of the two hypotheses frequently agree because the survival of the heterogametic sex benefits more from parental investment, or because of some other common cause. If it were the case that physiological debilities had a smaller effect in well-nourished individuals, the heterogametic sex might be both more variable and overproduced in favourable conditions.

Meanwhile, Myers (1978) has examined Trivers and Willard's claims under the assumption that parental abortions are the only method by which a mother can control the sex ratio (Trivers and Willard in fact suggested such control could be by differential mortality of Y vs. X-bearing sperm or mortality before the end of parental investment). Myers provides a brief model that assumes that when fewer males are born to a mother, the same number of daughters is born, existing daughters do not receive any extra investment, and that whether or not sons are produced in addition to the fixed number of daughters has no effect on future reproductive success. This is essentially equivalent to assuming that sons require no investment at all, or that the female is somehow unable to increase her investment in existing daughters, and that all savings made by not producing sons are purged at the end of the breeding season. The model further assumes that males born in any condition have a non-zero probability of contributing to the next generation. The straightforward conclusion of this model is that if "stressed" females can give birth to additional sons at no cost, even if the probability that those males will reproduce is very small, it is adaptive to produce such sons.

Myers' conclusions are valid for all such cases as long as fitness of males increases disproportionately compared to females, an assumption that may not hold in species where the outcome of fatal encounters between males depends on parental investment, such as elephant seals. It is also known in many species that a large proportion of males leave no offspring (Chararas 1962, Verner and Willson 1966, Clutton-Brock *et al.* 1982, 1986). There may also be more complex fitness functions with parental investment in species such as those that have small "sneaker" males (e.g. Atlantic salmon *Salmo salar*, at least three *Onchorhynchus* species (Coho salmon *O. kisutch*, chinook salmon *O. tshawytscha*, and rainbow trout *O. mykiss*), rainbow darter *Etheostoma caeruleum*, and bluegill *Lepomis macrochirus*).

1.17 Female dominance hypothesis: testosterone as explanation or mechanism?

In 1980, William H. James suggested that gonadotrophin levels of the mother at the time of conception might influence offspring sex (James 1980a,b). He expanded this hypothesis to include oestrogen and testosterone (James 1986), with high levels in these hormones favouring male births (James 1987b). In 1990, Valerie Grant reiterated this view, but added that hormones were not simply a mechanism to bias the offspring sex ratio, but that offspring fitness did not depend on maternal nutritional status, as Trivers and Willard had originally proposed, but that hormone levels could replace nutritional condition and were superior because a "biologically-based characteristic" that could explain "anomalous findings", for instance, by MacMahon and Pugh (1954), who found raised sex ratios in white US American families during and immediately following the Second World War (James (1987a) has reviewed a larger body of evidence confirming this fact), and Verme (1969), who found that red deer overproduced males in bad condition, a finding contradicted by a later study by Clutton-Brock *et al.* (1984). Grant takes an unusual approach in giving specific weight to the Verme study, which other authors tend to accept as a possible false positive, and proposing the female dominance hypothesis not so much as a mechanism of condition-dependent sex allocation, but rather, positioning it as an alternative explanation of the results of sex ratio studies. What I have found troublesome in Grant's writing is that she suggests that good condition stems from dominance (Grant 2003, p. 98), that is, dominance is an arbitrary signal that agents choose without any recourse to actual resources that might back up this signal, but it enables them to obtain good condition, presumably through uncontested priority access to resources. This would suggest her model of conflict to be a pure dove model, in which threats are never contested. (The hawk-dove model is a classic game theory model, in which "hawks" will always challenge threats, getting hurt whenever they meet other hawks, but always winning over doves, and doves often lose resources, especially to hawks, but never get hurt. Neither strategy is an evolutionarily stable strategy, so a pure dove model is an implausible assumption.)

1.18 Putative mechanisms in mammals

Having discussed some of the physiological evidence underpinning the hypothesis that differential allocation is widespread among birds, it is fitting to discuss similar evidence in mammals.

A recent study by Cameron *et al.* (2008) has sought to test a hypothesis, suggested by an earlier review and meta-analysis by Cameron (2004), that in mammals, sugar levels in the pregnant female have a major effect on offspring sex. Cameron's 2004 meta-analysis of mammalian studies of condition-dependent sex allocation had shown that studies that assessed sex ratio in response to availability of food, and measured sex ratio close to conception, most strongly supported condition-dependent sex allocation in the predicted direction (males favoured when conditions are good). Cameron et al.'s hypothesis is also supported by evidence suggesting mothers can respond differently to male and female conceptuses (Larson *et al.* 2001), with female recruitment decreasing just prior to the blastocyst stage in media with increased glucose levels (Larson *et al.* 2001). Cameron *et al.* used DEX, a glucose transport inhibitor, to lower plasma glucose concentrations in some treatments, and show that these treatments resulted in more female births. In combination with other evidence reviewed by Cameron *et al.* (2008), this suggests that nutritional status of mothers could be regulating sex ratio of offspring through the effect that availability of glucose has on implantation success of conceptuses. It would be nice to know exactly how glucose is sensed, and by what mechanism conceptuses are lost in response to this variable. So far, Cameron *et al.*'s results have been supported by other recent studies. In a study of 42 female voles, Helle *et al.* (2008) demonstrated a positive correlation between mother's glucose levels and sex, as well as mother's testosterone levels and sex. Glucose and testosterone were, if anything, negatively correlated, although not significantly so. Another result that is consistent with Cameron's hypothesis is Mathews *et al.*'s 2008 study demonstrating a correlation between dietary intake of British women prior to conception and the sex of their offspring - more sons were born to mothers whose diet had a higher energy value overall (whether through simply eating more, or having a larger proportion of their diet made up of carbohydrates). Neither corroborating study refers to Cameron's contemporaneously published manuscript (although both cite two of her earlier papers), so may be considered less exposed to publication bias. On the other hand, the effect size reported by (Mathews *et al.* 2008) is, again, small.

Temperature may also influence sex in humans. A recent paper by (Catalano *et al.* 2008) presents data from a Scandinavian cohort to show that lower ambient temperatures during gestation lead to disproportionate survival of male conceptuses to term, as well as longer life spans for those males in later life. I would characterise this as an intriguing preliminary finding that awaits further study.

1.19 Non-human primates: local resource competition vs. condition-dependent sex allocation

Some primates may simply follow the same logic of condition-dependent sex allocation as has been shown for so many other mammals. For instance, in chimpanzees, high status mothers spend more time on their sons before consorting with sires again (Boesch 1997), whereas low status mothers have higher interbirth intervals when rearing daughters (although not significant in this study). The higher survival of high status females' sons compared to other males hints at a fitness gain when these females invest more in sons (Boesch 1997).

But another special sex allocation case, local resource competition, also contains condition dependence. The classic case as told by Clark (1978) has daughters sharing the territory of the mother as they grow up, while males disperse to other social groups and do not compete with their mothers. That is, the condition of the mother (territory) is passed on only to daughters. As territory is a limited resource, mothers may produce a smaller number of female offspring, resulting in a population-wide sex ratio shift towards males. Moreover, a mother's dominance and territory size or resource privileges within her group have no bearing on the reproductive success of her sons, since they leave the group as or before they reach reproductive maturity. Thus we can see that this is a special case of condition dependence where male fitness does not vary with the abundance of the mother's resource (territory), whereas a daughter's fitness depends on her mother beyond reaching puberty.

Silk (1983) described several further hypotheses derived from Clark's, namely, assuming that females control territories and compete for them, it is adaptive for them to suppress production of daughters by other members of their group (again, males are irrelevant to the argument as they disperse). She suggests that selective aggression towards other female's daughters could explain the observed sex ratio through disproportionate female deaths, but that ultimately, this will select for a situation where only intimidating females will produce daughters. A recent paper by Ostner *et al.* (2005), for instance, found a correlation between maternal status and offspring sex in a wild population of Hanuman langurs, and that females from larger groups (who experience more competition and stress) produced more daughters. They argued that this was because males disperse and compete population-wide, so that males from larger groups won't be successful.

If one hypothesises that in these typically matrilineal societies, females have greater variance in reproductive success than males, population-level bias towards males would result. In possibly the most comprehensive review of primate sex ratio literature yet, Schino (2004) showed that female dominance rank influences the sex ratio of their offspring when population growth is high and sexual dimorphism small. Maternal investment was not correlated with sex in his meta-analysis. Schino does not report whether litter weight was significantly related to sex. Perhaps more importantly, though, he does show that the sex ratio effect is repeatable within given populations, using eight populations from six species, that have been studied at least twice. The caveat with that result is that all but one of the species are from the genus *Macaca.* It's also interesting that Schino's data shows sex ratiod to be more biased in favour of females when sex dimorphism is greater (males being larger) - a prediction of both the Fisher and Trivers-Willard models of sex allocation.

Having said that local resource competition with matrilineal resource inheritance is a special case of condition-dependent sex allocation, in which it is the purportedly less fitness-variable sex that requires the larger investment (or benefits more from it), and is hence underproduced, it is interesting to note that local resource competition is also a special case of Fisher's Principle. In the case outlined above, we expect a male-biased sex ratio because males are less costly to the mother (Clark 1978). That is, because a smaller investment is made in males, they are produced in greater numbers - this is Fisher's prediction exactly.

The interesting question, now that we've shown that local resource competition is a subset of both the set of cases obeying the assumptions of Fisher's Principle *and* of condition dependent sex allocation, is whether it is possible to unify the two theories; in other words, accommodate one within the other. Below, I shall describe a model by that extends conditional sex allocation to include cases where continued inheritance of condition is more important than immediate fitness benefits from higher investment in males.

1.20 Evidence in humans

Having discussed the putative mechanism in humans and hence two of the potential correlates of sex (high blood sugar and low temperature for survival of male conceptuses and to term, respectively, Cameron *et al.* 2008, Catalano *et al.* 2008), I'd like to point out some of the other patterns that we have evidence of. Clearly, we have more evidence about the sex allocation patterns of humans than any other species. As might be expected of such a complex species, the patterns that emerge are far from simple.

The best supported hypothesis is differential provisioning. Several anthropologists have sought to demonstrate condition-dependent sex allocation by selecting ethnic groups or social strata that are at the bottom or top of a society, and then showing that sexes benefit from additional investment to different degrees, and that parents allocate more resources towards one sex. Among the studies that show biased investment is Cronk's 2000 study of the Mukogodo people, a hunter-gatherer tribe who recently adopted pastoralism. Cronk found that they are investing more parental care in daughters than sons. This is likely because these sons cannot compete with the dowry payments of males from wealthier tribes (those already more established in the pastoralist lifestyle).

Bereczkei and Dunbar (1997) criticised that previous studies had only demonstrated either that the allocation of resources was skewed towards one sex, or that one sex benefited more from a given level of investment than the other, but never both for one study population. In their own study of Hungarian Gypsies, Bereczkei and Dunbar were able to demonstrate both an increased investment towards girls, and an adaptive rationale in terms of "marrying up".

Correlational studies on human populations have shown that engineers have more sons, while nurses have more daughters (Kanazawa and Vandermassen 2005) and violent men have more sons (Kanazawa 2006), but beautiful parents of either sex tend to have daughters (Kanazawa 2007) - all apparent examples of condition-dependent sex allocation. It is intriguing that Kanazawa's data stem from developed countries where infanticide would be diligently prosecuted, suggesting his data show deviations in primary sex ratios, whereas those of Cronk and others are based on differential investment post partum. Interestingly, according to an account given by Parkes (1971) of a book by Starkweather (1883), the latter proposed a vague and exclusively verbal theory according to which attractive males should sire daughters. He seems to say less on the subject of what beautiful women should beget, at least according to excerpts presented by Parkes. Normark (2004) may have the clearest evidence on human phenomena, in a study that shows children conceived by women who were cohabiting with a male partner to have a higher sex ratio (more boys). Normark relates this to the lower likelihood of extra-pair paternity in cohabiting couples, although her data affords no conclusive (genetic) test of this.

James (1987a) has reviewed a wealth of studies showing various other correlations, including smoking, oral contraceptives, infectious disease, and various cancers. The consensus from his work is that adverse conditions favour female bias, which may of course be nothing but a re-statement of the fact that females are the default sex in humans. After all, the strength of selection on the sex ratio depends on the size of local populations, or, conversely, on typical migration distances (both are accounted for in the metric known as effective population size).

Another widely publicised correlation is that of periods of war coinciding with an increase in male births (reviewed in James 1987a). Manning *et al.* (1997) have suggested that this may be due to a concurrent increase in marriages between young wives and older husbands, an explanation that could also explain findings from three industrial nations presented by Mueller (1993), that the socioeconomic elite produces offspring with a higher sex ratio than other sections of society. Manning *et al.*'s result only includes cohorts briefly before the first until just shortly after the second world war (1911-1952), but Mueller (1993)'s finding stands on a wider footing, including three cohorts starting 1789 (Britain), 1830 (Germany), and 1860 (USA), with the last of the cohort being born in 1925, 1939 and 1939 respectively. All of these have sex ratios > 1.1, significantly exceeding the background sex ratios of around 1.06 in each case. Note that this is in agreement with Kanazawa's result suggesting the professional class has more boys (Kanazawa and Vandermassen 2005), and with Bereczkei and Dunbar's suggestion that girls marry up Bereczkei and Dunbar (1997), as well as Voland's finding that female infant survival was highest in the lower classes in 17th century Germany (Voland 1988). However, the relationship between social class and sex ratio was drawn into question in a review paper by James (1987a), citing two empirical studies with very large datasets (five million and one million) that failed to find any significant trend. James (1987a) concludes that such trends seem to be restricted to the uppermost end of the socioeconomic gradient.

Clearly, a coherent picture of the physiological mechanisms will be needed to clarify among these seemingly contradicting correlational findings. Cameron *et al.* (2008) and Catalano *et al.* (2008) have played a promising overture.

1.21 Leimar and conceptual synthesis

Note that some of the extensions of the original condition-dependent sex allocation model do not generalise to some related cases, such as sex changers (Charnov and Bull 1989). In the original model, it was suggested that the variance in quality was, as at least in ungulates is seems to be, inherited through the mother. This asymmetry violates implicit assumptions of some of the early models, leading not to the established threshold behaviour (Frank and Swingland 1988), but a situation where some mothers when in good condition will produce daughters (Leimar 1996), which we can paraphrase as putting the money in the bank to wait for interest before making a purchase. The daughters will eventually produce sons, who will be among the very best males in the population, outweighing the possible fitness gains sacrificed in (the) previous generation(s). Leimar's conclusion only holds when the condition of offspring depends on the less fitness-variable sex. That is, the conclusion holds when offspring condition depends on their mothers and males are the more variable sex (as is the case in most mammals and birds), or when offspring condition depends on fathers and males are less variable (likely true for several marine fish, such as the pipefish (Berglund 1994)). Furthermore, the sex ratio control must rest with the offspring or the less variable parent. As it is very likely that the sex making the greater investment is also more constrained in its reproductive potential, i.e. less variable, Leimar's conclusions may widely apply.

We can see, then, that in the classic scenario envisaged by Trivers and Willard (1973), condition is mainly passed on through the mother (less variable sex) to offspring of both sexes, while the conclusions generalise to cases where the (esp. genetic) condition of the father is (more variable sex) the determinant of the bias in sex ratio. Leimar's 1996 scenario is a further subset, in which female condition is preserved along maternal lineages, but lost when sons are produced. Wild and West (2007) have included Leimar's concerns in a model by letting mothers' nutritional status depend on the food quality in the habitat patch they live on, and assuming complete male dispersal, with some proportion $1 - d$ of daughters remaining on their mother's patch, with patch quality unaffected. It's furthermore interesting in that it is not maternal condition that is inherited by daughters, but, d willing, a proportion of the mother's patch. This avoids some of the problems encountered in other models where a maternal or genetic component can rise 100% frequency in the population. However, the central interest of Wild and West is showing under what conditions patterns of local resource competition are expected to be observable versus those of condition-dependent sex allocation. Note also that because the model is set up to allow only one sex to avoid dispersal, it is not a symmetric model (with the obvious exception where $d = 1$).

The general expectation in simple condition-dependent sex allocation models is, of course, that sons are produced by mothers on high quality patches (type 1) when the competitive ability of males born on patch type 1 is sufficiently greater than that of males born on patch type 2, and vice versa, when the competitive advantage of daughters born on patch type 1 is relatively small. Both local resource competition (Clark 1978) and strong inheritance of patch occupancy (and hence quality (Leimar 1996)) down the maternal lineage would predict a bias towards daughters on high quality patches, and vice versa for low quality, while producing more males in total (males being defined as the sex that do not compete for resources on their mother's patch).

These expectations are generally born out by the results of Wild's and West's study. However, the switching between male production and female production is not always gradual. Rather, there are regions of the parameter space where the stable strategy is to produce all males on one patch type, and all females on the other, mimicking the threshold behaviour of early condition-dependent sex allocation models presented by Bull, Charnov and Frank. The size and position of this region of parameter space depends on the magnitude of the fitness "advantage" (*sensu* Wild and West) of one sex over the other, that is, $\frac{w_{m,1}/w_{m,2}}{w_{f,1}/w_{f,2}}$ where $w_{s,i}$ is the competitive ability of individuals of sex s on patches of type i. For instance, when this parameter is appreciably larger than 1, no males are produced on patch type 2 when female dispersal is high. When the "advantage" is closer to 1, no females are produced over some of the same region of parameter space, again for patch type 2. This represents the two cases that were exhaustively numerically investigated by Wild and West. This model could be expanded to include possible paternal effects, as well as the effects of autosomal loci under natural selection. I will present a simple investigation of genetic implications later in this thesis. Suffice to say here that Wild and West have also suggested the utility of using a population genetic approach to simplify analysis, although this does require the frequency of recombination between the loci determining different traits to be explicitly defined, as well as the definition of one or several genetic architectures for the behaviour, which can be troublesome when there are different ways to parameterise the model.

1.22 Role of sex ratios in the mechanism of evolution, and behaviour

There has been a lot of interest in recent years in understanding the consequences of variation in dominance status, mate quality, and quality of environment, amongst others. There has also been interest in distinguishing between the good genes and sexy sons hypotheses (Kokko *et al.* 2002, see Radwan 2002 for a brief review), and some of the evidence in this debate has come from the previously mentioned studies. There has also been interest in determining the upper limit of the rate of adaptation of natural populations (e.g. Hill and Robertson 1966, Barton 1995, Barton and Partridge 2000).

Other groups have attempted to understand the evolution of mating systems (Shuster and Wade 2003), sex determination mechanisms (Bull 1983), and the consequences of sexually antagonistic loci (e.g. Arnqvist and Rowe 2005). Sexual dimorphism of multicellular organisms (as opposed to gametes) has been reviewed, for instance, by Lande (1980) and Andersson (1994).

I'll be referring back to the sexy sons hypothesis occasionally in later chapters.

1.23 Sexually antagonistic effects

Sexually antagonistic effects are factors whose effect on males and females differs. This includes sexually antagonistic alleles, which may benefit males and harm females, or vice versa.

Sexually antagonistic effects may arise from a number of circumstances. The resource requirements of males and females may differ, especially where either sex has specialised sexual organs. If the distribution of these resources in the environment is heterogeneous, then we may expect different patches in the environment to produce different sex ratios. There may be social factors in species with parental care - some females may be better equipped to raise sons, others daughters. Finally, it has been observed that sexually antagonistic loci (henceforth SAL) do segregate in *Drosophila* (Chippindale *et al.* 2001, Rand *et al.* 2001).

Siller and Agrawal (both 2001) have suggested that sexual species may have an advantage because sexual selection can speed up rates of evolution. It is not clear what role SAL would play in such a scenario, since during their transition from unregulated to sex-specific expression, they can markedly decrease the fitness of one sex - if the females are worse affected (or in hermaphrodites), this can lower the growth rate of a population.

1.24 Mating system and variance

In order for some males to reach extreme fitness, the mating system of a population needs to be polygynous (it can be polygamous, but polygyny needs to be a component). Therefore male fitness variance is limited by how polygynous the mating system they are in.

Similarly, when antagonistic loci are segregating, the optimal sex ratios show more variance as time progresses. This could be used as a diagnostic in selecting model species selection to study sexually antagonistic loci and the evolution or degeneration of sex chromosomes. Translocation of SAL can also affect this deviation.

1.25 Fitness as a discrete or continuous variable

The simplest way to measure fitness is to simply count the number of offspring of an individual. This number will always be an integer. However, this is a narrow view of fitness that only takes into account a single generation. As of the F2 generation, different haplotype blocks may suffer different fitness fates, and the relatedness with any grandchildren is no longer deterministic, as evident in Mendel's Second Law. This variation is not, usually (except in cases of meiotic drive), a reflection of actual fitness as defined by selection, but rather, a component of genetic drift. Such variation would not, therefore, enter into estimating an expected fitness value for an individual. A more inclusive view of fitness was given by Hamilton in 1964, termed *inclusive fitness.* Hamilton pointed out that copies of an individual's genes are also present in related individuals. This is relevant when the behaviour of the focal individual influences the survival of these alternate copies of his or her genes (kin selection). Taking this into consideration, fitness could become a fractional value, with $\frac{1}{2}$ added for every child, $\frac{1}{4}$ for every nephew or niece, and so on. Typically, we would normalise these values to give a population average of 1, assuming stable population size. Populations are rarely stable in size, however. Populations can expand and contract, sometimes randomly, sometimes in cycles, depending on the ecology of the species (Begon *et al.* 1986, p. 559-563). We initially said that fitness could be represented in integers; however, this is not true if we wish to correct for predictable demographic fluctuations. Fisher (1930, p. 82) pointed out that the probability of future survival of an allele depends on the imminent demographic events in the population. In Fisher's simple scenario, the probability of survival increases when the population is growing, while it decreases when the population contracts. This is a simple application of the concept of effective population size. An alternative view of this situation is to say that the fitness of alleles also depends on such demographic factors. Of course, the number of copies of an allele in a given population is, again, an integer, *except* when we are trying to *predict* future fitness, in which case, assuming a changing population size becomes yet another factor that might render such a fitness estimate to be a fractional value. Whether one should control for population size depends largely on the intended application of the calculation, the details of which need not be discussed here.

In any case, it is clear that while observed fitness values are constrained to be integers, the factors causing fitness may be measurable experimentally at a finer scale. For instance, male attractiveness could be scored by assessing the reactions of a large number of females. This example has yet its own caveats and pitfalls, which there is no further merit in discussing here. The message is simply that we could devise methods for measuring significant aspects of phenotype at a fine scale, and hence approximate the actual fitness potential of an individual to an increasingly accurate extent (but no doubt conforming to the principle of diminishing returns).

In fact, both the confinement of fitness to be expressed in integer values, and the independent segregation of mother's and father's haplotype blocks in the formation of the F2 generation, are two sources of genetic drift (among others that occur at various stages of the life cycle, most notably chance event such as encountering a predator or mate), and as such, should not be taken into account when retrospectively estimating fitness. On the other hand, when simulating forwards in time, it is useful to be aware how they need to be interpreted.

The analogy between natural selection and artificial selection has not only been important in making a compelling case for the former in Darwin's time; it has also been an important tool in understanding what may happen when nature favours the reproduction of some individuals over others. Quantitative genetics is a field that was developed in order to understand how we can become more efficient artificial selectors. Among many other useful techniques, quantitative genetics has a metric known as breeding value. Breeding value is the expected phenotypic value for the offspring of a given individual, assuming random mating, as is often done when considering natural populations. Estimating breeding values is based on knowing the genotype of an individual at one or several loci, and knowing how the alleles it carries contribute to its phenotype.

When the frequency of the A1 allele is p and the frequency of A2 q=1-p, and where the difference in phenotype between A1A1 individuals and A2A2 individuals is $2a$, and the A1A2 heterozygotes have a phenotype $a + d$ higher than A2A2, d absorbing any dominance effect that may be present, the breeding values are (Falconer and Mackay 1996, pp. 110-115):

genotype	breeding value
A1A1	$2q\alpha$
A1A2	$(2q-1)\alpha$
A2A2	$(2q-2)\alpha$

where $\alpha = a + d(2q-1)$.

This metric, the breeding value, has several convenient properties, partly based on the assumptions typically made about it. In artificial breeding, the effects of alleles remain the same through time (this is assuming the absence of frequency-dependent selection and interactions between loci, known as epistasis). However, the allele frequencies can change, so breeding values also change through time, as, typically, the benefit of being of the preferred genotype declines as it becomes more common.

The simplest way to measure fitness is to simply count the number of offspring of an individual. This number will always be an integer. However, this is a narrow view of fitness that only takes into account a single generation. As of the F2 generation, different haplotype blocks may suffer different fitness fates. One way to allow for this would establish the correlation between an indicator of quality and average RS; for instance, in the wasp egg problem, one might establish how viability varies with egg size, and then multiply this function with the size distribution of given individuals' eggs. However, the extent to which this is genetic or due to maternal effects (genetic and otherwise) will influence the observed sex ratio (Leimar 1996). Also, this correlation changes as selection erodes variation in fitness-related traits, and is thus of very short utility relative to the timescale of any longitudinal study, although scaling laws may apply. A better way to proceed may be ancestor reconstruction, which can lead to fairly precise estimates of the fitness effects of different haplotype blocks. Note, however, that such an analysis cannot give estimates of historical fitness distributions without additional assumptions, as there is no information about extinct haplotype blocks. Typically, one would assume constant population size. In this line, Visscher *et al.* (2006) have presented an approach that uses marker information to establish the "true" relatedness of individuals that are not parents and offspring of each other (the only case where genetic similarity has an exact, constant value). Visscher *et al.* estimate heritability based on haplotype information in a method that works well for organisms with a small genome, when a large sample has been geno- and phenotyped, including many related individuals).

Trivially, artificial selection provides a beautiful model system for testing effects of variable fitness on mating success, because whatever trait is being selected, assuming an efficient selection strategy on the part of the animal breeders, becomes closely correlated with fitness.

In the next chapter, we'll find out more about the meaning of fitness variance, and how it can be increased by properties of the mating system, without any natural selection involved.

Chapter 2

Neutral model of fitness variance and fertilisation probability

2.1 Introduction

Since I will be dealing with fitness variance in detail in chapter 3, it is fitting that I should give a discussion of the relevance of fitness variance deriving from a variety of sources in this preceding chapter.

Fitness in biology is typically defined as the number of offspring that an individual produces between its becoming sexually mature, and its death, and who in turn survive to sexual maturity. Differences in fitness variance between the sexes underlie a number of theories in evolutionary biology, such as mating systems (Shuster and Wade 2003) and condition-dependent sex allocation (Trivers and Willard 1973, Frank 1987, Frank and Swingland 1988), and are integral to models of sexual selection (reviewed in Andersson 1994). Since Bateman (1948) demonstrated that in a competitive situation, females have more constant fitness than males, and argued that this was because of intrasexual selection, research has remained focused on male competition and female choice as the factors causing such differences in fitness variance (Trivers 1972, Trivers and Willard 1973, Shuster and Wade 2003). However, Sutherland (1985) proposed a model in which a sex difference in mating success resulted from chance encounters of mates, and used this to reanalyse Bateman's data. Sutherland's analysis showed that Bateman had not, in fact, succeeded in showing female choice at all, since the variance in mating success among males in his data did not significantly differ from that expected due to random encounters.

This paper extends Sutherland's insight by applying a similar logic to broadcast spawners and wind pollinating species, where the random encounters are not between individuals, but between gametes. The model shows that the sex producing more gametes per individual will have greater fitness variance, all other things being equal. A crucial assumption in the model is the neutrality of gametes, that is, all gametes have the same initial probability of fusing with a gamete of opposite sex (cf. Hubbell 2001).

Trivially, if population size is constant, and each sex is producing only the replacement number of gametes, and fertilisation is ensured, no variance in fitness will be observed, as each individual will be a genetic parent to exactly two offspring. This model would necessitate the absence of mortality, which, besides perfect fertilisation success, is another unrealistic assumption. However, this simple case serves to illustrate the idea in this paper, which can be roughly paraphrased as, "the fewer gametes are produced by one sex, the smaller the fitness variance of that sex".

2.2 Model

Suppose we have a large container in which individual organisms place their gametes for random mixing. Assume the gametes are of two types, large and small, and that each individual will only produce one type of gamete. Denote the number of small gametes by n_S and the number of large gametes by n_L. Let the number of males (individuals producing small gametes) in the population be N_m and the number of females (individuals producing large gametes) N_f. Assume that all individuals of each sex produce the same number of gametes, and that all gametes are equal. Assume that the size of the container is such that each gamete collides once and only once with another gamete; if the gametes are of different kind, the collision results in fertilisation, and the gametes henceforth act as one with respect to further collisions. The probability that a large gamete remains unfertilised is $\frac{n_L-1}{n_L+n_S-1}$; similarly, the probability that a small gamete remains unfused is $\frac{n_S-1}{n_L+n_S-1}$. If n_L is small and n_S large, these probabilities approximate $\frac{n_L}{n_S}$ and $\frac{n_S}{n_L+n_S}$, which approach 0 and 1, respectively. The model thus accurately captures the trivial expectation that all large gametes get fertilised, while the vast majority of small gametes do not.

Strictly speaking, the number of fertilisation events per individual follows a variant of the hypergeometric distribution. However, I shall continue to assume that n_L is small and n_S large, which means that the difference between a model with replacement of the fused gametes, and one without replacement (which is the more accurate model) becomes negligible, since the number of fused gametes can never exceed the number of large gametes, which we assumed to be small. Let the number of fertilisation events per individual thus be approximated by a Binomial distribution parameterised with $p_f = \frac{n_L-1}{n_L+n_S-1}$ and $n_f = \frac{n_L}{N_f}$ for females, and $p_m = \frac{n_S-1}{n_L+n_S-1}$ and $n_m = \frac{n_S}{N_m}$ for males. This distribution describes the number of successful fertilisation events, which I shall assume to be linearly proportional to fitness (number of individuals surviving to reproductive age). Hence, the variance of the distribution describes fitness variance, and can be written as

$$\frac{n_S}{N_m}\frac{n_S-1}{n_L+n_S-1}(1-\frac{n_S-1}{n_L+n_S-1}) \tag{2.1}$$

for males, and

$$\frac{n_L}{N_f}\frac{n_L-1}{n_L+n_S-1}(1-\frac{n_L-1}{n_L+n_S-1}) \tag{2.2}$$

for females.

Assuming again that n_L is small and n_S large, the above can be rewritten as

$$\frac{n_S}{N_m}(1-\frac{n_S}{n_L+n_S}) \tag{2.3}$$

for males, and

$$\frac{n_L}{N_f}\frac{n_L-1}{n_S} \tag{2.4}$$

for females.

If we assume that n_L is small relative to n_S, the ratio of male fitness variance to female fitness variance simplifies to $\frac{{n_S}^2 N_f}{n_L(n_L-1)N_m}(1-\frac{n_S}{n_L+n_S})$. If we also assume a 1:1 sex ratio, N_f and N_m cancel, and the expression simplifies to $\frac{{n_S}^2}{n_L(n_L-1)}(1-\frac{n_S}{n_L+n_S})$.

There may be cases of broadcast spawners that are so rare and have so dispersed a distribution that the probability that each gamete collides is significantly less than 1. In these cases, it is sensible to assume that the overall number of gametes of either size is large enough that the number of collisions per gamete is relatively constant, and to restrict our argument to gametes that do collide, in which case, the above exposition is once again correct.

The data in 2.1 was generated from a simple Mathematica script, the core of which is

```
GamsPerMale = 2;
malenum = 50;
MaleGametes = Table[0, {malenum}, {GamsPerMale}];
femalenum = 50; GamsPerFemale = 2;
For[a = 0, a < (femalenum*GamsPerFemale), a++,
```

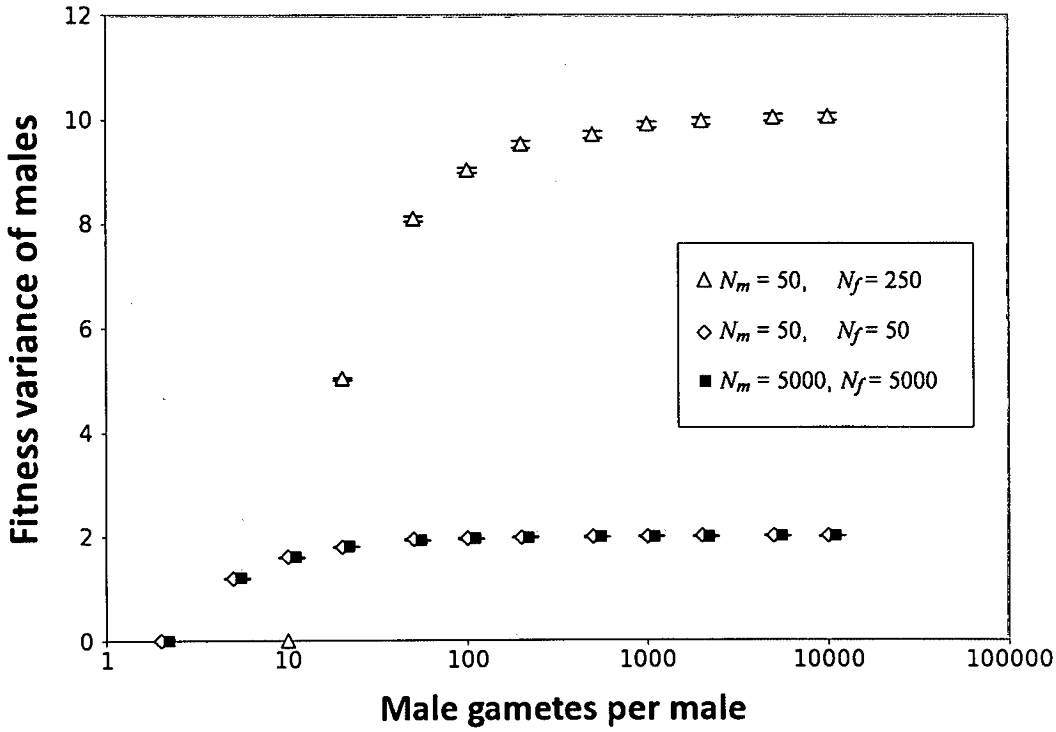

Figure 2.1: Fitness variance of males against number of male gametes produced, assuming females produce a replacement number of gametes. The white diamonds have been displaced to the side to avoid complete overlap with the black blocks.

```
   idx1 = Random[Integer, malenum - 1] + 1;
   idx2 = Random[Integer, GamsPerMale - 1] + 1;
   If[MaleGametes[[idx1, idx2]] == 0, MaleGametes[[idx1, idx2]] = 1,
   a--]
];
sums = {};
For[i = 1, i <= malenum, i++,
 AppendTo[sums, Total[MaleGametes[[i]]]]
 ];
Variance[sums] // N
```

2.3 Results and discussion

I have shown that sex differences in fitness variance can arise from random sampling processes, using entirely neutral assumptions. I have assumed that all gametes are dispersed into one large pool, or "container". What kinds of species is this model realistic for? Clearly, the argument fits broadcast-spawning animals,

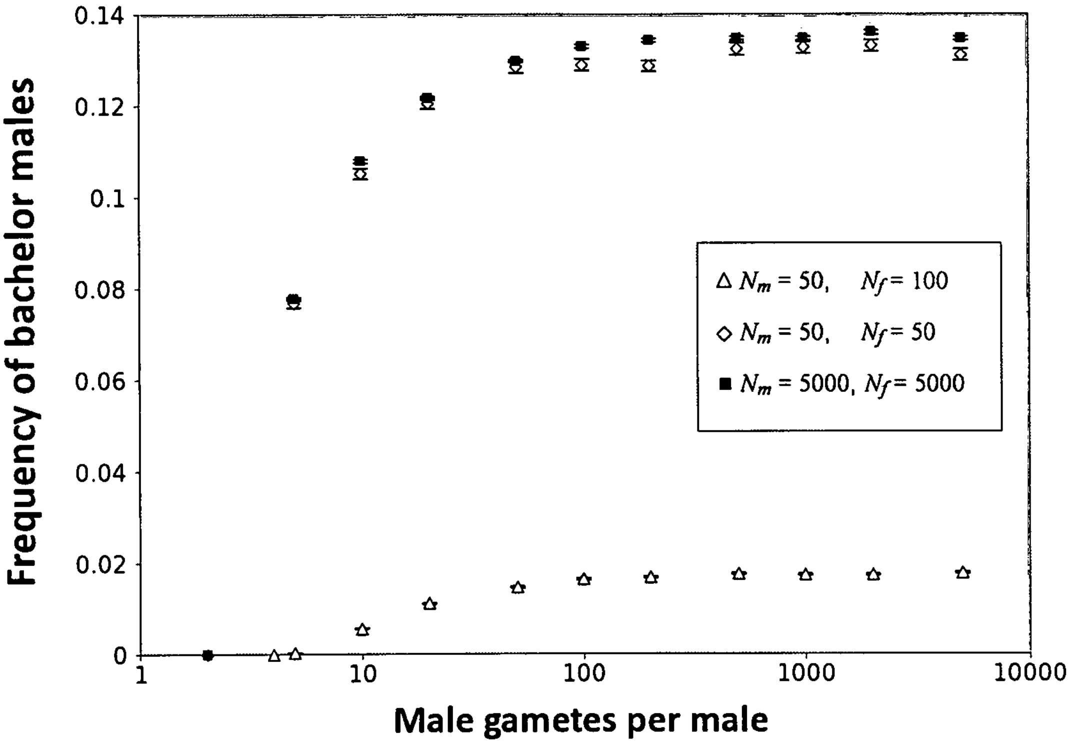

Figure 2.2: Frequency of bachelor males against number of gametes produced.

particularly sessile animals and others who have no explicit mating behaviour in which the male and female release their gametes in close proximity. It also applies equally well to wind-pollinating plants, and possibly to plants who follow the ancient habit of releasing their sperm from a specialised gametophyte stage into a film of water, such as ferns. The model will also fit those insect-pollinated plants in whom each flower is visited by many insects, and where the average insect has already visited many other flowers previously. It will particularly fit species where the male flowers are visited first, and female flowers later (or where the stamens develop before the stigmata). The model may, however, fail for plants that can self-fertilise and where many flowers are on the same plant, and visited by insects sequentially.

We can illustrate exactly how internally fertilising organisms deviate from the proposed model. Imagine a population of animals in which each male mates each female exactly once, and releases exactly the same amount of sperm each time. Imagine that semen is mixed up inside the female such that the order of insemination has no influence on probability of paternity. Alternatively, we can assume that mating occurs in random order, and a particular ejaculate (perhaps the first or second to be delivered) always wins, but results in only a single

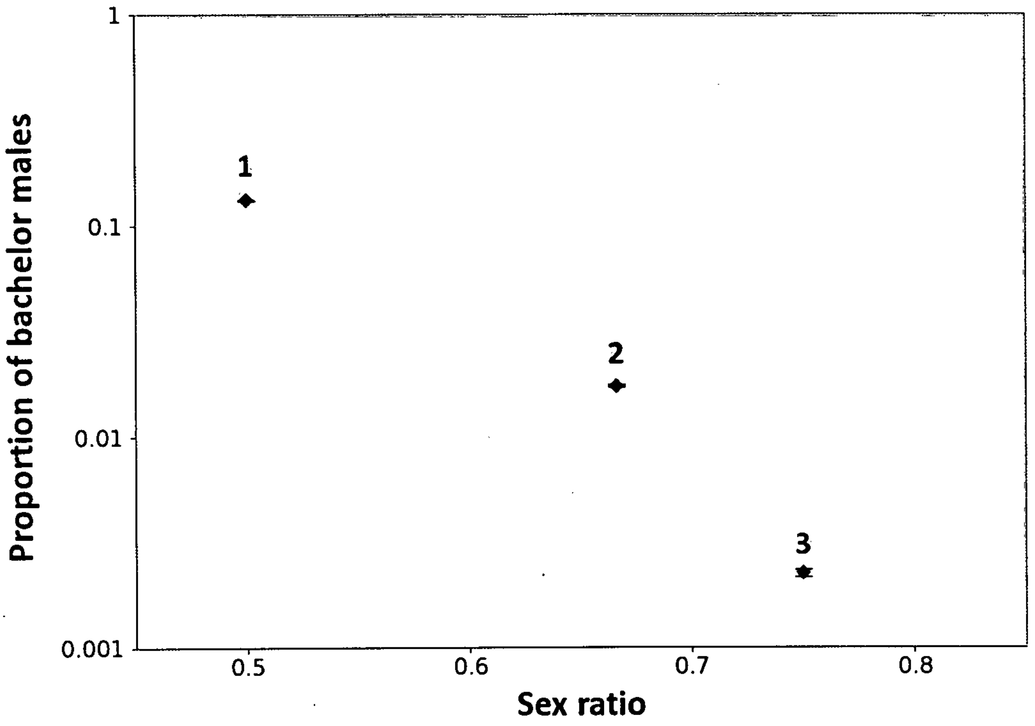

Figure 2.3: Proportion of bachelor males quickly declines as the number of females (or number of female gametes) increases. Proportion of bachelor males is plotted against the sex ratio here. Numbers of female-equivalents per male are indicated above each data point. Accordingly, females produced two gametes each, males 1000 (see Figure 2.2 to confirm this is at the plateau for numbers of bachelor males). Population sizes are 100, 150, and 200 respectively. Error bars indicate standard error. Data from 5,000 replicates.

fertilisation, so that further matings are necessary for subsequent fertilisation events. In these cases, the model will behave nearly as before, with a female variance of 0, and a negligibly increased male variance because use of a male's sperm in one female cannot influence success of the same male's sperm in another female.

How then does behaviour in real species differ from this mathematical ideal? We've seen that in internally fertilising species, each female is a separate "container" in which collisions between a set number of male and female gametes take place. We can make several observations about mating behaviour in real populations:

- It is rare that all females mate each male in the population. The population would have to be small for this to be possible.
- Of those males with whom a given female does mate, she will typically mate some more often than others.
- Males differ in mean ejaculate size.
- Each male is likely to give ejaculates of different size in each given mating, due to complex factors, e.g. assessment of the female's quality, assessment of the female's promiscuity, dividing up his chances among an expected number of matings with no excess (e.g. Simmons and Kvarnemo 1997, Gage 1998, Reinhardt and Arlt 2003, Oliver and Cordero 2007).

As a rule, however, we can say that unless the species is tending towards monogamy (where reproductive variance is low), the subdivision of the original whole-population container introduced by internal fertilisation will produce some additional fitness variance among males (because males cannot now fertilise females they have not explicitly mated). This is also clear if we consider the fact that this model describes a layer that is additional to Sutherland's encounter model, which already predicts increased fitness variance for males.

Lower male fitness variance than predicted by the model is hence expected when females are monogamous, and the number of males is not significantly greater than the number of females. If the number of males is greater, there is of course a sampling effect that will raise male fitness variance to significantly above female variance. Another factor that can lower male fitness variance is spatial structure. If habitat quality does not vary, we would expect individuals to be evenly spread out across the habitat. Both sexes will tend to avoid resource competition with other members of the population, but males will also try to avoid competition with each other, and hence overdisperse. Females may then also disperse more evenly to avoid competition with the now evenly spread males. In reality, this will be a more gradual, almost tit-for-tat process than illustrated here (and there are other factors, such as birth locality and inheritance of territory, as well as resource heterogeneity). Shuster and Wade (2003) have shown in great detail how features of the environment can select for particular mating systems. The model outlined in this paper, however, assumes free mixing of the individuals in the population (panmixis), an assumption that is broken by non-random spatial structure. The spatial structure I have just proposed will tend to reduce male fitness variance, as all males have similarly restricted access to females, putting an upper limit on the number of gametes they can contribute to the next generation. However, the assumption that habitat quality is constant is itself unrealistic, and a patchy habitat will tend to increase male fitness variance.

Fitness variance reaches a plateau at about 100 gametes per individual at even sex ratio, and somewhat later for species with more female-biased ratios. These numbers are small relative to the gametes produced by males of most species, but large relative to female numbers, suggesting that these results may be more usefully applied to females. To put these gamete numbers into a more precise context, it may be useful to refer to the World Health Organization's definition of a normal human ejaculate, which they give as containing 4×10^7 spermatozoa (Organization 2003). It is clear that, since males produce far in excess of 5,000 gametes each, there will be no differences in fitness variance among any number of broadcast-spawning or wind-pollinating species.

It is useful, however, that the fitness variance due to this neutral effect has this asymptotic behaviour, because this means that while an excess of male fitness variance can't be taken as evidence of non-neutral mechanisms such as that described by Trivers and Willard (1973), such mechanisms will still be detectable in those species in whom male fitness variance exceeds that achievable by neutral effects.

Sutherland (1985) gave an argument in terms of random encounters. This again relied on females having reproductive assurance, and a smaller number of gametes. This work falls under the same general framework as that presented here, there being a basic equivalence between gamete numbers and rate of movement. With female reproductive assurance and random fertilisation, the rate of movement of males relative to the distance between females determines an upper limit on the number of females they can fertilise, in the same way that the number of gametes produced limits the number of fertilisation successes, showing a basic mathematical equivalence.

It is worth putting the effect described in this paper in the context of two other effects. I shall avoid the much larger issue of why sex evolved, and whether variation in propagule size preceded the number of mating types converging on two, and simply note here that studies have shown that mutations that lead to production of more numerous smaller gametes can spread, and in turn create selection for a class of gametes that is large (Parker *et al.* 1972, Hoekstra 1987, Bulmer and Parker 2002). This aspect is known as the evolution of anisogamy, meaning unequal gametes. Once two different size classes of gametes had evolved that could only produce viable offspring by each combining with its opposite, the fitness variance of individuals producing small gametes would be increased as described in this paper. It is important to note that this is different from an effect that has been understood in various levels of detail for a long time, namely that genetic variation increases when chromosomes recombine (e.g. Weismann 1889, Burt 2000, Rice and Chippindale 2001). The increase of genetic variation arises only from non-neutral assumptions (different alleles at two loci or more), and will only increase fitness variance when there is selection for one allele or the other, and they are not at some equilibrium (such as in frequency-dependent kinds of selection). The effect I have described in this paper is specific to anisogamous species; the increase in genetic variation described by Weismann and subsequent authors affects all sexual organisms, even if "male" and "female" gametes are the same size.

It is also worth noting that my result suggests a further inherent asymmetry between the sexes. It has previously been noted that the certainty of being the true parent of an offspring is often much lower for the male parent. I have shown here that males also inherently have greater fitness variance, even if all males are equally attractive partners - except in cases of strict monogamy (as opposed to social monogamy). The fact that males will tend to be more variable may be useful in explaining that polygyny is more common in higher taxa, and that males tend to be larger, keeping in mind that natural selection favours, in the long term, a maximisation of the geometric mean of the number of offspring in subsequent generations of a lineage (A. Gardner, pers. comm.).

Chapter 3

Condition-dependent sex allocation when condition and fitness are continuously distributed

3.1 Introduction

3.1.1 Definitions

Two definitions are necessary for this chapter, of *fitness* and *condition*. I have discussed various definitions of fitness in section 1.25. Fitness can be most conveniently defined in terms of the change of gene copy number between a particular life cycle stage in one generation, and the same stage in the following generation. Note that this makes sense in mathematical terms even in non-genetic models (e.g. Stubblefield 1980). For the purpose of this chapter, assuming this life cycle stage to be the entry into sexual maturity is as convenient as any other, and is what I shall proceed with. I will also use the term *fitness variance* to refer to the statistical variance in this fitness among members of one sex only, either males or females, except where explicitly stated otherwise.

I use a very broad definition of condition that encompasses all the kinds of traits and modes of inheritance that the mathematical model here described can be applied to. The *condition* of an offspring is the set of factors that have a bearing on its fitness (but, to avoid circularity, explicitly excluding its own sex and the population sex ratio). Condition will include where an offspring is born, how much food it receives, its genotype, and whether its father is providing "indirect benefits", that is, defending a territory for its mother, or providing food.

3.1.2 Aims

In this chapter, I will present a framework for analysing condition-dependent sex allocation (also known as the Trivers-Willard scenario, hypothesis, or model) primarily developed by James Bull, Eric Charnov, and Steve Frank. I will present a new way to parameterise the model that reduces complexity and increases convenience, by allowing trait values for both sexes to be drawn from distributions that are members of distribution families, with known parameter values. I will discuss the suitability of several statistical distributions for this problem, and show how one can derive two instances of the Gamma distribution with a known ratio of variances and constant mean. I will discuss the meaning of fitness variance in the model, and, building on results from the previous chapter, explain why it has little utility in empirical work. Finally, I will present numerical results to show the magnitude of sex ratio deviations we should expect, and discuss these in light of recent reviews and hypotheses of empirical sex ratio data.

3.2 Significance of fitness variance

The theory of condition-dependent sex allocation as originally formulated by Trivers and Willard (1973) posits that the optimal sex ratio is a function of the variance in fitness of the two sexes. This prediction about variance has not, to my knowledge, actually been used as a test on empirical data, and I will explain why it is not a reliable prediction, and therefore should not be used. It is seductive in this context that fitness is a quantity we can directly measure, so at first glance, it should be possible to identify instances of condition-dependent sex allocation by measuring variance in fitness in natural populations. Unfortunately, in this model of dioecious individuals (those that have only one sex), only the fitness of one sex is ever revealed to the observer for any given individual. We cannot know what the fitness of that particular individual would have been, had it been born as the other sex. This problem extends to sequential hermaphrodites, who display the same kind of threshold behaviour (Charnov 1979b, Allsop and West 2003a), where once an individual has switched sex, its fitness had it continued as the previous sex will never be known, or, equivalently, the fitness of individuals if they were the later sex is not known for young individuals that haven't switched yet.

An additional layer of complexity arises from the fact that we are not simply talking about one distribution that is cut into two by some threshold (see chapter 3 and Figure 1.1), so that we could see that the variance among individuals

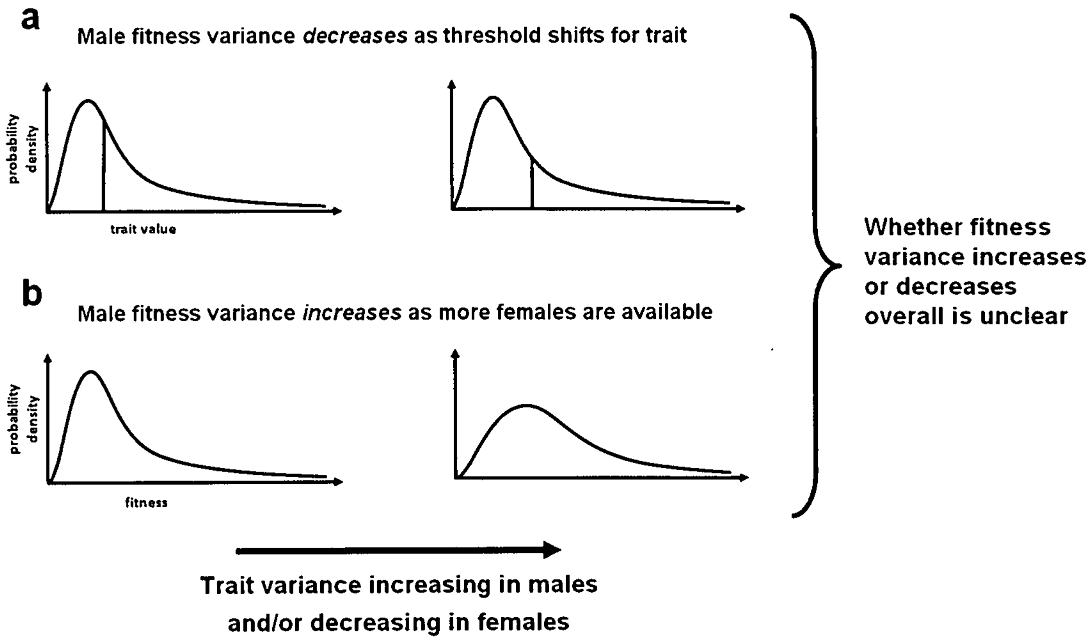

Figure 3.1: While condition-dependent sex allocation can be caused by a sex difference in variance for some trait that affects fitness, it is not clear that this difference in variance will be detectable in the same population after the sexes have been thus expressed. This is because establishing a threshold above which individuals will be of one sex only, and below which, of the other (see Fig. 1.1), has two opposing effects on the eventual fitness variance, illustrated here as (a) and (b). Firstly, (a) a shift of the threshold to the right (or equivalently, establishment of a threshold where previously there was none) will reduce the variance since the area above the threshold is only a part of the distribution, and will contain less variation (although variance may increase, depending on the exact properties of the distribution, see main text). However, the variance of the sex above the threshold is (b) simultaneously increased because that sex' contribution to the next generation is now divided among fewer individuals.

above the threshold is not larger than of individuals below. Note that sex ratio is a frequency-dependent game, where the fitness of individuals, say, above the threshold, is modified by the actual sex ratio. Unfortunately, this is a case where the fitness variance increases as the sex becomes rarer (Fig. 3.1); that is, as the proportion of the variance in the trait that is allocated to one sex decreases, and so variance in that trait also decreases, the variance in fitness derived from any constant subset of that variance, increases, so that it is not clear whether fitness variance increases or decreases or remains constant overall when the threshold is shifted.

It is easy to see that if sex expression were random, the increase in variance would be as $1/P_m$, but this exact predictability is lost here because with the threshold model, a small change in sex ratio could mean a large shift in the position of the threshold, all depending on the exact distribution of z, and the ratio of the variances of the distributions of ζ_m and ζ_f.

Figure 3.2a shows an example where in spite of the scaling of fitness effected by the threshold, and resulting increase in fitness variance (illustrated in Figure 1.1), the sex produced in better condition would show lower variance in fitness than the sex that due to its *a priori* smaller variance in fitness is being produced when conditions are less favourable. These distributions are known as negatively skewed. These are typical distributions for extremely k-selected and monogamous species. This typically applies to species that are widely dispersed across their habitat (Shuster and Wade 2003) and engage in brood care.

Figure 3.2b shows an example of the opposite phenomenon, where a long tail of the fitness distribution leads to an extreme difference in fitness variance between the two sexes, with the initially more variable sex remaining more variable after condition-dependent sex allocation has evolved. It is not clear what the minimum skewness of the trait distribution would be for given ratios of linear functions (the most tractable case) of male and female derivative traits with z that would ensure observing a larger variance in fitness for the sex that is in fact produced in better condition.

It is possible, however, to show for numerical examples which sex has the largest variance. As predicted, positively skewed distribution instances drawn from the Gamma distribution result in males having larger trait variance when the optimal sex ratio is realised, that is, at Nash equilibrium (Fig. 3.3a). However, a negatively skewed example from the Beta distribution shows that female fitness variance can also be higher at the optimum (Fig. 3.3b). In conclusion, we would expect male trait variance to remain higher in the common case of a positively

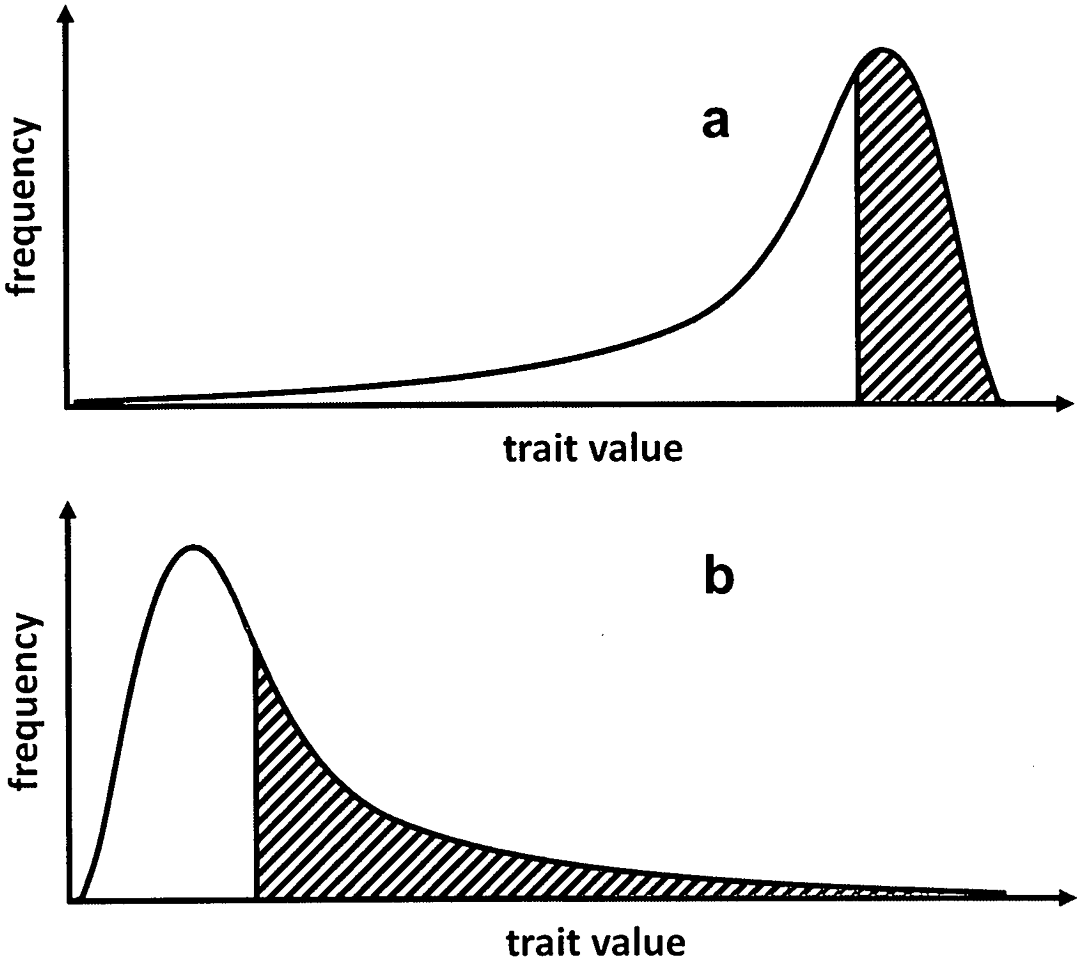

Figure 3.2: (a) An example where measuring the variance in fitness of the two sexes in a population could lead to erroneous conclusions. In this case, the sex produced in better conditions is likely to actually have lower fitness variance (trait variance, hatched area), opposite to what one might expect based on a face value interpretation of Trivers and Willard's formulation of the hypothesis. (b) An example where the correct conclusion would be drawn based on an examination of fitness variance. The sex produced in better condition would show greater fitness variance both with random sex expression and condition-dependent sex allocation with a strict threshold, as illustrated. Hatched area represents sex produced in better condition.

skewed fitness distribution. In the example, the difference was a twofold difference of trait variance for a twofold difference if sex expression were random. This is partly due to the choice of parameters, but really leaves no scope for before and after comparisons - the difference, if any, may be too small to detect. So, since this affects a part of the parameter space, and the parameter space of negatively skewed distributions will show females more variable in the trait even though males are the preferred sex at better condition, the scope for detecting condition-dependent sex allocation by considering trait variances of the two sexes in isolation is non-existent, even though the problematic case of negatively skewed fitness distributions may be rare, and the part of parameter space in which the trait variance difference under condition-dependent sex allocation exceeds that under random sex allocation, may contain a large number of species (namely, the vast majority of those we would describe as promiscuous). Finally, since variance is a second-order property, the sample sizes required to show up differences in variance to the required accuracy for a significant result will be prohibitive.

Many empirical studies of fitness distributions concern populations that to the best of our current knowledge have random sex allocation with respect to any condition as here defined. Many of these studies would fall between the two extremes illustrated here. Most have a significant, often modal, zero fitness class and a long tail (e.g. kittiwake gulls *(Rissa tridactyla)*, Thomas and Coulson 1988; Northern elephant seals *(Mirouanga angustirostris)*, Le Boeuf and Reiter 1988). Some are somewhat symmetrical (e.g. empirical data from Kipsigis women, Mulder 1988; see Mace (2000) for a more modelling-driven approach). In conclusion, then, given our current understanding of fitness distributions, especially in strongly polygynous species where condition-dependent sex allocation is thought most likely to evolve, it is likely but by no means guaranteed that the sex produced in better condition will also exhibit greater fitness variance. If used as a test of whether sexes are expressed conditionally in a given population, fitness variance will frequently lead to false negatives, or to misidentifying the sex that is actually produced in better condition, and hence misidentifying which set of conditions is better. In short, assessments of fitness variance should never be used in isolation to infer any of the above properties of a population.

However, I will discuss at the very end of this thesis how different indicators can be combined to give a more comprehensive test for condition-dependent sex allocation.

Finally, a few words on comparative studies: It may be tempting to try and examine fitness differences within recent taxonomic groups - genera perhaps, where

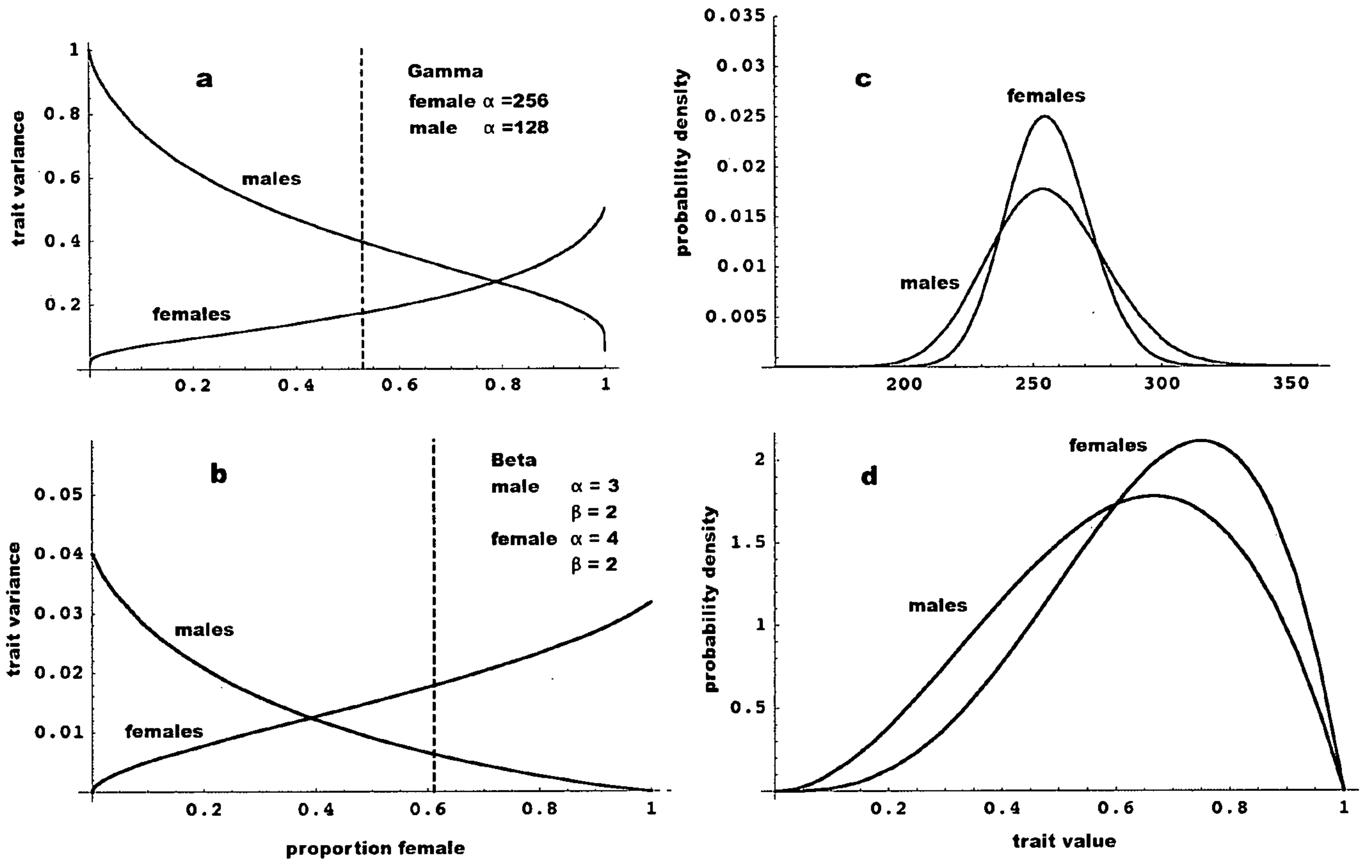

Figure 3.3: Whether males or females show greater variance in the derivative trait depends on whether fitness distributions are generally positively skewed (a, very mildly positively skewed) or negatively skewed (b). Only when fitness distributions are negatively skewed, do females show greater derivative trait variance. Stippled line indicates proportion of females at optimal sex ratio. (c) and (d) are plots of the trait distributions corresponding to (a) and (b), respectively. Distributions and parameters used are indicated in the figure. Trait variance for the Gamma distribution is standardised by the maximum.

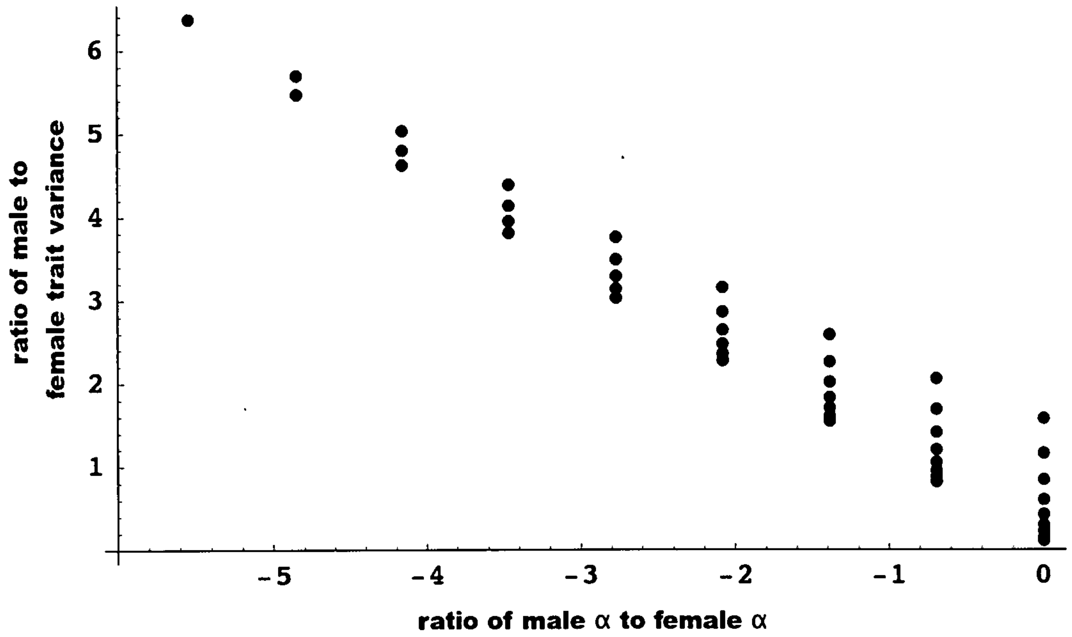

Figure 3.4: Ratio of male over female derivative trait variance with Gamma distributions where male and female shape parameter (α) vary. The ratio of female shape parameter to male shape parameter emerges as the main determinant of trait variance.

morphological similarity might suggest that the shapes of male and female fitness distributions would also be similar between species, and suspect higher levels of polygyny and condition-dependent sex allocation in those species with greater differences in fitness variance between the sexes. However, unless divergence is by allopatric separation of populations, species diverge because of differences in their environments or microhabitats (e.g. parasitic species whose hosts share a range). These environments may enforce different fitness distributions, and it's not at all clear that closely related species differ less in their fitness distributions than more distant relatives; in fact, it is possible that closely related species differ more as a result of their recent divergence. Furthermore, it is not currently clear what happens when two species are close to the switch point where the less variable sex overtakes the more variable, but where each of the two species has a different sex as the most variable. For strongly spiked (leptokurtic) distributions, it is possible that two species have a similar threshold for sex expression, but vastly different variances, while for shallow (platykurtic) distributions, even if one species produced females in better conditions, and the other males, their fitness variances may not be so different.

3.3 Experiments to reveal true fitness distributions

Given that part of the fitness distribution is hidden in natural populations, can we reveal it by an experiment? Several such experiments can be designed; some require few assumptions, but more work, while others are more economic by use of convenient assumptions. I will describe two experiments here, starting with one that uses fewer assumptions. The underlying technique for both experiments is experimental manipulation of the sex of an individual, that is, choosing one or several individuals from a population and forcing them by some technique to be of a particular sex.

The nature of such a manipulation would depend on the sex determination mechanism of the species; species that take external cues into account may be manipulated by less invasive techniques; for instance, to manipulate the sex in an animal with temperature-dependent sex allocation, it may be sufficient to change the incubation temperature at some pivotal phase of its development. For other animals, only a direct hormonal manipulation may result in the desired effect. A lot has been written, for instance, on the female dominance hypothesis, which suggests that sex in mammals may often be influenced by testosterone levels in the mother, and that this may explain some of the sex expression patterns we see (e.g. James 1986, Grant 1990). If we had a species in which this were true, we could choose females whose testosterone levels were so low that they were unlikely to bear sons (as is suggested by the hypothesis), and supplement them with additional testosterone. My opinion is that direct treatment with hormones or other chemical cues of development is likely to yield more accurate results than manipulation with external cues that may closely correlate with the selective pressure that led to the non-random sex expression in the first place. To give a hypothetical example, imagine an animal in whose development there is some pivotal stage at which a particular enzyme is expressed that is crucial in the formation of some organ that plays less of a role in males than in females. Suppose that the action of this enzyme is temperature-dependent, so that there is now selection for individuals to be made male or female depending on their temperature at that particular stage. Suppose furthermore that, as is likely given sufficient time, such temperature-dependent sex expression does, indeed, evolve, and acts at the precisely relevant time. If we use such a species for our experiment, and attempt to change sex by manipulating the temperature at the relevant moment, we will succeed, but our "would have been" assumption is broken, because our manipulation already favours the development of, say, the female-specific organ, so we are not assessing the fitness of a male in which this organ is well developed, as we set out to do. This example illustrates that it is crucial that we manipulate sex directly without affecting any of the factors that may give a selective advantage to one or other sex.

This problem is to a large extent eliminated by a second assumption that must be satisfied in order for our experiment to generate meaningful results. This second assumption is that we can determine the underlying trait value, z, in given individuals. This assumption is not trivial, because we need to ensure that we can determine z at a life cycle stage that precedes sex determination, or else we are open to the possibility that whatever measure we choose may actually be affected by sex. One may also be concerned that, as has been proposed (West and Sheldon 2002 have a good review), the fitness of the two sexes may be biased by future events, such as population density or climate, that parents may have some ability to predict. However, note that even when we measure only one component of the condition or *quality* of an individual, we can draw meaningful conclusions given enough statistical power.

Assuming that we can overcome both of these caveats, what would be the statistical design of our experiment? The conceptually easiest experiment is to manipulate the sex of an individual within a large population. By choosing the population to be large, we ensure that we are not going to be affected by nonlinear effects that may occur when, say, competition between males is made weaker or stronger by a change in the abundance of that sex (e.g. Eshel 1979, Shuster and Wade 2003 pp. 38-73, Kokko and Rankin 2006). The disadvantage is that we need many large populations within whom to manipulate individuals with different trait values in order to build up a dataset. Since one should ideally avoid temporal replication, where one may be introducing additional time-dependent factors to consider in the analysis, such as varying climate, this experimental design is clearly unsuitable for large animals, where the researcher would be required either to be maintaining large populations, or to travel between them, if in a semi-natural setting.

Far preferable for these kinds of species would be a method that makes more efficient use of individuals. Consider again that the reason for switching only a single individual was a concern over nonlinear effects. Given that in the first design, we changed the sex of a single individual, changing the sex ratio in the population, which could be a main contributor to nonlinear effects, it would seem wise to select a second individual and perform the opposite manipulation in order to at least keep the sex ratio the same (although not the trait distribution of individuals by sex).

The idea of simultaneously switching two individuals is the basis of the second design I propose. Specifically, having measured the sex ratio in an entirely natural population, we could take a population and divide it, either randomly or by our assessment of z (or a component thereof) to be the opposite of the expected sex expression in a natural population (in order to gain as much additional data as possible, in addition to the data assumed to have been already obtained in a previous study of the natural population). By doing this, we have drastically altered the trait distribution within each sex, but we have used the natural sex ratio, and thus we may have eliminated at least the main potential source of nonlinear effects. For the case where condition is a function of genotype, this experiment has been conducted by Rice (1992), who used a population polymorphic for two eye colour loci located on different chromosomes, and by artificial selection controlled which sex these loci appeared in. While his study focused on keeping a particular chromosome confined to one sex, and let it accumulate mutations that are beneficial within that sex, the same method could be used to force individuals of a particular genotype known to be co-segregating with a particular eye colour allele, to be born as one sex, or, equivalently, to ensure that the frequency of a particular allele co-segregating with a colour allele to be p in each sex, where p is the mean frequency of the allele across sexes, $p = (p_m + p_f)/2$.

Arguments have been made that a number of taxa in whom condition-dependent sex allocation has been predicted, do not seem to show strong deviations from a 1:1 sex ratio, and reasons have been put forward why this might be. Two suggestions are worth noting; one is that taxa with chromosomal sex determination may not be able to adjust their sex ratios appropriately (Maynard Smith 1978, Williams 1979, Charnov 1982, Clutton-Brock 1986, Clutton-Brock and Iason 1986, Palmer 2000, also see Krackow 1995, West *et al.* 2005); a second suggestion is that overlapping generations (I could not find any prior mention of this in the literature) and lack of environmental predictability (Leimar 1996, Sheldon 1998, West and Sheldon 2002) prevent individuals from predicting future fitness of their offspring accurately. However, if we are going to give any weight to any argument that sex adjustments are limited in scope in some taxa, we must first learn the magnitude of deviations that we would expect. I hope that I have shown that if we are going to predict expected sex ratio deviations, we must determine underlying trait distributions for each sex first, and the only way to do this (with the possible alternative of studies of applicable quantitative trait loci with very large sample sizes) is to conduct the sort of experiments here proposed.

3.4 Model

3.4.1 Threshold model

We can formalise Trivers and Willard by saying that individuals have a trait, z, that may reflect their genetics or physiological status, or some combination thereof. The fitness, w, of an individual as either sex is a function of this trait value z, and written $w_{sex}(z)$. I will use indices m for male and f for female.

The model I shall detail here relies on there being some threshold of this trait z above which individuals are exclusively born as one particular sex, and below which, of the other (see Fig. 1.1). This is an optimal strategy for individuals of the population to follow if a number of other assumptions hold: w_m, w_f and w_m/w_f must be monotonically nondecreasing in z. These conditions are necessary to ensure that there is a single threshold, although it is worth noting that when $w_m(z)$ and $w_f(z)$ are collinear, the threshold strategy is not the only correct strategy. (When there is only one correct strategy, I will refer to it as the "necessary" strategy. Strategies which are necessary in this sense are Nash equilibria (Nash 1951).) In order that individuals can follow the threshold strategy, they also need to have knowledge of their own z as well as that of every other member of the population, along with the mappings $\zeta_m(z)$ and $\zeta_f(z)$, although this may be in some way implicit in their physiology, rather than expressed in conscious thought or other faculties of the brain.

It is assumed in the model that the threshold, z^*, can evolve freely, and it is this threshold we wish to mathematically solve for. Important findings on this model are by Bull, Charnov, and Frank. Bull and Charnov argued that the sex produced in poorer condition (below the threshold) would be more abundant (Bull 1983 pp. 129-131, Charnov 1982 p. 40), and Bull (1981) used a genetic modifier approach to prove this for a model where the zygote controlled its own sex, and where male and female fitness differed only depending on the habitat, not on the number of competing males and females. A more general proof was given by Frank and Swingland in 1988 for the case where female and male quality are monotonically nondecreasing functions of each other, that is, the quality of a given individual were it male being predictable from the quality of that individual were it female, and where quality is proportional to fitness at given sex ratios. I shall repeat their proof here in some detail, using my own notation for consistency.

Imagine that the survival of male and female individuals to adult age depends on a trait z, and is defined as $w_m(z)$ and $w_f(z)$, for males and females respectively. In fact, survival here may be substituted by any other fitness component, such as fecundity, or mating success. Let the trait z take continuous values standardised to lie between 0 and 1. The sex ratio at fertilisation is $P_m + P_f = 1$. Suppose now that the sex ratio depends on z, giving probabilities π_m, π_f that an individual with z will be male or female, respectively, where $\pi_m + \pi_f = 1$. Therefore the ratio of males to females among surviving offspring will be

$$P_m \int_0^\infty w_m \pi_m \, g(z)\, dz \colon P_f \int_0^\infty w_f \pi_f \, g(z)\, dz \tag{3.1}$$

The threshold z^* is the trait value at which "an individual enjoys the same fitness whether it expresses itself as male or female". It can be shown that the sex produced at the more favourable set of conditions will be produced in smaller numbers, while receiving the majority of the resources spent on producing offspring by the population. I will describe their proof in some detail, and use this as a basis for my own points.

Because males and females must contribute equally to the next generation, the fitness values of males and females with trait value z^* would be in the ratio

$$\frac{w_m(z^*)}{P_m \int_0^\infty w_m \pi_m \, g\, dz} \colon \frac{w_f(z^*)}{P_f \int_0^\infty w_f \pi_f \, g(z)\, dz} \tag{3.2}$$

The optimal strategy is to produce sons when the left function is greater, and vice versa (that is, $\pi_m = 1$ or 0 respectively).

Frank and Swingland use the functions $\zeta_m(z)$ and $\zeta_f(z)$ to be proportional to fitness of males and females, respectively. I call these the *derivative traits*, as they result from some mapping $z \longrightarrow \zeta_m \quad z \longrightarrow \zeta_f$. There is again, a probability distribution *g(z)*, as before, and z is standardised to take values between 0 and 1. Total male and female fitness are, accordingly,

$$T(\zeta_m) = \int_0^{z^*} \zeta_m \, g(z)\, dz \quad T(\zeta_f) = \int_{z^*}^1 \zeta_f \, g(z)\, dz \tag{3.3}$$

where ζ_f and ζ_m are assumed monotonically nondecreasing in z, $\zeta_f \neq \zeta_m$, and $\frac{\zeta_f}{\zeta_m}$ is assumed monotonically nondecreasing to ensure there is only a single crossing point. Note that the frequency-dependent nature of the system ensures that there will be at least one crossing point. If the functions cross just once, with males having higher fitness above z^* and females higher fitness below, then the optimal sex expression has all individuals above z^* as males, and females otherwise.

Table 3.1: Summary of notation.

Symbol	Meaning
$\zeta_m(z)$	Male derivative trait
$\zeta_f(z)$	Female derivative trait
$z^*(\zeta_m, \zeta_f)$	Threshold value
$w_m(\zeta_m, \zeta_f, z^*)$	Fitness as male
$w_f(\zeta_f, \zeta_m, z^*)$	Fitness as female

It is important to understand here that there are three different levels of traits. There is the underlying trait, z, that, as per Frank and Swingland, determines the derivative traits that I denote $\zeta_m(z)$ and $\zeta_f(z)$ by some monotonically nondecreasing relationship, such that $\zeta_m(z)$ and $\zeta_f(z)$ are also nondecreasingly monotonically correlated to each other (this latter function is actually the crucial one, as we shall see when we eliminate z later on). Finally, there is a fitness value that, within each sex, is a linear function of the derivative trait value of individuals for that sex; however, between the sexes, these fitness values are related by a constant that is determined by the realised sex ratio, be this optimal or not. Table 3.1 summarises these relationships.

The sex ratio is $S = \int_0^{z^*} g(z)\,dz$. At the crossing point, which is where males and females have equal fitness returns, $\frac{\zeta_m(z^*)}{T(\zeta_m)} = \frac{\zeta_f(z^*)}{T(\zeta_f)}$, which can be rearranged as

$$\zeta_f(z^*) \int_0^{z^*} \zeta_m\, g(z)\,dz = \zeta_m(z^*) \int_{z^*}^1 \zeta_f\, g(z)\,dz \tag{3.4}$$

The monotonicity assumption ensures that male fitness peaks at z^*, $\zeta_m(z) \leq \zeta_m(z^*)$ for the interval *(0,z^*)*, and female fitness has its minimum at z^*, $\zeta_f(z) \geq \zeta_f(z^*)$ on the interval *(z^*,1)*, hence

$$\zeta_f(z^*)\,\zeta_m(z^*) \int_0^{z^*} g(z)\,dz > \zeta_f(z^*)\,\zeta_m(z^*) \int_{z^*}^1 g(z)\,dz \tag{3.5}$$

or

$$\int_0^{z^*} g(z)\,dz > \int_{z^*}^1 g(z)\,dz \tag{3.6}$$

where the left hand side represents the proportion of males, therefore the sex ratio, $S > (1/2)$. Therefore the sex that is produced in poor conditions (in this case, males), will be more abundant.

This is as far as the proof given by Frank and Swingland (1988) goes.

3.4.2 Parameterisation

An ideal distribution to draw trait values for investigating condition-dependent sex allocation from has the following characteristics:

1. Can be parameterised to change the degree of skewness - because skewness of fitness distributions varies in nature
2. Can be parameterised to change the variance
3. Can be parameterised to change the variance, independent of skewness - because condition-dependent sex allocation depends on differences in fitness variance between the sexes
4. Values drawn all greater than zero - because fitness values must be positive or zero
5. The origin of the variance scaling is not zero

The last point may need expanding. It may be easiest to discuss by introducing a definition of a reference point. By reference point, I mean the point from which the distribution expands when the variance is increased. For the Gaussian, the mean is the reference point; for the Gamma distribution, it is 0. Individuals only compete within their own sex, that is, two males chosen at random will always make the same relative contributions to the next generation, no matter how great female fecundity is. Therefore, an increased variance only has an effect when the origin of the scaling is not zero - otherwise the derivative trait ratio between any two males remains the same. Essentially, distributions with different variances are only comparable when their means are the same (or the standard deviation scaled by the mean prior to analysis), and the distribution is not scaled to zero.

It is worth noting that several of these points are mutually exclusive: for bounded distributions, it is not possible to change the variance without affecting either the shape or mean of the distribution. Table 3.2 shows how well some of the more popular distributions fit the needs of a distribution that allows a comprehensive investigation of sex ratio patterns with conditional sex allocation: none of the distributions are perfect, but the Beta and Gamma distributions match the criteria best.

For instance, the Gamma distribution has two parameters, one of which may be called the shape parameter, and the other the scaling parameter. For condition-dependent sex allocation, the scaling parameter is irrelevant for the

Table 3.2: Suitability of various distributions for model.

Distribution	Skewness parameter	Variance parameter	Variance par. indep. of skew	Has lower bound	Has upper bound	Scaled about 0	Total matches
“Ideal”	Yes.	Yes.	Yes.	Yes.	No.	No.	6
Gaussian	No.	Yes.	No.	No.	No.	No.	2
Beta	Yes.	Yes.	Yes.	Yes.	Yes.	No.	5
Gamma	Yes.	Yes.	Yes.	Yes.	No.	Yes.	5
Gaussian e^x	No.	Yes.	No.	Yes.	No.	No.	4

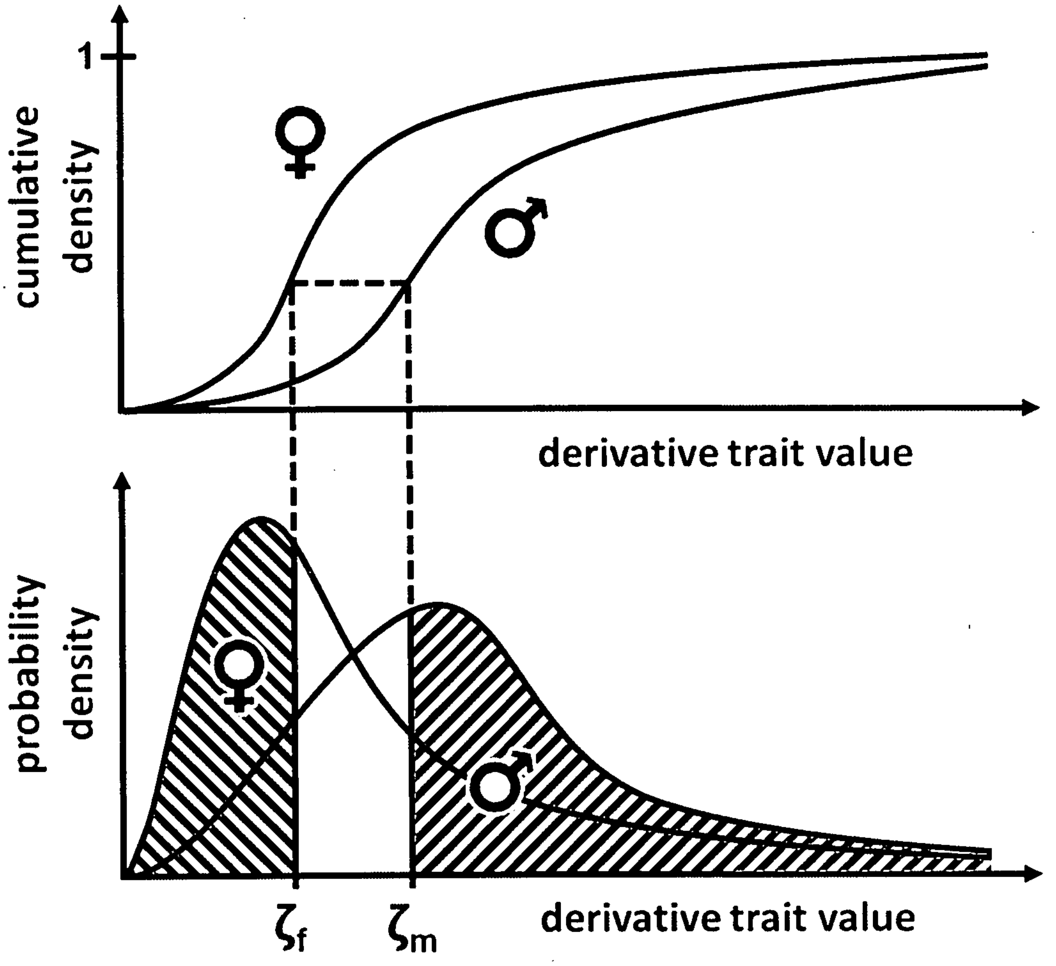

Figure 3.5: The utility of using a distribution function to obtain corresponding values from two distributions. Here, a value ζ_f drawn from a less variable distribution is related to a value ζ_m from a more variable distribution. If ζ_f is at the threshold, then so is ζ_m, and, assuming males more variable, the hatched areas from left to right give the distribution of female and male derivative trait values, respectively. Together, the hatched areas add up to 1. The probability density functions will usually, although not necessarily, cross between ζ_f^* and ζ_m^*.

aforementioned reason, that males compete only within their sex, and the Gamma distribution has a reference point of zero.

In addition to the above constraints, an ideal distribution would encompass fitness distributions that typically occur in nature, so that results may be applicable to real populations. I would suggest that these range from exponential distributions to Gaussian ones (all, of course, ultimately discretised - I will come to that).

Fitness distribution data from actual populations suggest that these distributions frequently have a zero fitness class and a long tail (e.g. kittiwake gulls *(Rissa tridactyla)*, Thomas and Coulson 1988; Northern elephant seals *(Mirouanga angustirostris)*, Le Boeuf and Reiter 1988). Some also show evidence of symmetry (e.g. empirical data from Kipsigis women, Mulder 1988). The Gamma distribution includes all of these, with one of its parameters known as the shape parameter, whose value determines whether a given instance of a Gamma more closely resembles the exponential, Poisson, or Gaussian. Note that one disadvantage of using the Gamma distribution family is that it does not contain any negatively skewed distributions. Figure 3.2a includes an example of such a distribution.

So far, I have presented a model with three levels, and two possible transformations between them. At the base of the model, there is the trait, z, from which are derived the "derivative traits", which are one male value and one female value for each individual in a population. We can think of these derivative traits as attractiveness, or fecundity. We then note that neither attractiveness nor fecundity predict fitness, because fitness depends on how much competition an individual faces. All males may be equally attractive, and there may be so many females in the population that in spite of having a high fecundity, one would only have very few surviving offspring to the next generation - what we initially defined as fitness. It is convenient to assume that the transformation between the derivative trait and fitness is linear at given sex ratios, since any other transformation can be absorbed (in the statistical sense) into the transformation of z into ζ_{sex}.

In terms of analysing such a model, there is a concern that even though z may follow a distribution with convenient properties, after an arbitrary transformation, ζ may not, hampering analysis. (The exponential transformation is generally popular, as it avoids negative values.) It would be preferable if each of ζ_m and ζ_f followed some well-defined distribution.

If we consider two traits, values of each of which are drawn from a known distribution, with a single monotonically nondecreasing function that can map

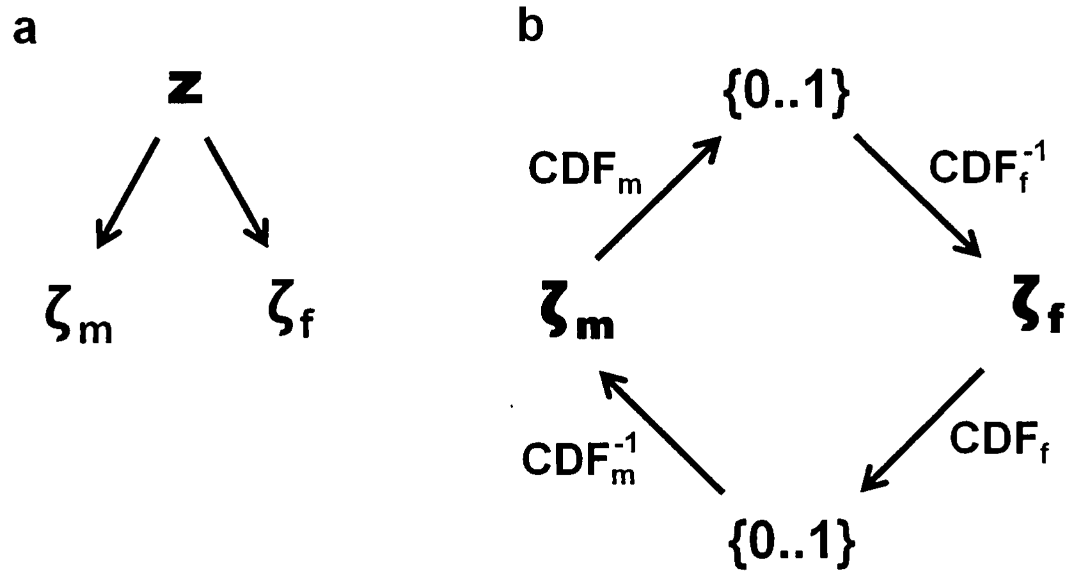

Figure 3.6: Rather than (a) deriving both ζ_m and ζ_f from z, we can (b) have both ζ_m and ζ_f follow known distributions, and transform them by a mapping that uses their cumulative density functions (CDF) and their inverse functions (CDF^{-1}). The variables shown in extra bold (z in (a), and ζ_m and ζ_f in (b)) are those that can be ensured to follow distributions whose properties are understood in detail, such as those from known distribution families. The fact that this transformation goes via an intermediate value that is in the interval 0..1 can be helpful to standardise values for illustration, especially where at least one of ζ_m and ζ_f is from a distribution that has no upper bound.

values drawn from one distribution onto values drawn from the other, how can we discover this function, and carry out the mapping for simulated data?

The way to do this is to go via the inverse cumulative density function, which maps any distribution to the interval 0..1. By then using the forward cumulative density function, we can predict trait values of the second trait.

To apply this technique to the problem at hand, let's assume that we would prefer ζ_m and ζ_f to each follow one instance of the Gamma distribution (usually, but not necessarily, two different instances), how can we achieve this while maintaining the monotonically nondecreasing relationship between them? It is in fact possible to use the distribution functions (also called cumulative distribution functions), *D(x)* of the two distributions to derive a mapping, as shown schematically in Figure 3.5.

The mapping function can essentially be constructed by first deriving the inverse distribution function for one of the distributions, $D^{-1}(x)$. The mapping is then

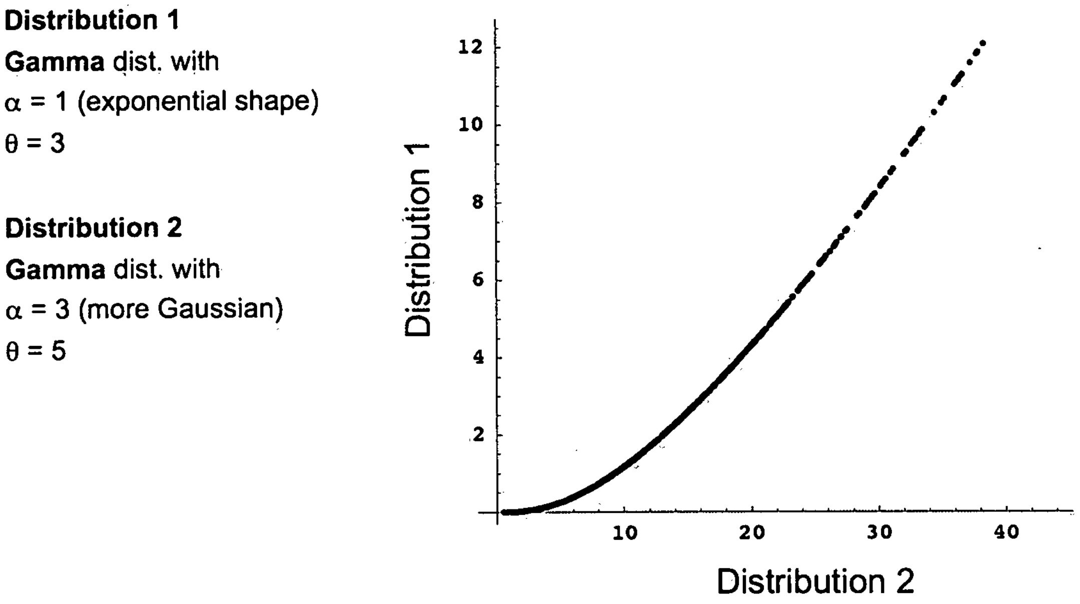

Figure 3.7: An example mapping of two phenotypic distributions against each other, which can be derived by the inverse cumulative distribution function method; due to technical limitations of the computer algebra system used, a random sample is plotted here.

$$y = D^{-1}D'(x) \tag{3.7}$$

where the x are values from distribution D', and y from D. For simulation purposes, pairs of values can now be created by first randomly drawing a value from D', and creating a twin value by use of the mapping function (the twin is an appropriate metaphor here, since the meaning of the two values is that of a pair of identical twins, one born male and one female, as is possible in all taxa with environmental sex determination).

To do this in the computer algebra system *Mathematica*, we would first define our two distributions, *zetam* and *zetaf*:

```
zetam=GammaDistribution[alpha1,beta1];
zetaf=GammaDistribution[alpha2,beta2];
```

We can declare *alpha1* through *theta2* directly as values using an assignment statement, e.g.

```
alpha1=0.5; beta1=3; alpha2=1; beta2=1.5;
```

Finally, we can now use the following algorithm to find the optimal sex ratio to a given accuracy (determined here by the loop termination condition set on the variable *judge*):

```
upper = 500; lower = 0.01;
Do[zstar = (upper + lower)/2;
        judge = (
        NIntegrate[x*PDF[zetaf, x], {x, 0, zstar}] /
        NIntegrate[x*PDF[zetam], x], {x, zstar, 8}]
        ) * (
        theta[zstar, alpha2, theta2] / theta[zstar, alpha1, theta1]);

        (* termination condition *)
        If[judge < 1.00001 && judge > 0.99999, Break[]]

        If[judge > 1, upper = zstar, lower = zstar], {200}];
```

Once we have thus found z^*, we can find the sex ratio, SR:

```
females = NIntegrate[PDF[zetaf], x], {x, 0, zstar}]; males =
NIntegrate[PDF[zetam], x], {x, zstar, 8}];
SR = males/(females + males)
```

This will output the optimal sex ratio. We can also output z^* by simply issuing the command

zstar

We have now directly related the distributions of ζ_m and ζ_f without the need for z as an intermediary. We have preserved the ability to find a threshold, as each value of ζ_m implies a value of ζ_f by a one-to-one mapping, so we can still find numerical solutions for sex ratio and investment for given pairs of distribution instances.

Note also that we are not at all restricted to drawing both distributions from the same family - we could reasonably combine a Gaussian and a Gamma, and derive a mapping function to connect them. This will hold true for any pair of distributions for whom cumulative distribution functions can be defined. Finally, it is worth noting that inverse cumulative distribution functions are generally useful to standardise any mathematically defined distribution to the 0..1 interval (see Fig. 3.6).

3.5 Results

Figure 3.8 shows what happens when we have two underlying distributions of equal variance. Note that we are here assuming that despite males and females

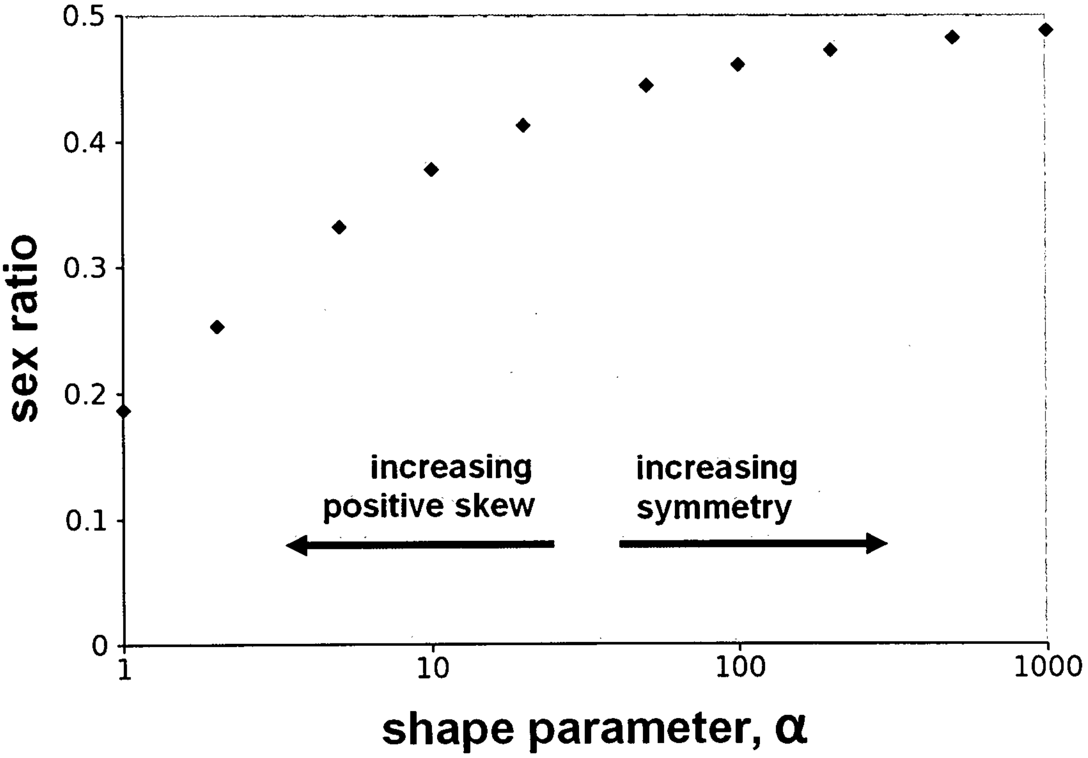

Figure 3.8: Optimal sex ratio with varying degrees of , when male and female trait distribution are an identical Beta distribution, and the model assumes that males are produced above the threshold. Shape parameters above 250 give sufficiently symmetric distributions to produce the correct sex ratio of 0.5.

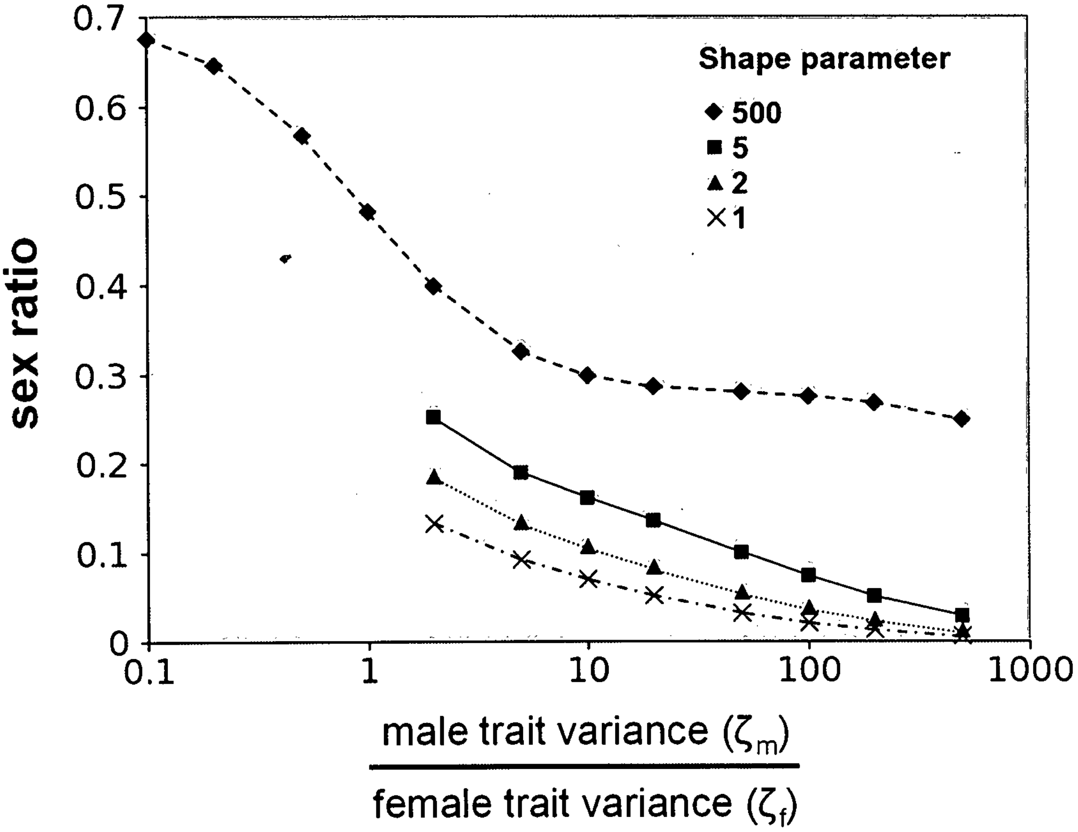

Figure 3.9: Sex ratio plotted against varying male to female trait variance ratio for various values of shape parameter α. Shape parameter $\alpha = 500$ to generate symmetrical Gamma distributions; $\alpha = 5, 2, 1$ represent increasingly skewed distributions; at $\alpha = 1$, the Gamma distribution converges on an exponential distribution. Curves are only shown for the part of parameter space where they are both a correct and necessary solution (see main text for definitions).

following the same underlying distribution of trait values, a threshold exists below which individuals will tend to be female. As the shape parameter of the Gamma distribution increases, the distribution becomes more symmetrical. The figure shows that as this happens, the sex ratio approaches 0.5. Based on our previous discussion of skewed fitness distributions, this is how we would expect the model to behave.

It is important to note that while it is unrealistic that the sex ratio should deviate from 0.5 when the distributions in males and females are the same, this is exactly what would happen in a population that had evolved a threshold mechanism and was facing a change in its fitness distribution. Most importantly, this persistence of an unequal sex ratio raises the question of what would happen in a population that had evolved with males initially being the more variable sex, but where females had since become more variable. Optimality theory, ignoring possible selective constraints, would predict a male biased sex ratio with females being produced in better condition, to be now favoured. Whether such a transition could be possible would seem to depend severally on physiological factors, the genetic architecture by which the sex ratio is controlled, and the extent of genetic drift, but to explore this is beyond the scope of this chapter.

Note that when it is not known which sex has greater trait variance, only symmetrical distributions will give us reliable results. In biological terms, this means that when one sex was initially more variable in fitness, and therefore selected to be produced when conditions are good, it may still be the minority sex even though the other sex has since acquired greater trait variance. Only when the distributions are symmetrical (or negatively skewed) does the sex ratio cross the 0.5 mark when the sex with greater variance changes. The conditions necessary for the sex produced in better condition to switch (in addition to the ratio of fitness variance between sexes crossing 1) will be interesting to explore, but, again, is beyond the scope of this chapter.

Figure 3.9 shows how, for highly symmetric gamma distributions, the sex ratio varies as a function of the ratio of male to female trait variance.

3.6 Discussion

In this chapter, I have further explored a model originally formulated by Frank and Swingland (1988). I have presented a more convenient parameterisation of the model, and discussed what known distributions might be convenient to use for future simulation work. I've also presented some numerical data, and provided examples that highlight some of the problems with using fitness variance as a metric to learn about how populations allocate sexes.

In chapter 2, I showed that so long as female fertilisation is assured, males may have a much higher fitness variance due to neutral causes, that is, causes not due to any inherent ability of the given males to compete better than others. I've also shown that this function approaches an asymptote which, in broadcast spawners, wind pollinaters, etc., and assuming an even sex ratio, puts male fitness variance at five times the female value. In this chapter, I've shown that in order for the observed ratio between the male and female fitness variance to reflect the ratio to be observed if sex allocation were random, the underlying (non-observable) fitness distribution has to be positively skewed. Kassen and Bataillon (2006), in investigating allele fitness effects in bacterial populations, have presented data showing a positive skew in the distribution of individual fitness, across several different media. Similar evidence comes from a study of the plant *Impatiens pallida*, although in this case with a large zero fitness class, and an essentially exponential distribution (Bell *et al.* 1991). Yamazaki and Hirose (1984) raised fifty lethal-free lines randomly sampled from nature, which, when made homozygous, also showed a positively skewed fitness distribution across lines (no data for within lines).

This is consistent with earlier data compiled by Williams (1975), on fecundity in plants and fish. The original source of the plant data, Salisbury (1942), has several exceptions to the rule that fecundity is a positively skewed distributions, two of which, *Pedicularis pedestris* and *Rhinanthus minor*, are classed as semi-parasitic, with a third, *Goodyera repens*, "semi-saprotrophic". None of these, however, are clearly negatively skewed, and *Linaria spuria* even has a distribution that seems uniform across its range. Two other negatively skewed examples are *Thlespi arvense* and *Arenaria serpyllifolia*. In some examples, Salisbury looks at several populations in different habitats, and in this data, some of the populations have a negatively skewed distributions; however, looking at the sum across all habitats, no clear bias is in evidence. Other examples suffer from the fact that data is presented as seeds per fruit and fruits per plant or equivalently for other anatomical units, but not seeds per plant, which would be necessary for data to be conclusive. Williams has also criticised that the data is mostly from herbaceous plants of medium fecundity, while omitting "large woody plants". It would have to be conceded that obtaining seed counts for trees, especially those living in the wild, is an onerous task. For what it's worth, however, Salisbury's data supports positively skewed fecundity distributions as the most abundant pattern by far.

Similar data from birds has been presented by Lack (1954), and mostly supports a symmetric distribution close to a Gaussian, with negatively and positively skewed examples also present. However, Lack's data has greater sampling error due to the numerically limited clutch sizes, and some of the data groups brood sizes into categories, e.g. 1 to 3 eggs and 4 to 6 eggs. Both studies suffer the problem that tradeoffs have been demonstrated between fecundity per unit time, and longevity (e.g. Miyatake 1997, Ramesh and Manickavasagam 2003, Messina and Fry 2003, Mishra and Omkar 2006[1]), as well as fecundity and investment per offspring (e.g. Kim and Thorp 2001, Gillespie *et al.* 2008), with investment known to affect longevity of the parent and offspring (e.g. Kaplan 1992, Lummaa 2001).

This limited knowledge we have about the shape of fitness distributions, in combination with the results from this chapter, which indicate that fitness variance may only tell the truth about which sex is actually more variable when fitness distributions are positively skewed, this suggests that fitness variance is not a useful metric in natural populations.

One important caveat to mention with respect to this chapter is that when some aspect of condition, such as parasite load or nutritional state, is passed on from mother to both sons and daughters, but only in turn passed on to the following generation by daughters, not by sons, some special considerations apply. This kind of transmission is known as a "maternal effect", and Leimar (1996) showed that when condition is quite faithfully passed on in this way, it is adaptive even for high quality mothers to produce daughters, contrary to theory developed in the 1980s that did not recognise this effect. I allude here to Leimar's result only because those who are familiar with it will want to see a brief justification for not including it. As stated before, Leimar's proposition applies to a limited set of cases. While it was conceived with the same biological system in mind as the paper that originated the idea of condition-dependent sex allocation (Trivers and Willard 1973) - that is, polygynous ungulates - it does not extend to the host of cases where *condition* is some property that cannot be maternally transmitted. Among these cases are the effects of paternal genotype, and sequential hermaphroditism, both accountable for a large proportion of the literature on sex ratios, and understood to be significant phenomena at least in

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[1] It turns out that most individuals do not specialise in longevity or fecundity, resulting in population-wide positive correlations between longevity and fecundity (e.g. Tantawy and Vetukhiv 1960, Messina and Fry 2003). Also note that female longevity especially can decline with the number of matings as a result of sexual conflict (Arnqvist and Rowe 2005), confounding the reported trade-off (could explain trade-off between number of matings and longevity reported by Mishra and Omkar (2006), and plays a role in the experimental system used by Messina and Fry (2003), *Callusobruchus maculatus*, which has a violent intromission organ).

birds and marine fish, respectively. I shall therefore assume for the remainder of this paper that the effect described by Leimar is negligible, which is equivalent to assuming a high K/T in his model.

There are a number of particular issues that apply to empirical data and need to be clarified before the ideas in this chapter can be usefully applied to empirical data. They are discussed below.

3.6.1 Costs

Cost-benefit analysis has become an important part of behavioural ecology and other parts of evolutionary biology. It is fully applicable to the sex ratio problem, one of our questions being whether parents create sex ratio or sex investment biases, and to what extent in each given case, or whether this is perhaps too costly. Pen and Weissing (2002) have commented that only a minority of sex allocation theory papers take into account the costs of sex ratio adjustments. Pen and Weissing, in turn, have been the most determined authors in pursuing an analysis of models that include costs - in particular, costs that correspond to particular physiological mechanisms of sex ratio control.

The method used by Pen and Weissing is evolutionarily stable strategy (ESS) analysis, which is simply the process of finding genotypes whose behaviour excludes any invading genotype from the population. That is, the fitness of the resident genotype must be greater than that of any invader. (Note that some models will have more than one ESS.)

Using this method, Pen and Weissing (2002) found that when sex ratio control is by the abortion or abandonment of a propagule (e.g. an egg or foetus), and is only costly when a propagule is actually aborted, there exists a threshold of costs below which sex ratio control and hence abortion take place, and above which, there is no manipulation at all. This disproved an earlier hypothesis of there being a grey area in which *only some* foetuses of the undesired sex are aborted (Box 1 in Oddie 1998).

3.6.2 Uncertainty

The models discussed so far have rested on the assumption that individuals in the population have complete knowledge of their own quality and that of others. This is highly unrealistic in biological systems, not least because populations evolve - relevant here are genetic drift and all kinds of selection except stabilising selection - which can change any signals used in the estimation. However, sex ratios are one example where individuals can avoid competition by being honest.

For instance, were a mother-to-be to pretend to be in a condition to be bearing sons - causing others to bias their sex ratios in favour of daughters - but is in fact carrying daughters herself, then she will have lowered the fitness of her daughters by increasing competition within that sex. Note that benefits of honest signalling accrue to the whole group, so selection is likely to be weak. Note also that more complex scenarios are known where cheating can be of an advantage - for instance, where females carrying foetuses of a particular sex are harassed by dominant females (e.g. Simpson *et al.* 1981). However, evidence of such behaviour is so far restricted to few taxa, notably primates.

Even with no genuine attempt at cheating, there will be costs associated with assessing the status of others, and avoidance of such costs will lead to mistakes being made. There are two principal kinds of mistakes: *decision errors* and *estimation errors.* Decision errors happen when an individual has obtained some information about the population, but fails to express the sex that is optimal given that information. These kinds of mistakes relate to costs in the physiological mechanisms underlying sex manipulation.

With error rates e_m, e_f, the effect of a decision error is the same as when $\frac{e_m}{\iota}$ (if the resulting individual is male) or $\frac{1-e_f}{\iota}$ (if the resulting individual is female) individuals had not allocated at all. The realised sex ratio for any trait value z will be $(1-\epsilon)\ \omega(\zeta_m, \zeta_f) + \epsilon\iota$ where ϵ is the error rate in sex allocation decisions, ω is the optimal sex ratio and ι the "inherent sex ratio", that is, including any biases that arise from different weights of sex-determining sperm, or other features of the reproductive process. ι will typically be a value close to 0.5.

We can correct for decision error in empirical data if we assume that (a) we are dealing with a large population, (b) the value of ι is known, (c) the values of z are known, and (d) at the value of z where the observed sex ratio is at its minimum, the optimal sex ratio is 0 (we could alternatively assume that optimal sex ratio is 1 at the observed maximum). It follows that $\epsilon = \frac{o_{min}}{\iota}$ where o_{min} is the minimum of the observed sex ratio data.

It is also true that those individuals within the population who do allocate correctly can compensate for mistakes made by others. As mentioned before, this is both selfish and altruistic; however, it will only fully compensate as long as the mistaken individuals are few - that is, under stable conditions, the cost of sex allocation will be directly measurable as a deviation from optimal allocation.

The other kind of error, estimation error, arises when an individual obtains less than complete information about itself and/or the rest of the population. In this case, the individual will not correctly estimate its status relative to the rest of the population. For individuals close to the true optimal threshold, this can lead to expressing the wrong sex. While this may seem to imply that individuals closer to the threshold benefit more from carefully assessing others, note also that individuals close to the threshold will pay a smaller penalty for an incorrect decision than individuals whose offspring should more clearly be one particular sex. Whether these effects cancel or not will again depend on the exact distributions of the derivative traits.

Note also that a systematic error in estimating an individual's own condition is equivalent to a systematic error in estimating all other individuals' condition, in the opposite direction. The effect of estimation error can be most easily seen by simulation.

3.6.3 Fitness as a discrete variable

Fitness, the trait we are interested in here, is a discrete trait that is typically measured as the number of offspring surviving to reproductive age. For any given individual, this number would be a whole number. However, for argument's sake, if we had a sufficiently large population that each genotype occurred more than once, we could obtain the number of offspring of each clone in that genotype, and hence get an average fitness that, as the numbers in the clone increase, we could be increasingly content to consider as a continuous variable. (However, note that if the largest clone were of size four, the resulting variable would still be discrete inasmuch as it would be in wholes, halves, thirds and fourths of whole numbers. It is really the assumption that the size of the population is very large that allows us to consider the average fitness (of a clone whose size is now essentially unbounded) to be continuous.)

In genetics, it is often argued that the sum of the members of a genetic clone really constitutes a single organism. In this view, the fitness of this organism would be the total number of surviving offspring produced by the clone, rather than a mean value. This puts us back to dealing with a discrete distribution composed of integer values. This view holds particular weight when the size of the clone may itself be under selection - this will be the case, for instance, when several members of the clone are produced from the same parent, or when they live close enough to be competing more intensely than expected by chance. I would therefore suggest that the value of the mean-fitness-by-clone perspective is restricted to experiments in which we wish to test the fitness of a selection of clones. I described two variants of such an experiment in an earlier section on experiments to reveal true fitness variance.

So it is important to confirm that the conclusions drawn from treating fitness as a continuous variable are not qualitatively different from what is observed when fitness is discrete. Failing that, a framework in which to investigate real, discrete fitness distributions, is needed, in order to see whether anything additional can be learnt about condition-dependent sex allocation.

Most of the framework presented here is as before - we have some continuous variable that represents the trait value, z, of an individual, and we have, again, consequences of this trait for males, ζ_m, and females, ζ_f. The only difference is that at the last step, ζ_m and ζ_f translate into discrete fitness values, typically with an error term thrown in that describes any risks that are not currently mitigated by any allele present in the population, known as environmental variance.

We need, therefore, a transformation that allows us to convert values of ζ_m and ζ_f into discrete values, while ensuring that each individual in the subsequent generation still has a mother *and* a father, that is, the number of offspring produced by males must equal the number of offspring produced by females - simply rounding our previous values for w_m and w_f could lead to rounding error. (Let's also remind ourselves at this point that expressions for w_m and w_f must take into account the advantage given to whichever sex is rarer than predicted by optimality criteria.) The simplest way to conduct a simulation is to drop the decimal part of female fecundities, so they become integers, and then find the factor that the male equivalent value (ζ_m) needs to be multiplied by so that the sum of integer values that results when the decimal portion is dropped is the same as the sum for females. Note that this modifies the previous model in such a way that z^* is no longer predictably on the basis of ζ_m and ζ_f alone: the rounding step must be considered, too. In Mathematica code, we would write this male fudge factor as $Sum[IntegerPart[females]]/Sum[IntegerPart[males]]$.

If we detect a sex ratio deviating from 50% in any given population, we may suspect one or several of a number of causes to be at work, among them condition-dependent sex allocation. If individuals have both their reproductive success and sex recorded, a simple comparison of the variance of the two sexes can be insightful. However, condition-dependent sex allocation acts if the original reproductive variances of the two sexes differ; depending on the type of distribution and its parameters, and the strength of condition-dependent sex allocation (we could think of this as *penetrance*, to use a term borrowed from medical genetics).

There may be rare cases of populations for whom reproductive success has been recorded, but not the sex of the individual. In these, as in other populations, we can do one further test to see whether predictions of condition-dependent sex allocation are met. Fitness is a discrete variable. One may be led to think that because different parts of the genome - the haplotypes - can suffer different fates, fitness is not altogether discrete. However, recombination and independent segregation together are one of the components of genetic drift. Hence, it is not helpful to treat fitness as anything other than a discrete variable that can be usefully expressed in the number of children or grandchildren. Let us suppose, then, that we have obtained the fitness of each individual in the population by an appropriate genetic test, and that we are ignorant about the sex of each individual in the presumably shallow pedigree thus obtained. (Fitness could also be obtained by direct observation (e.g. Clutton-Brock *et al.* 1986), but this would make it more likely that the sex is also known, which renders this test confirmatory at best, and possibly superfluous.) We can order the individuals by their reproductive success, and plot graphs to illustrate the shape of this distribution in our population. We can consider the data in two ways, either by a direct plot, or by a plot of the differences between nearest data points in the distribution. If we find that the entire sample could have reasonably been drawn from one distribution, we may conclude that there is no evidence for condition-dependent sex allocation. If we can show, however, that the assumption of distributions with two different variances for the two tails of the data, with the higher values also having higher variance, is warranted by the data, then we can take this as supporting evidence of condition-dependent sex allocation.

The method outlined here assumes that one can measure the reproductive success (RS) of individuals in a population. If you then rank individuals by RS, make a sorted list of these values, and take the distance between every two adjacent values (e.g. Figure 3.10 for an underlying normal distribution), a sliding window analysis will clearly show the switchover point (Fig. 3.11). Another way to perform this analysis is to consider differences between data points more distant from each other - say, 10 steps apart. With this kind of data, using the differences between distant points actually reduces the amount of pseudoreplication over a sliding window analysis, yet the standardised variance in the data remains unaffected.

The Gamma distribution has a mean of $\mu = \alpha\theta$ and a variance of $\sigma^2 = \alpha\theta^2$.

We can therefore derive a formula that allows us to change α and β such that the mean stays the same, while the variance is changed by a factor of our choosing. In symbols, we write

$$\alpha_1\theta_1 = \alpha_2\theta_2 \tag{3.8}$$

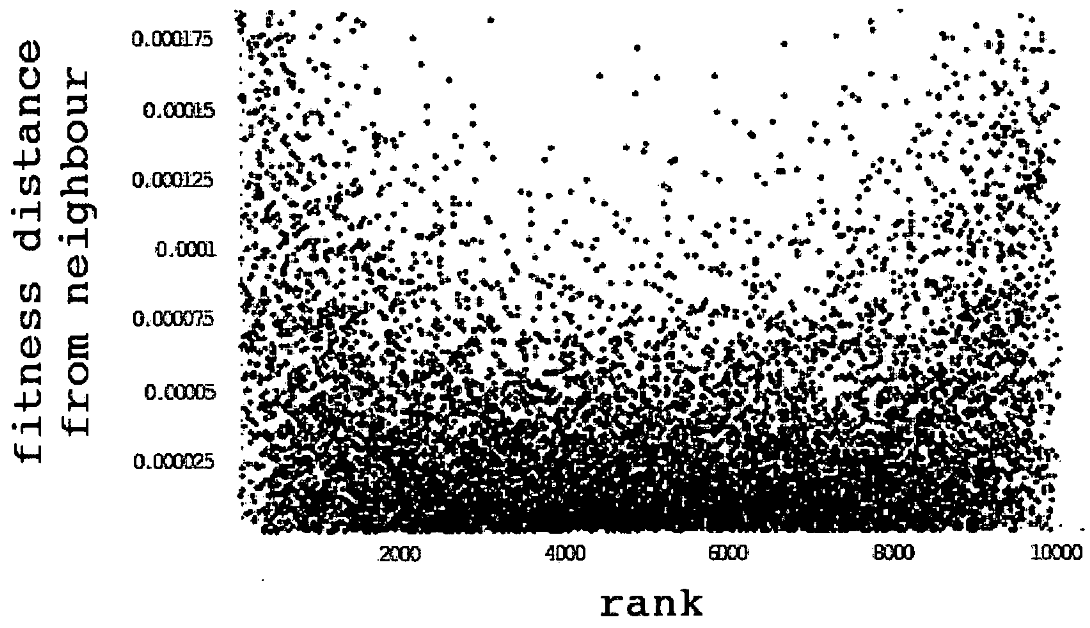


Figure 3.10: Fitness spectrum of a population with normally distributed individual fitness. Individuals are ranked by their fitness (x axis) and then the fitness difference to the next individual plotted on the y axis.

Figure 3.11: Sliding window analysis of a Trivers-Willard fitness spectrum. Population size 10,000; fitness variance difference factor 2. Window size was 50. Fig. 3.10 is an example of the raw data.

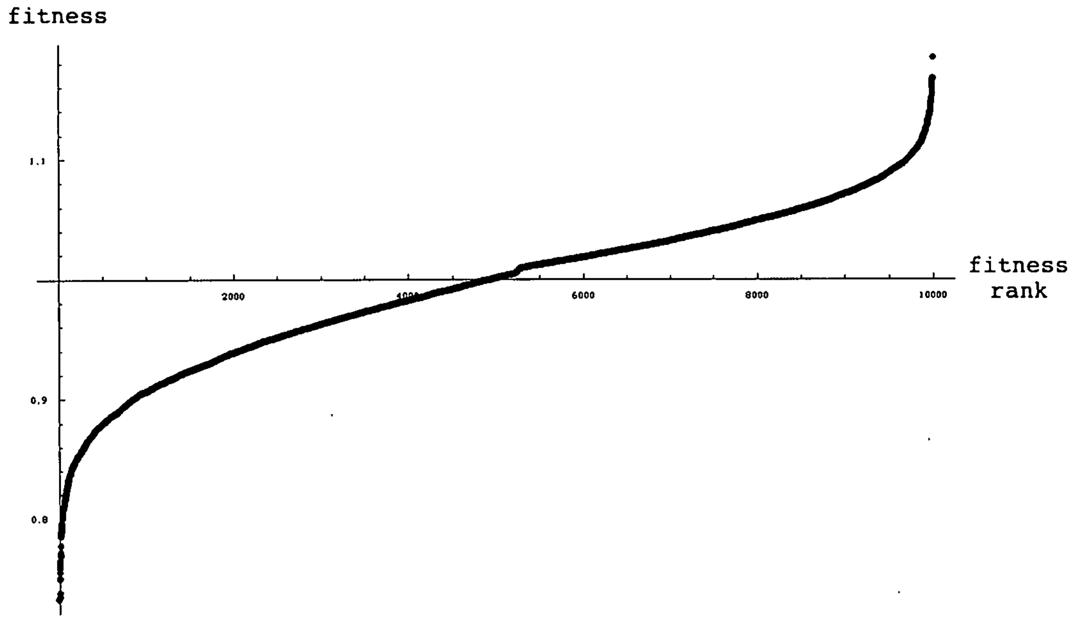


Figure 3.12: Data points ordered by size and then plotted for a sample from the normal distribution.

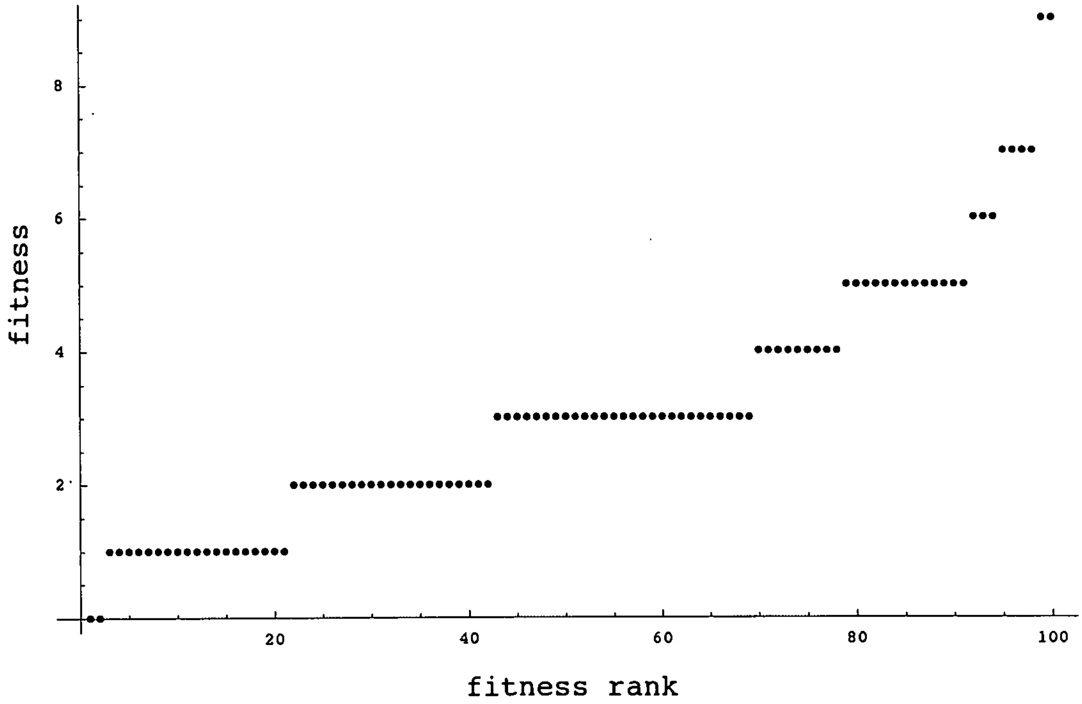


Figure 3.13: Plot of ranked values from a single discretised Gamma distribution for 100 individuals. $\alpha = 3$; $\lambda = 1$. Compare with Fig. 3.12, which is an example of the same plot from a continuous distribution.

$$c\alpha_1\theta_1^2 = \alpha_2\theta_2^2 \tag{3.9}$$

where c is a constant representing the desired ratio of (male to female fitness related trait) variance. We now want to find α_2 and θ_2 in terms of α_1, θ_1 and c. The desired expressions are

$$\alpha_2 = \frac{\alpha_1}{c} \tag{3.10}$$

$$\theta_2 = c\theta_1 \tag{3.11}$$

While breaks in direct plots of ranked data as well as sliding window plots of the differences between adjacent values can readily be detected in samples from continuous distributions, even of moderate size, discretised distributions are far less informative. In such cases, it is necessary to fit curves to data. Knowing what curves to fit can be difficult, since the underlying data can be impossible to obtain. To give an example, it may be true that the fitness of individuals in a particular population in the absence of sex ratio manipulation may follow a Gaussian distribution a separate Gaussian for each sex, where the means are the same (this is always true for populations with 1:1 sex ratios, since each sex must contribute half of the next generation), but variances differ. However, we already suspect that there is manipulation of the sex ratio, or else we would not be considering the question. Assuming this is the case, members of the more variable sex would be expected to be under-represented at the bottom end of the distribution, and vice versa. However, since we do not know what the relationship is between a given individual's fecundity were it female, and its attractiveness if male, we cannot know what the missing part of the curve would look like and recover it. Reasonable approximations in this question are thwarted by the fact that what's best for males may not always be best for females; for instance, it is known that development in the mammalian womb is influenced by the mother's hormonal spectrum, which in turn can be influenced by the number and sex of previous offspring, and the effect of this on any current foetus depends on the sex of that foetus (e.g. Dufty Jr *et al.* 2002, Cohen-Bendahan *et al.* 2005).

Note that the greatest need is for learning the *shape* of the distribution. This being the case, it may be helpful to find a population of a species closely related to our study species, in which several studies using different approaches had already failed to detect any evidence of sex ratio manipulation. Perhaps it would then be reasonable to make the assumption that the underlying distributions of fecundity and attractiveness are the same in that species and our population under study.

I will introduce here the concept of an *earlier distribution*; I chose this term to avoid confusion with the *prior distribution*, which has a specific, not entirely unrelated meaning in statistics. The *earlier distribution* is the raw trait distribution, which is seen before any frequency-dependent selection or discretisation is applied. I shall typeset it in italics to avoid linguistic ambiguities.

Now, what distribution would be suitable for simulating the aforementioned properties of a population? We need a distribution that fulfils several criteria. It must be a discrete distribution that is bounded below by zero. We expect that few individuals excel at reproduction, but there is no hard boundary for how many offspring an individual could have - in a system whose dynamics are determined by processes such as Brownian motion, and random mutation, and where reproductive success depends on number of offspring produced each year as well as longevity (which can be increased by positive mutations in the germ line and decreased by deleterious somatic ones), there can be no absolute limit where it wouldn't be conceivable for the individual to have produced just one more offspring. Therefore our distribution should be unbounded at the top, and be Poisson-like in having an "early hump and long tail". The Poisson distribution takes a single parameter, and therefore mean and variance cannot be changed independently. This is not a problem for modelling the *later distribution*, where, if there is condition-dependent sex allocation, the frequencies and fitness means of males and females differ. However, it is not useful for producing the *earlier distribution*, which requires that the variance, but not the mean, should change.

Note that I am not suggesting that if the *former distribution* follows the Gamma distribution, the *later distribution* will always follow a Poisson. The *later distribution* will be a transformation of the *former distribution*, and it would be a matter of chance if it happened to be an exact Poisson distribution. The Poisson and Poisson-like Gamma distributions both have a very small probability of producing extreme values that are unrealistic especially for females, not to mention that real populations are of finite size.

With those caveats clearly stated, I shall proceed to use a discretised Gamma distribution, which has a humped, long-tailed distribution among the shapes it can produce, and allows mean and variance to be manipulated independently by virtue of its two shape parameters, which according to one of several competing conventions are called α and θ.

Chapter 4

Population genetics and conservation ecology of condition-dependent sex allocation

4.1 Introduction

In chapter 3, I showed exactly how sex ratios and fitness variances deviate from equality under different assumptions about the fitness curve, and I again referred to how condition-dependent sex allocation can arise when one sex chooses mates more carefully than the other. In this chapter, I will expand on the previous chapter by showing how condition-dependent sex allocation creates costs and benefits in adaptive evolution, weigh these against each other, show how this could influence extinction rates and hence clade selection, and show how selection acts on traits that determine sex allocation strategies.

As previously discussed, evolutionary theory predicts that females can benefit from producing sons when mated to genetically superior males, and empirical evidence suggests that they do (Svensson and Nilsson 1996, Rathburn and Montgomerie 2005, Sheldon *et al.* 1999, Calsbeek and Sinervo 2004). It has also been understood for a long time that sex might be maintained because it can increase genetic variability for selection to choose from (Weismann 1889, Fisher 1930, Muller 1932, Burt 2000). Sexual selection can speed up adaptation if the traits selected for actually enhance survival (this is especially true of mate choice - much more so than intrasexual combat, for instance). That is, the genes selected are "good genes" rather than just "sexy". A recent suggestion has been that the greater efficiency possibly given to natural selection by the mechanism of sexual selection could be a major factor in maintaining sex (Siller 2001, Agrawal 2001).

Frank and Swingland coined the term "condition-dependent sex allocation" to refer to the situation where individuals differ in condition, and where this variation affects their fitness more or less severely depending on their sex (Frank and Swingland 1988). This chapter is the first investigation of the joint effects of sexual selection and condition-dependent sex allocation on the spread of beneficial genes.

One increasingly popular method to investigate such problems in behavioural ecology is the modifier locus approach. The approach allows behavioural strategies, including conditional strategies, to be investigated by defining loci whose configuration of alleles determines the strategy followed by the carrier of those alleles. The method also requires explicit linkage relationships to be defined, which brings this method closer to evolutionary reality than general optimality models.

To give a very general example, if we define two loci with two alleles each, we can use the method to determine whether there is any combination of alleles at loci 1 and 2 that leads to a particularly successful behaviour (in terms of reproduction and survival of offspring). When such combinations become apparent in a population, we say there is genetic linkage disequilibrium, which is a measurable quantity both in theoretical models and in genomic data. Moreover, such a finding of linkage disequilibrium would imply the existence of fitness epistasis between the two loci. Technically, linkage disequilibrium is a statistical association between the incidence of alleles at two different loci. The concept can therefore be extended to looking for associations between particular alleles and their environment (or context) (Kirkpatrick *et al.* 2002).

Previous uses of the genetic modifier framework include several studies of sex allocation (Spieth 1974, Eshel 1975). Courteau and Lessard (2000) have used a quantitative genetics framework to also analyse sex ratios, although not using the same framework (whose use for quantitative genetics was explored by Barton and Turelli (1991)). Joshi (2000) used a single-locus population genetic model with females either in good or bad condition (same as the basic model in Leimar 1996) to assess whether a conditional sex expressing allele would succeed over a randomly allocating allele, but with the added peculiarity that his condition-dependent sex allocation allele (T) produced half sons and half daughters when the bearer was in worse condition, and only sons when in good condition.

By contrast, I present simulation results from an haploid sexual genetic model of sex expression, first for an infinite population, showing that selective sweeps proceed faster when sex expression is random with respect to genotype, and then from a finite population, showing that more beneficial fixation events occur when sex expression depends on an individual's genotype.

I will continue with a modifier model of sex allocation to generalise results to a two-locus case. Because this will show whether the locus causing condition-dependent sex allocation increases in frequency, it can tell us whether, and under what conditions, condition-dependent sex allocation will evolve.

In the third model to be presented in this chapter, I'm concerned with the long term consequences of a population's adopting condition-dependent sex allocation. I previously introduced the idea that the condition, z, and sex of an individual would determine its fitness in a given population. In the case of females, condition is the same as fecundity in these models. Since the fecundity of females in a population is a limiting factor for its growth rate, we can compare maximum growth rates under condition-dependent sex allocation with those under random sex allocation, and ask whether condition-dependent sex allocation improves or jeopardises a population's chance of survival. Keep in mind also that if populations were to go extinct more or less quickly based on their mode of sex allocation, this would constitute clade selection, and contribute to the spread or decline of condition-dependent sex allocation.

Cost benefit analysis has been widely used to study the evolution of life history traits and specifically to study the evolution of sex ratios. Many different costs have been studied. The creation of males and daughters may require different expenses (Clutton-Brock *et al.* 1981, Takahata *et al.* 1995, also see references in Torres and Drummond 1999), skewing the optimal sex ratio (as per Fisher's principle, Fisher 1930, pp. 142-143) such that the more expensive sex is proportionally less represented.

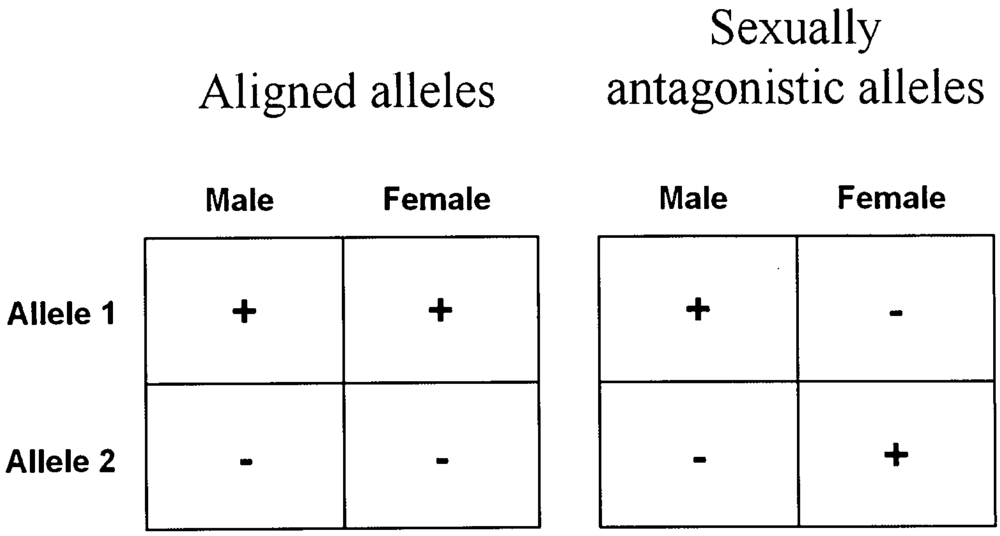
We can also think of costs that may be incurred when parents determine the sex of their offspring, and the effect this has on the actual observed sex ratio. For instance, Trivers and Willard originally discussed selective abortion as a plausible such mechanism. Selective abortion has several consequences and can be modelled in an explicit way to take account of these (Pen and Weissing 2002). One recent finding in this area is that when sex ratio control is by the abortion or abandonment of a propagule (e.g. an egg or foetus), and is only costly when a propagule is actually aborted, there exists a threshold of costs below which sex ratio control and hence abortion take place, and above which, there is no manipulation at all (Pen and Weissing 2002).

If the end result of sex ratio manipulation is a skewed population level sex ratio, one can think of selective abortion increasing the cost for the over-represented sex, as abortion will more often be necessary to create individuals of that sex. This being true, individuals of that sex will also tend to be born later in the season, which is known to be deleterious to prospects of survival (e.g. Pianka and Parker 1975, Reiter *et al.* 1978) and fitness (e.g. Reznik and Braun 2004). If a female's number of available ova is limited, those consumed in the creation of aborted offspring may compromise her prospects of future reproduction - a third kind of cost. Finally, selective abortions may carry an even higher cost in species that lay more than one egg or bear more than one foetus at a time, because either the mother cannot compensate for the lost offspring and instead re-allocates energy to its other offspring, or she would have to accept that some of her offspring will hatch later. A plausible strategy would be to produce an excess of offspring in the first place, and keep only the best. However, such individual-based rejection will have a higher initial cost than wholesale rejection, which could presumably be achieved with much cruder hormonal control. (Interestingly, some species, such as many birds of prey, deliberately produce offspring in series and then favour the first-born over the second-born and so on. Hence, animals do sometimes produce offspring in spite of there being only a small chance that they will prosper.)

A different mechanism would be a general one where sex ratio manipulation has a permanent cost that is paid at conception. One such proposed mechanism is modification of the composition of the vaginal mucus to favour X- or Y-bearing sperm (Shettles 1961).

I'll refer back here to the chicken-and-egg problem mentioned in the introduction: It is unclear, even when condition-dependent sex allocation is known to be acting, whether the mother's good condition comes first, or the inclination to produce the sex with greater fitness variance. If it were shown that, say, female ungulates that have daughters eat less than they could, and that this has implications for their longevity, then it's plausible that these are costs at the individual and population level. Individual level because producing sons sacrifices longevity, rather than being a simple function of pre-existing nutritional condition without affecting health in the long term; and population level, because the individuals producing sons would be raising the per capita consumption of food. The environment will therefore sustain fewer individuals, exacerbating all the problems typical of smaller populations: inbreeding depression and Allee effects (sex ratio stochasticity should not be as much of a problem if the sex ratio of new recruits is manipulated in this way).

An optimal strategy when some alleles show antagonistic effects would be to preferentially allocate the sex that has higher fitness, but keeping in mind that as



Aligned alleles	Male	Female
Allele 1	+	+
Allele 2	-	-

Sexually antagonistic alleles	Male	Female
Allele 1	+	-
Allele 2	-	+

Figure 4.1: The difference between sexually antagonistic and aligned pairs of alleles. A plus symbol indicates the allele with higher fitness within each sex.

one sex gets rare, it also contributes proportionately more to the next generation. Principally, again, it's best to be the sex that is fittest in the given situation. Sexually antagonistic alleles are believed to play a role in the evolution of sex determination systems.

4.2 Model 1: Fixed frequency of strategies

The model is concerned with how an advantageous allele, initially at low frequency, increases in frequency in a population depending on how many individuals follow a particular sex allocation, or more precisely, sex expression strategy previously shown to be adaptive (Trivers and Willard 1973, Frank and Swingland 1988, chapter 3 this volume). I shall introduce this model step by step.

I assume a haploid, sexual population with discrete (non-overlapping) generations. Mate choice and fecundity selection act at the haploid stage, for simplicity, with the diploid stage merely included to allow recombination. The fitness of the resident allele, Q, is set to 1, and the new invading allele, P, to $1 + s_m$ for males and $1 + s_f$ for females, and $s_m > s_f$, so that individuals of the sex denominated "males" are under stronger selection, that is, sexual selection. The frequencies of P and Q are p and q, respectively.

The first set of simulations will assume a set proportion of the population follow a strategy whereby new recruits into the population that are of the fitter genotype are more likely to be males, whereas recruits below the threshold are

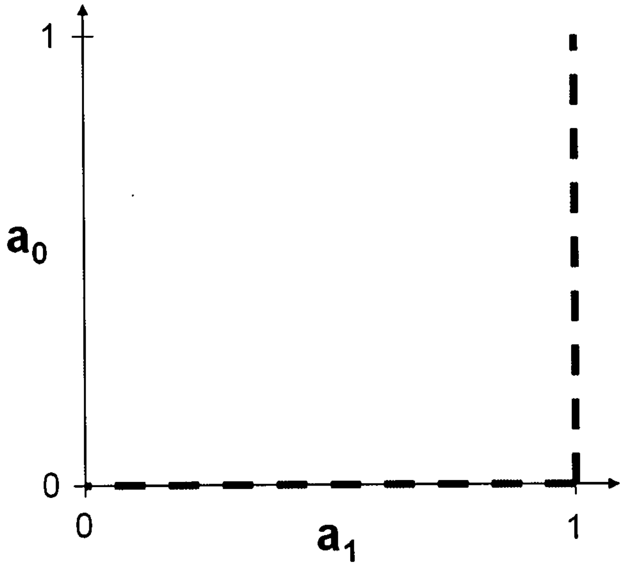


Figure 4.2: The dotted line shows the portion of parameter space along which optimal solutions fall. See main text.

more likely to develop as females. This is a discrete interpretation of the continuous model given by Trivers and Willard, and the adaptive benefit of the strategy is due to males enjoying a greater fitness increment (Trivers and Willard 1973, Frank and Swingland 1988, chapter 3 in this volume). If fitness is determined by a locus with alleles P and Q as above, then we can capture the logic of Trivers and Willard's insight by saying that individuals carrying the Q allele are produced with a sex ratio of a_0, and carriers of the P allele with a sex ratio of a_1, and observe that optimal solutions must lie along the two connected lines illustrated in Figure 4.2. To describe this in words, the work done by Trivers, Willard and Frank implies not only that in the optimal case, $a_1 > a_0$, but also that $a_1 = 1$ when $a_0 > 0$, and $a_0 = 0$ when $a_1 < 1$, a much stronger and more complicated prediction. The part of parameter space for a_0 and a_1 where optimal solutions lie is shown as a dotted line in Figure 4.2.

In addition, let there be a proportion v of individuals who choose a sex randomly, the probability of males being ι, and only the remainder of the population, u (hereafter "the manipulators") follow the Trivers and Willard strategy (hereafter "condition-dependent sex allocation"). In the second set of simulations and model, v and u will be given a genetic interpretation. This notation is in Table 4.1.

An alternative view of the meaning of v is that it describes the accuracy with which "condition" can be assessed, that is, the sensitivity of the sex determining

Table 4.1: Summary of notation.

Symbols	Meaning
u	Proportion of individuals who follow optimal strategy as per Trivers, Willard, Frank, Swingland et al.
v, ϵ	Proportion of individuals who allocate sexes randomly. $v = 1 - u$
ι	Sex ratio produced by random allocators.
s_f	Selection coefficient promoting favourable allele in females.
s_m	Selection coefficient promoting favourable allele in males.
p	Frequency of favourable (good) allele.
q	Frequency of deleterious (bad) allele. $q = 1 - p$
r	Rate of recombination between locus determining fitness and locus determining sex expression strategy.
D	Genetic linkage disequilibrium between P and U loci.
L	Sex ratio.

agent (usually the mother or offspring itself) to offspring genotype. So we can think of u as the *recognition rate.*

I shall continue with the assumption that individuals who can manipulate sex also have knowledge about both the sex ratio "decisions" of those individuals who manipulate sex, and those who produce sexes randomly. This may seem a slightly esoteric assumption, since in reality, some individuals will make their decision first and others later, so some information will not be available to earlier choosers. However, the assumption is somewhat more realistic when sex expression is influenced by hormone levels over an extended period of time, or when the fitness genotype of an offspring is apparent to other members of the population, likely the case in monogamous populations, especially with assortative mating (although the latter case introduces additional complexities that shall not here be discussed).

In the data to be presented, I track deterministic selective sweeps, before subsequently including genetic drift. The advantageous allele is initially present at a frequency of 0.01. Due to the asymptotic behaviour of the function with respect to 0 and 1, a frequency of 0.99 was used as a surrogate for fixation, and is referred to as "virtual fixation". A higher frequency could have been used, but this would only have affected results in a quantitative, rather than qualitative, way.

The haploid carriers of the allele have an "attractiveness" of $1 + s_m$ in males and a fecundity of $1 + s_f$ in females, where non-carriers have 1 and 1, respectively. Note that for "sexy sons" alleles, s_f would be zero (assuming that males cannot detect female genotype, otherwise they might prefer to mate with females who produce sexy sons).

The deterministic results are complemented by stochastic simulations, with population sizes between 100 and 500. These simulations suppressed any stochastic variation with respect to v, that is, the number of individuals produced through random sex allocation was the same in all generations of any runs having the same v. The purpose of this was to reduce error in the results - useful given the established expectation that 1/N selectively neutral mutations will reach fixation (in a haploid model such as this one; N is the number of individuals in a population), resulting in a large number of replicates needed to get a sufficient amount of data to detect differences between different starting parameter values. I investigated selection coefficients ranging from 0.025 to 0.2.

In the stochastic simulation, the first carrier of an allele, whether good or bad, was always set to be male, and the sex ratio in the first generation was exactly 0.5.

4.3 Model 2: Strategy determined by a second locus

The second model contains two loci: The existing locus and a new locus with alleles V and U. A genetic linkage disequilibrium term is needed to allow arbitrary rates of recombination; this term shall be D such that there are $pu+D$ individuals of genotype PU, $pv-D$ of genotype PV, $qu-D$ of QU, and $qv+D$ of QV. Alleles V and U cause carriers to follow the random and optimal allocation strategies respectively. Specifically, we expect $pu+D$ individuals to follow the optimal strategy for P, and $qu-D$ individuals to follow the optimal strategy for Q.

The optimal strategy can be determined by considering the relative fitness of Q and P individuals. The reasoning is this: If the relative fitness of male and female individuals of a given genotype isn't the same, there are individuals who could gain fitness by changing sex and hence should do so (remember that selection for sexes is frequency-dependent such that individuals who change to the rarer sex gain fitness). The relative fitness of a Q individual as a male is $\frac{1}{L(1+s_m p)}$, and as a female, $\frac{1}{(1-L)(1+s_f p)}$. By finding the equilibrium point of the two, we can determine how many Q males and females should be produced:

$$\frac{1}{L(1+s_m p)} = \frac{1}{(1-L)(1+s_f p)}$$

the solution of which shall be denoted L_Q^*:

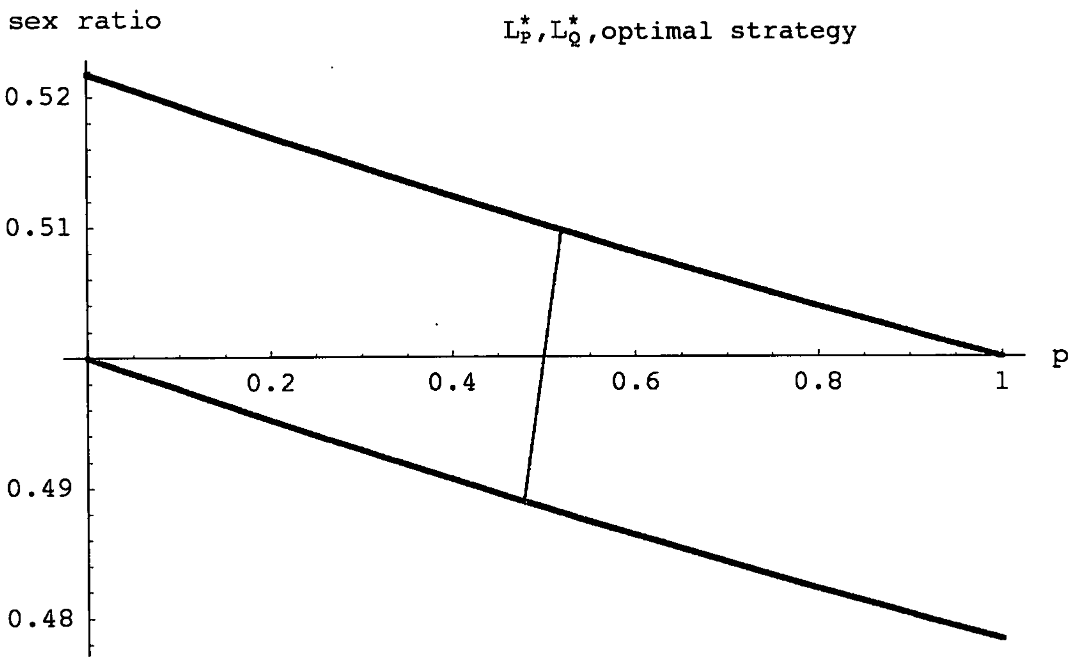


Figure 4.3: L^*_P, L^*_Q and the optimal strategy plotted against p for $v = 0.5$, $\iota = 0.5$. Absence of linkage disequilibrium is assumed in this graph: $D = 0$.

$$L^*_Q = \frac{1 + s_f p}{2 + s_m p + s_f p} \tag{4.1}$$

We can also similarly produce an equilibrium point for P individuals by noting that relative fitnesses are $\frac{1+s_m}{L(1+s_m p)}$ and $\frac{1+s_f}{(1-L)(1+s_f p)}$ for males and females respectively, leading to

$$L^*_P = \frac{(1 + s_f p)(1 + s_m)}{(1 + s_f p)(1 + s_m) + (1 + s_m p)(1 + s_f)} \tag{4.2}$$

Given that according the condition-dependent sex allocation theory, if male reproductive variance is greater due to non-neutral causes, high quality individuals will tend to be male, and low quality individuals female. Therefore PU individuals will tend to be male, and QU individuals female. It is convenient therefore to define the default sex ratio as $v\iota + pu + D$.

We can illustrate the optimal strategy graphically by plotting L^*_P, L^*_Q and the optimal strategy against p (Figure 4.3).

The area below both curves represents a female-biased sex ratio, and the area above both curves, a male-biased sex ratio. The three areas in the graph (below L^S_Q, between L^*_Q and L^*_P, and above L^*_P respectively) correspond to three cases of the relationship between the default sex ratio, $v\iota + pu + D$, and L^*_P and L^*_Q:

- $v\iota + pu + D < L^*_Q$ then all PU male, and sex ratio among QU is $\frac{L^*_Q - v\iota - (pu+D)}{qu-D}$

so that the sex ratio of the whole population comes to L_Q^*

- $L_Q^* < v\iota + pu + D < L_P^*$ then all QU female, all PU male
- $v\iota + pu + D > L_P^*$ then all QU female, and sex ratio among PU is $\frac{v\iota + (pu+D) - L_P^*}{pu+D}$ so that the sex ratio of the whole population comes to L_P^*

From this, we need to determine the new sex-specific allele frequencies and linkage disequilibrium terms. Again, in the same order for the three cases, for $v\iota + pu + D < L_Q^*$:

$$\begin{aligned}
p_m^* &= \frac{(D+pu)(1-\iota)+p\iota}{L_Q^*} \\
p_f^* &= \frac{(pv-D)(1-\iota)}{1-L_Q^*} \\
u_m^* &= 1-\frac{v\iota}{L_Q^*} \\
u_f^* &= 1-\frac{v(1-\iota)}{1-L_Q^*} \\
D_m^* &= \frac{\iota(pv+(D-pv)(v(1-\iota)+L_Q^*))}{(L_Q^*)^2} \\
D_f^* &= \frac{(D-pv)(1-\iota)(1-v(1-\iota)-L_Q^*)}{(1-L_Q^*)^2}
\end{aligned}$$

and for $L_Q^* < v\iota + pu + D < L_P^*$:

$$\begin{aligned}
p_m^* &= \frac{(D+pu)(1-\iota)+p\iota}{D+pu+v\iota} \\
p_f^* &= \frac{(pv-D)(1-\iota)}{qu-D+v(1-\iota)} \\
u_m^* &= \frac{D+pu}{D+pu+v\iota} \\
u_f^* &= \frac{qu-D}{-D+qu+v(1-\iota)} \\
D_m^* &= \frac{(D^2+pquv+D(pu+qv))\iota}{(D+pu+v\iota)^2} \\
D_f^* &= \frac{(D^2+pquv-D(qu+pv))(\iota-1)}{(-D+qu+v(1-\iota))^2}
\end{aligned}$$

and finally for $v\iota + pu + D > L_P^*$:

$$\begin{aligned}
p_m^* &= 1 - \frac{(D + q\,v)\iota}{L_P^*} \\
p_f^* &= \frac{p + (D + q\,v)\iota - L_P^*}{1 - L_P^*} \\
u_m^* &= 1 - \frac{v\,\iota}{L_P^*} \\
u_f^* &= \frac{u + v\,\iota - L_P^*}{1 - L_P^*} \\
D_m^* &= \frac{(D + q\,v)\iota(-v\,\iota + L_P^*)}{(L_P^*)^2} \\
D_f^* &= \frac{(1 - \iota)(D + (D + q\,v)(v\,\iota - L_P^*))}{(1 - L_P^*)^2}
\end{aligned}$$

All else is taken care of using standard recursions for random mating, meiosis, and selection in haploids. We can then simulate this system fully, including the disequilibrium terms.

Note that $L_Q^* < 1/2$ and $L_P^* > 1/2$; also note that for L_Q^* and L_P^* to apply as above, it is required that $v\iota + u \geq L_Q^*$ and $v\iota + u \geq L_P^*$ at the appropriate stages in the selective sweep. If the sex ratio produced by randomly allocating individuals, ι, is too extreme, and u too small, then it may not be possible to the population to reach the optimal sex ratio.

Figure 4.3 also provides a geometric interpretation of the biased sex ratios generated by condition-dependent sex allocation. When s_m increases, the curves defining L_Q^* and L_P^* are transformed down and to the left (with the endpoints at $p = 0, 1$ remaining fixed), while an increase in s_f bends them back towards a linear shape, or beyond that, up and to the right. One could imagine a physical model using pins and rubber bands to be used for educational purposes. The difference between the two curves is $\frac{(1+p\,s_f)(s_f-s_m)(1+p\,s_m)}{(2+p\,s_f+p\,s_m)(2+(1+p)s_m+s_f(1+p+2p\,s_m))}$.

As mentioned before, all of the above assumes that male fitness variance is greater than female, or more precisely, that selection coefficients are larger in males. However, the definitions of L_Q^* and L_P^* do not change when female selection coefficients are larger. What does change in that case are the strategies, that is, *PU* individuals will tend to be female, and *QU* individuals male.

4.4 Model 3: Sex expression and population growth with many loci

I have introduced several models for situations in which condition-dependent sex allocation arises, and here shall investigate one that encompasses sexually antagonistic alleles - a subject that has received much attention recently.

A number of allele frequencies and corresponding fitness effects were first created for each replicate population, the frequencies being drawn from a uniform distribution, and the allele effects from a Beta distribution of given variance. Note that for each locus, one allele was null and the other had a different effect in male and female, hence loci could be sexually antagonistic or synergistic. Assuming additive action of fitness mutations, fitness phenotypes can be calculated as

$$P_{i,s} = 1 + g_{i,1}e_{i,s,1} + g_{i,2}e_{i,s,2} + \dots + g_{i,n}e_{i,s,n} \tag{4.3}$$

where $e_{i,s,j}$ is the effect of locus i if sex s at locus j, and $g_{i,j}$ is the genotype (0 or 1 depending on allele) of individual i at locus j.

This can also be written as

$$P_{i,s} = 1 + g_i \cdot e_{i,s} \tag{4.4}$$

where P is scalar and g, e are vectors.

In the simple Trivers-Willard scenario run for one generation, it suffices to know the distribution of phenotypic quality in the population and the relative variances of male and female in order to predict the resulting sex ratio fairly accurately (chapter 2). Similarly, when the male and female qualities are allowed to follow arbitrary distributions (i.e. not being monotonic positive functions of each other), an optimal decision can be made based on the ratio of the two traits. Algorithmically, the individuals are added as males to an all-female population, starting with the individual with the highest male/female trait ratio (m/f), until the most recent male added has a fitness less than the female with the highest m/f. This last male added is changed to a female, giving the optimal population, or ESS strategy. Not only does this method allow including antagonistic alleles, but it also works regardless which sex has greater fitness variance.

4.5 Results

Figure 4.4 shows that the beneficial allele reaches low frequencies more quickly when there is condition-dependent sex allocation (at $\epsilon = 0$). However, fixation

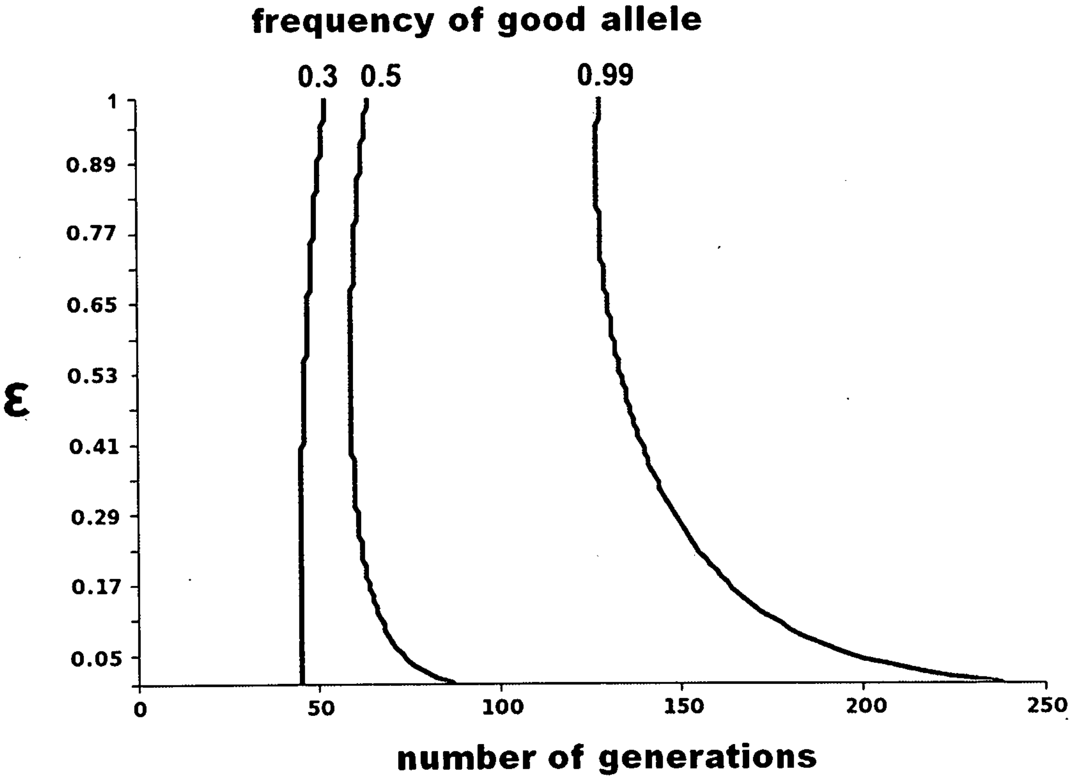


Figure 4.4: Numbers of generations taken to reach allele frequencies of 0.3, 0.5 and 0.99 with different ϵ. Note the fast early progress with low ϵ, but longer time taken to complete fixation.

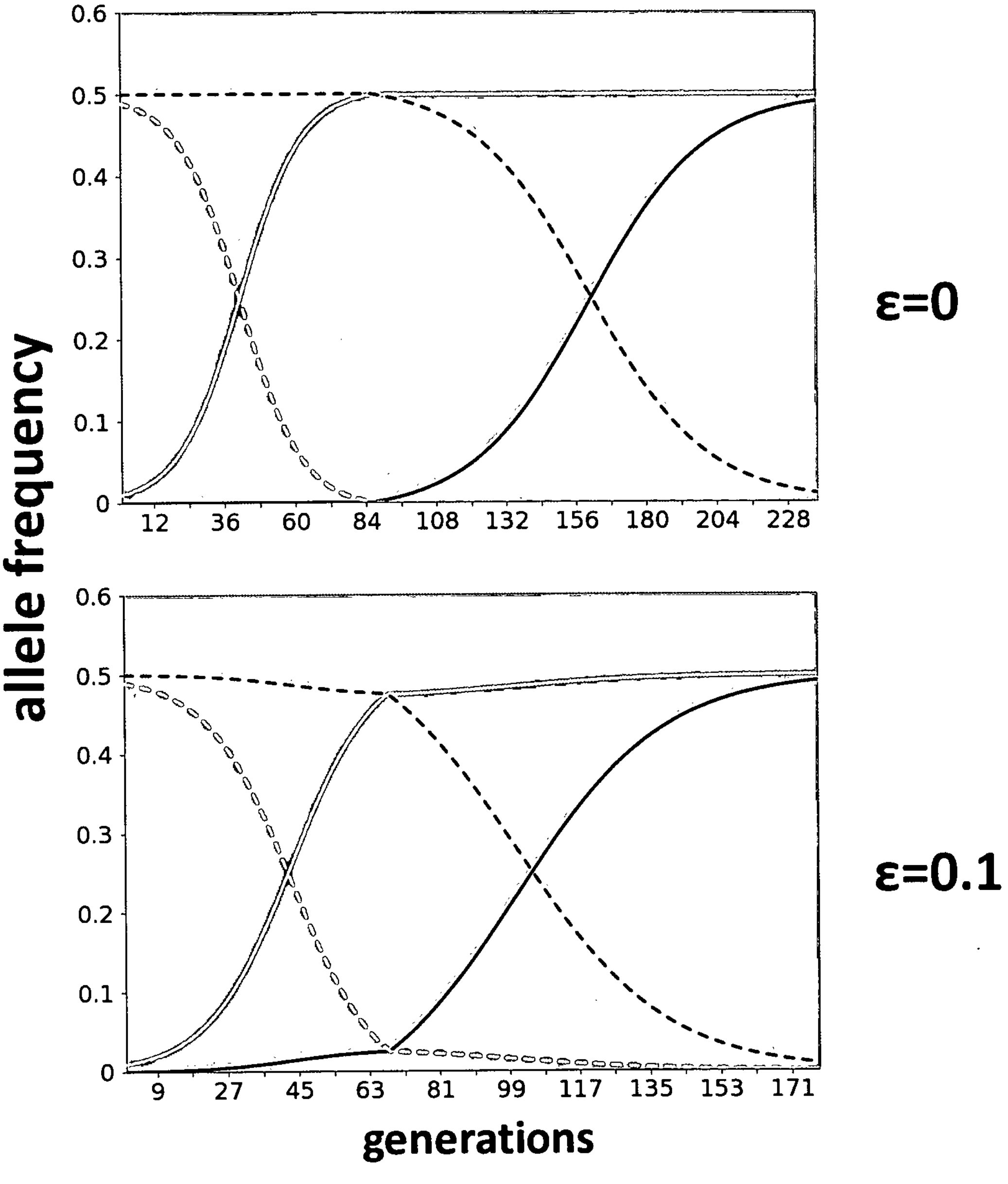


Figure 4.5: Allele frequencies through time with different ϵ. Black lines represent allele frequencies in females, white lines in males. Dotted lines are for the inferior (deleterious) allele. Note again the different number of generations taken to reach virtual fixation.

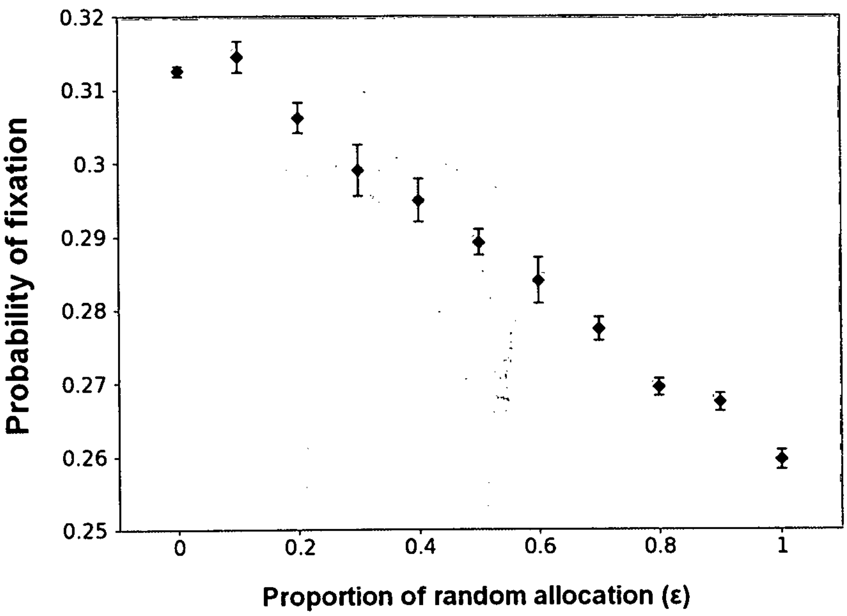


Figure 4.6: Fixation probability of the good allele with varying ϵ. Each data point is based on 50,000 runs of the simulation. Population size was 500, $s_f = 0.1$, $s_m = 0.2$. Error bars represent standard error. Note the interval of the y axis - the effect is not as large as might be expected.

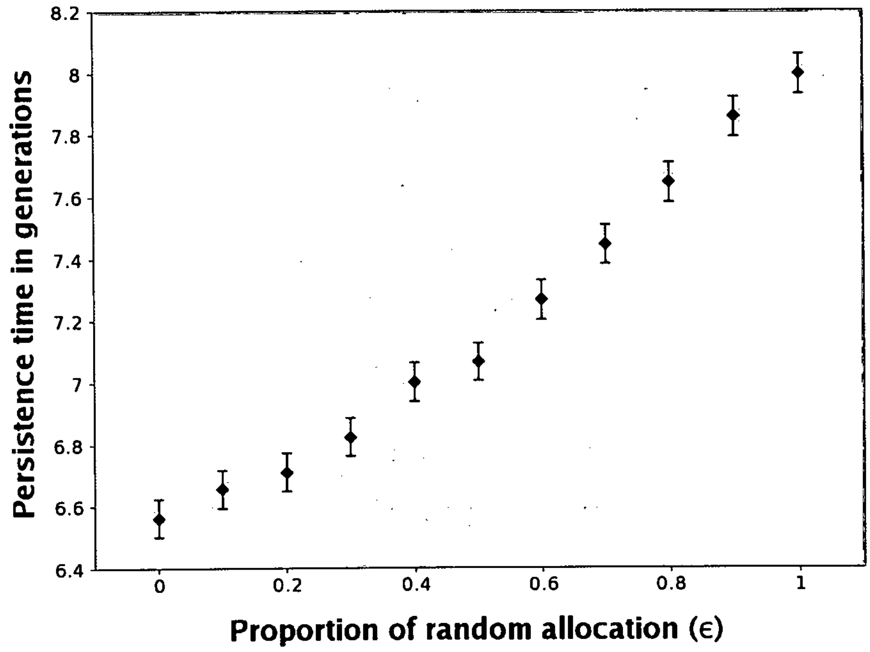


Figure 4.7: Persistence times of the beneficial, invading allele for cases in which it was lost, with varying recognition error, ϵ. Population size was 100, $s_f = 0.1$, $s_m = 0.2$. Error bars indicate standard error.

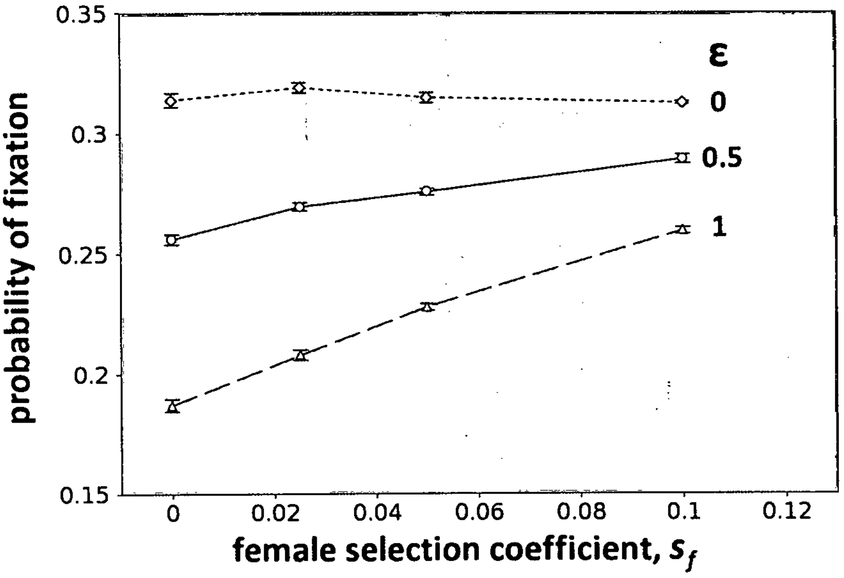


Figure 4.8: Fixation probability of the good allele with varying s_f and ϵ. s_m is 0.2, population size is 500. Data points are based on 50,000 runs. Error bars indicate standard error.

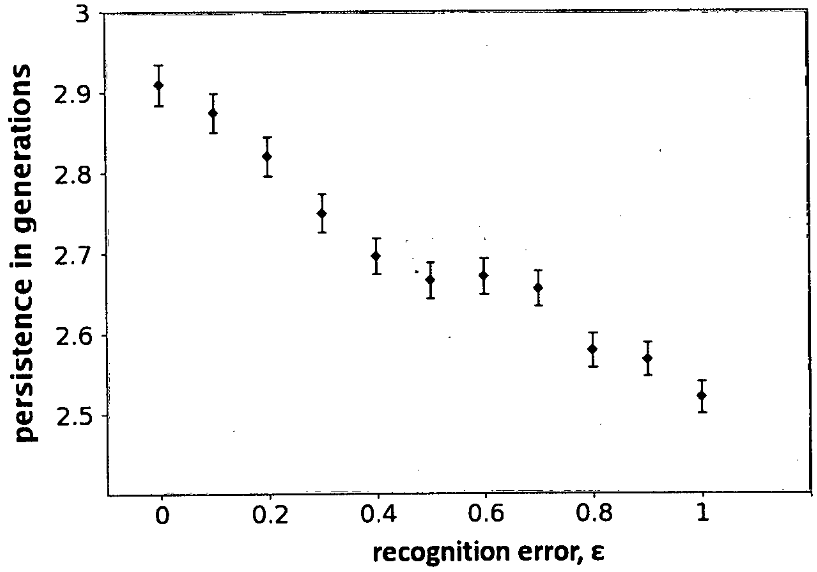


Figure 4.9: Persistence times of the beneficial, invading allele for cases in which it was lost, with varying recognition error, ϵ. Population size was 500, $s_f = 0.1$, $s_m = 0.2$. Each data point based on 50,000 replicates. Error bars indicate standard error.

happens fastest with entirely random sex expression (a high value of ϵ, also Fig. 4.4). We can understand this pattern by looking at the trajectories of increase in frequency followed in males and females separately (Fig. 4.5). When sex expression is tightly controlled, the beneficial allele sweeps through the male half of the population relatively quickly (because initially, all carriers of the allele are born male and benefit from a greater selection coefficient), but then sweeps through the female half slowly, since it is then driven by the lower selection coefficient. Having these two sweeps temporally separated, where first one bottleneck is crossed and then another, accounts for fixation being reached rather late. Figure 4.5b shows an example where ϵ is slightly greater, with $\epsilon = 0.1$. Now the strict partitioning breaks down. Essentially, any strategy that increases the time during which both alleles are present in males will make for faster fixation. This is what we see in Figure 4.5b: the allele remains "in transit" in both sexes until close to the end so that it could be said that the higher selection coefficient in males "supports" the spread through the female half. Essentially, when ϵ is small, the population behaves as though subdivided into a male and female subpopulation, with gene flow only occurring when the favourable allele has risen to sufficient frequency.

From Figure 4.4 particularly we would predict that in populations composed of discrete individuals, the beneficial allele would be fixed more often when ϵ was small, because this would allow it to escape the pull of genetic drift more often while it is rare, and pass into the zone where, given enough time, it will deterministically increase in frequency according to its selection coefficient.

Figure 4.6 shows exactly this - that the fixation probability is greatest at small ϵ, when sex allocation is mostly conditional on genotype. Figure 4.7 adds to this evidence by showing, for the number of times that the beneficial allele is lost through drift, how long it persists before this happens. If we assume that a longer persistence time also implies having risen to a higher frequency than does a shorter persistence time, then Figure 4.6 shows that even when the allele has risen to appreciable frequency at high ϵ, it can still be lost. Essentially, conditional sex allocation lowers the bar that corresponds to the transition from stochastic random walks in allele frequency to deterministic increase towards fixation. To be clear on what this means, this difference would only be seen when the assumption that the selection coefficient in males is greater than in females, is met.

Figure 4.8 confirms the hypothesis that it is escape of genetic drift through placing the good allele in the more strongly selected male sex early on that leads to greater fixation probability, since Figure 4.8 shows s_m to be the major factor determining fixation probability, with a lesser role for s_f. Figure 4.8 also confirms that s_m still plays a far larger role in fixation probability than does ϵ, which is just as we would expect from a locus which is under direct selection. We may also note that the effect of s_f on fixation probability is negligible when $\epsilon = 0$, and s_m accounts for almost all the variation we see. This is as we would expect, since the fixation probability of a positively selected allele is determined by its ability to escape genetic drift, and when the good allele is at a low frequency, it will only be found in males. Hence, s_m will be the determinant of that allele's success, not s_f. We do, however, expect s_f to have a significant role in determining the time taken to fixation, since the good allele will also be found in females eventually.

I repeated the stochastic runs for population sizes of 100 and 500, with the data for 100 being qualitatively identical to that for 500, and therefore not shown. Similarly, results for smaller selection coefficients confirm all the patterns described here.

The same data was also obtained for deleterious mutations (Fig. 4.7). This shows persistence time to decline with increasing frequency of condition-dependent sex allocation. Since under condition-dependent sex allocation, all females are less fit than the lowest male, and rare deleterious alleles always end up in females, copies of a rare deleterious allele will always end up in the least fit individuals of the population, and therefore be more quickly purged than is possible with random sex allocation. This finding is not sensitive to differences in the default sex ratio, ι, presumably because at all but the largest values of ϵ, the conditionally expressing proportion of the population ensure that the overall sex ratio is optimal.

Figure 4.10a shows a temporal correlation between the sudden sex ratio change and the changing sign of sex-averaged linkage disequilibrium. For much of parameter space, this correlation is much less pronounced. Figure 4.10b, in spite of the relatively low u compared to Figs. 4.5 and 5.1, still shows visible traces of the predicted double sigmoid shape in the values of p. Madsen and Shine (1992a) have reported an allele for bluish coloration that increased quickly in frequency, and at a much higher rate in males than in females.

Interestingly, the recombination rate has very little effect on the outcome of the model. Its only marked effect is on the sex-averaged linkage disequilibrium, but even though the frequency of the condition-dependent sex allocation allele increased from 0.5 to approximately 0.6 in the example, this was not greatly influenced by r; in fact, in the example the increase in u was marginally greater when r was high, peaking around 0.5. With very low r, the increase in u was lost again during later stages of the sweep. That's partly accounted for by the fact

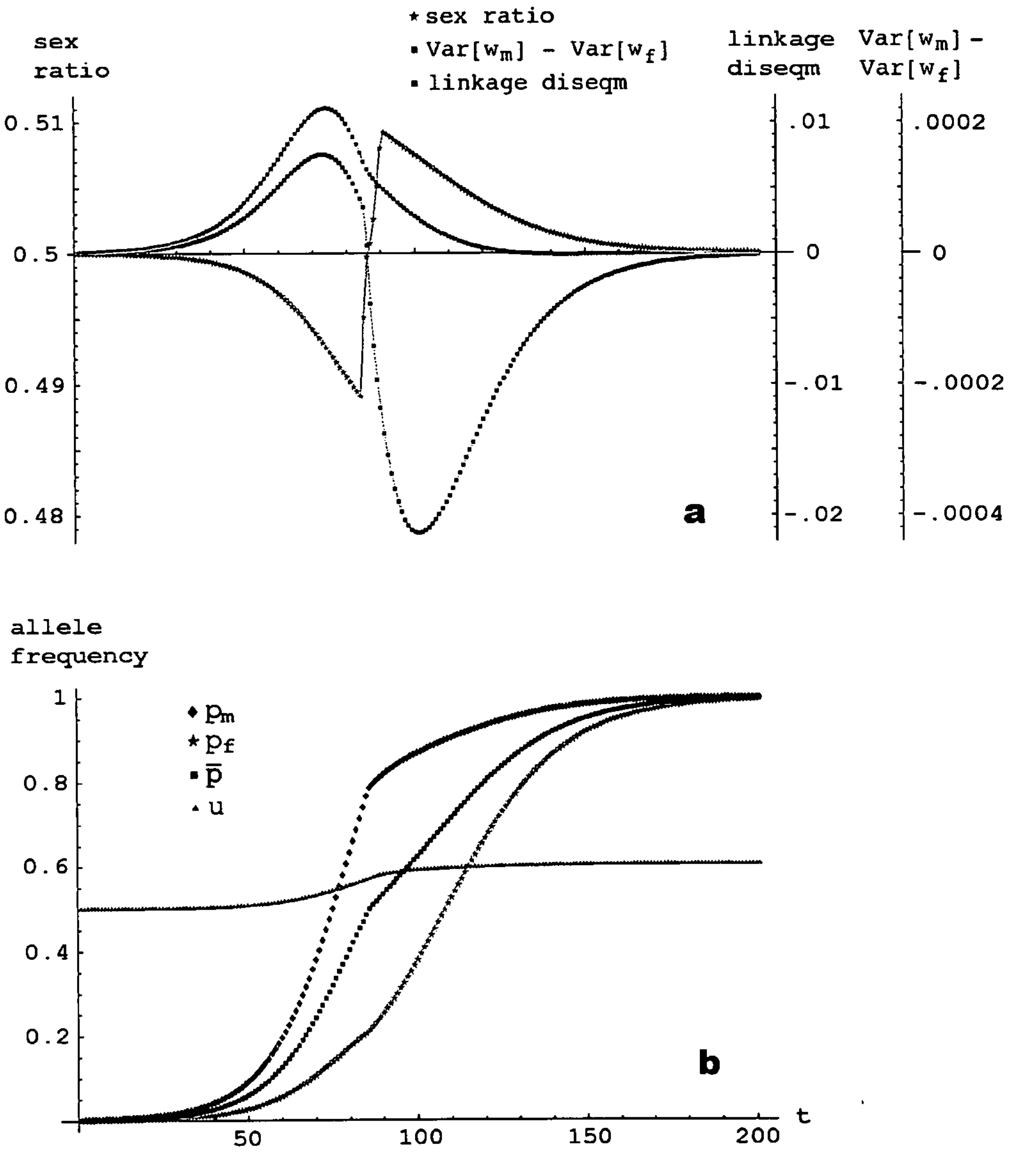


Figure 4.10: Sex ratio, linkage disequilibrium and difference in fitness variance (a), and allele frequencies p for both sexes and averaged, and u averaged only, plotted against time in generations. $s_m = 0.1; s_f = 0.05; r = 0.1; \iota = 0.5$. Initial values $D = 0; p = 0.001; u = 0.5$ for both sexes. *Linkage diseqm* refers to the linkage disequilibrium between the fitness and sex expression loci. The frequency of U is only shown as a sex-averaged plot, since sexes did not significantly differ in u at this scale (but see Figure 4.11). The x axes of parts a and b are identical, including units and scaling.

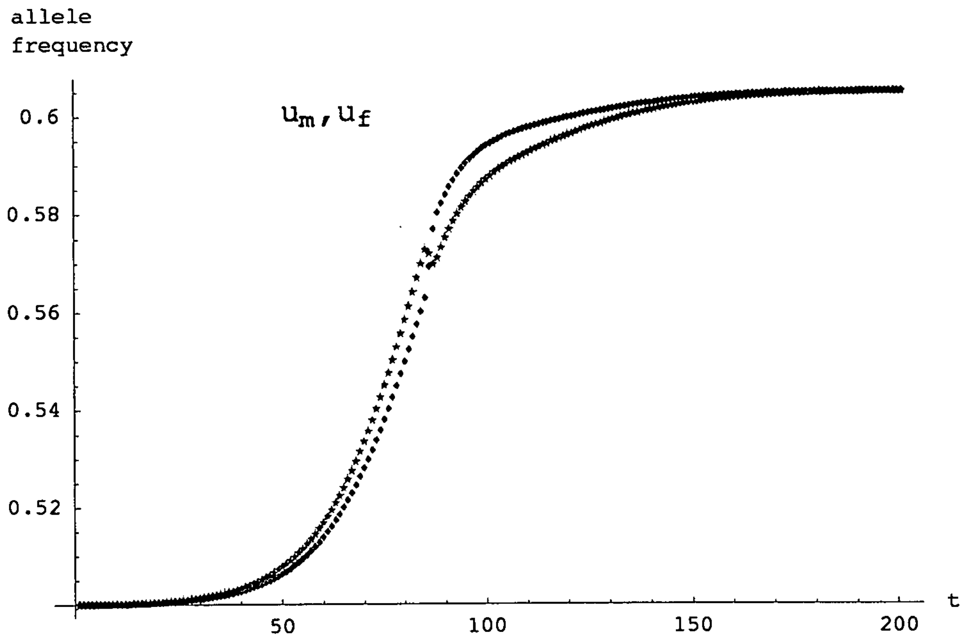


Figure 4.11: Allele frequency u for both sexes. Same parameter values as Figure 4.10. Although U individuals start expressing as females at around generation 82, the rate of increase in u is not affected.

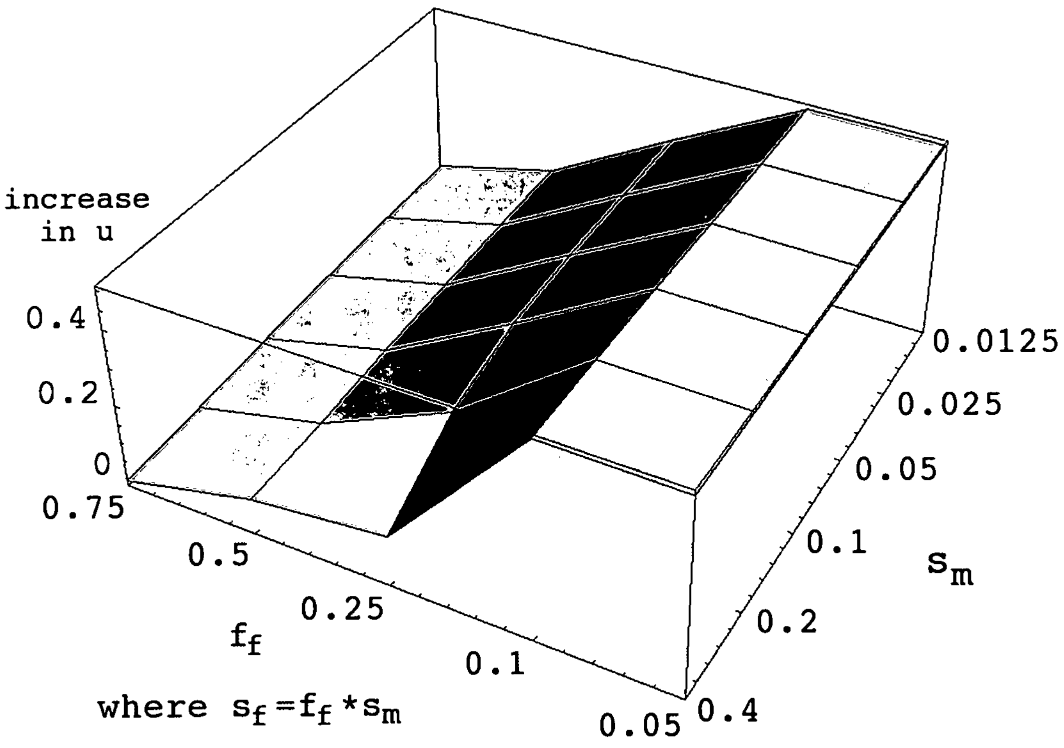


Figure 4.12: Increase in u after 10,000 generations for a single selective sweep event. $v = 0.5, r = 0.1$. Initialisation values as Figure 4.10. NB: A value of 0.5 is equivalent to fixation.

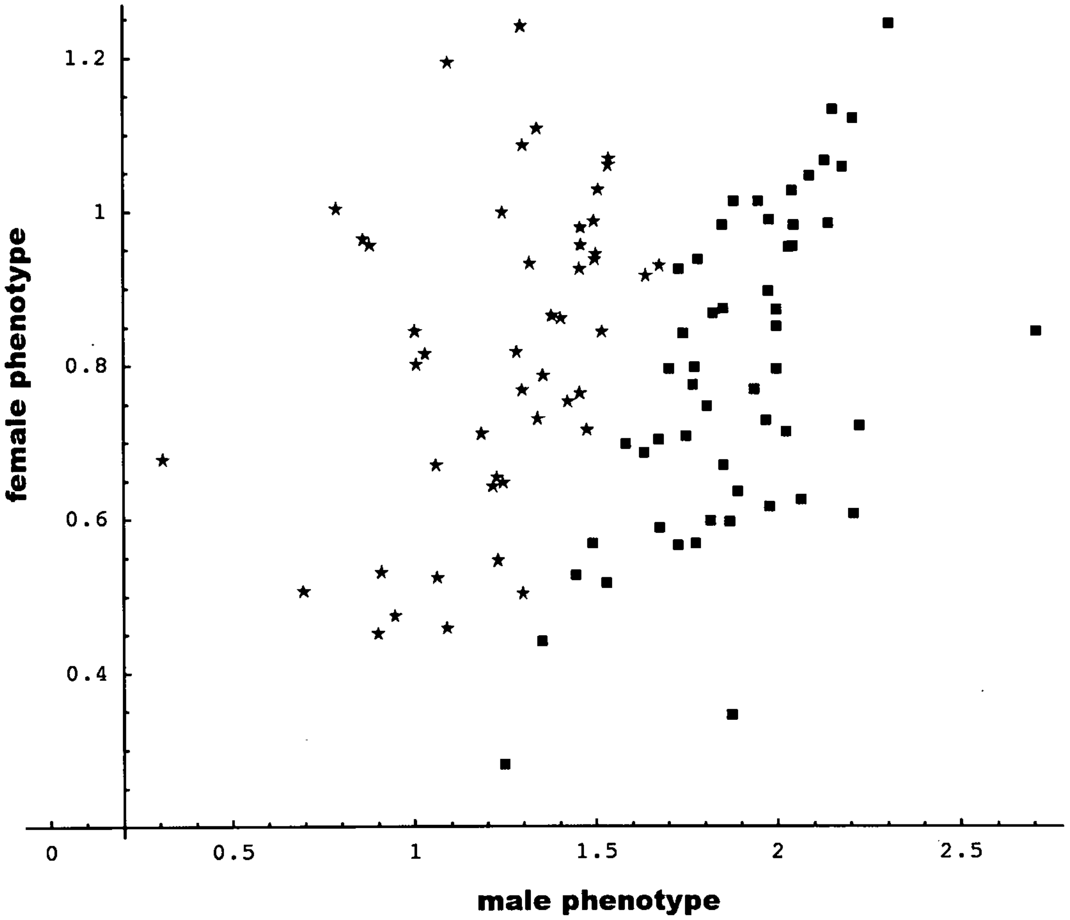


Figure 4.13: Plot of the phenotypic values, indicating sex allocated by the algorithm described. Stars indicate individuals allocated as females, squares represent males. Data is for 100 individuals whose phenotype was determined by 100 loci each. Standard deviation for allele effects was 0.01 for males, 0.0025 for females. Axes cross at (0.2, 0.2).

that some of the U alleles would persist longer than the V even when associated with Q alleles, because the U would tend to put the Q in the sex that's less exposed to selection. When r is low, these Q alleles cannot escape extinction in the long term, and their U allies go down with them when selection is low.

Condition-dependent sex allocation increased population growth rate in every case, compared with the neutral case of random allocation at the correct ratio. This is to a large part due to including sexually antagonistic loci, so that there are always some individuals who, although of low fitness as males, exceed other females in fecundity. Through allowing these individuals to become females, CSE gains extra fecundity. Assuming males are the more variable sex, when alleles are aligned, the opposite will be the case, and females will be less fecund than they would be under random allocation; however, if females are the more variable sex,

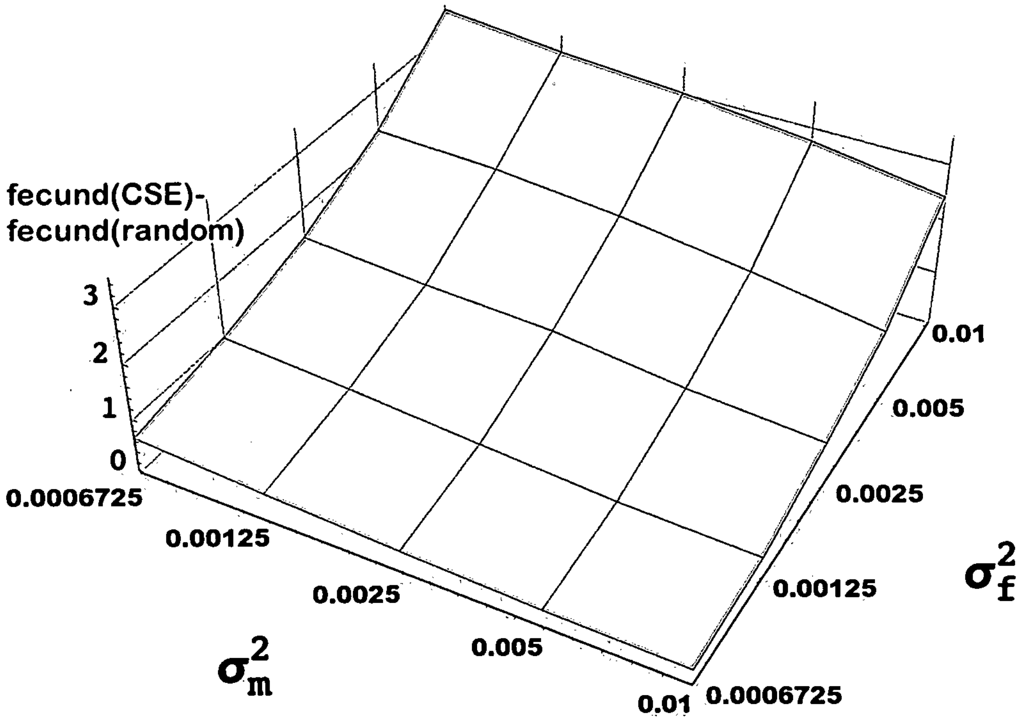


Figure 4.14: Difference in growth rate between a population where sex is allocated to the correct individuals according to the ratio algorithm, and where a correct sex ratio is achieved, but the allocation is random. A positive value indicates that growth rate is smaller with random sex allocation. Data shown is for 5 loci, population size of 1000, averaged over 500 runs. Distribution used for allele effects is a Beta distribution with variances as indicated (these can be transformed into $\alpha = \beta$ parameters for a symmetric Beta distribution by a transformation, $\alpha = \frac{1-4\sigma^2}{8\sigma^2}$).

the population will gain in potential growth rate. An alternative null hypothesis, of random sex allocation at sex ratios closer to 0.5, should be explored in future work.

4.6 Discussion

Joshi (2000) used a single-locus population genetic model with females either in good or bad condition to assess whether a conditional sex expressing allele would succeed over a randomly allocating allele, but with the added peculiarity that his condition-dependent sex allocation allele (T) produced half sons and half daughters when the bearer was in worse condition, and only sons when in good condition. The other allele, F, produces an even ratio regardless of quality. There are three parameters of note in his model: g, determining the proportion of mothers that are in good condition; R, which determines the resources available to mothers in good condition, whereas mothers in worse condition receive unity (1); and d, which determines the slope with which male fitness increment rises with better condition of the mother. It seems that the female slope is assumed to be constant - we might write $d_f = 1$, but being unity, the parameter never appears in any formulae. Likewise, $d > 1$ in all results presented in the paper. Progress of the "Triversian" allele, T, is limited by the proportion of females in bad condition, but reaches higher "equilibrium frequency" when R or d are large (as is evident from the fact that except for the case of daughters produced in the good environment - only possible with F allele mothers - R^d can be considered a single parameter). A similar result emerges from my model, where increase of the conditionally sex-allocating allele slows as polymorphism for condition is eliminated from the population. The main conclusion from Joshi's model would be that it is an unusual, asymmetric model, and that it stops short of exploring implications for adaptive evolution, as I have done here.

It is assumed in my first model that the choice of strategies is a property of the population, rather than being determined by rival segregating alleles. The weakness of this assumption has been addressed in the second model, which contains a modifier locus. The strength of the non-modifier model is its simplicity. It is not affected, for instance, by who determines sex - parents or offspring.

Reasons why natural populations might not allocate sexes perfectly as laid out in condition-dependent sex allocation theory (Trivers and Willard 1973, Frank and Swingland 1988) include such factors as costs in assessing genotype or "condition", costs in forcing sex to be non-randomly allocated, and hard evolutionary constraints (for examples, see Dawkins 1982 chpt. 3, Gould and Lewontin 1979). Note that in the recursive modifier model, it will become important whether mothers or offspring are sex-determining.

When mothers strongly prefer the production of sons over daughters when genotypic quality is high, the system behaves as though the population were subdivided into two haploid ones, a separate selective sweep occurring in each - first, a fast sweep in the male half, then a slower one in the female half, as follows from the selection coefficients entered into the model and the delay caused by first allocating male sex to individuals of better genotype. This results in a slower spread of the advantageous allele overall, although initially faster.

Subsequent numerical simulations show that the initially faster rate of spread helps advantageous alleles overcome the random variation in allele frequency known as genetic drift - through which advantageous alleles are otherwise often lost early on. This suggests an advantage of condition-dependent sex allocation especially in small populations, where drift acts more strongly. Other authors have demonstrated advantages of sex in small populations, including Otto and Barton (2001) and Barton and Otto (2005) (see Iles *et al.* 2003 for advantages of sex in large populations).

This also suggests that there is an advantage from conditioning sex on genotype when there is a beneficial allele at low frequency in the population, and that there is an advantage to following random allocation when the beneficial allele has escaped the zone of susceptibility to drift. The upshot is that sweeps of major effect (large selection coefficient) may select against condition-dependent sex allocation in the long term. I say major effect, because it is likely that several alleles of minor effect will be in transit concurrently, and their effects will average out. I am thus defining a major allele as one whose effect is so large that such an occurrence will be rare enough that the allele will not interfere with another its own size. If for the genetics of the sex expression model, we have two alleles, one for condition-dependent sex allocation, and one for random sex expression, the above hypothesis suggests the prediction that there will be a positive association between the allele under direct selection, and the allele for random sex expression in the later stages of the sweep. This possibility merits further investigation.

Note that modelling sex allocation as a population-level strategy is equivalent to assuming that females have a certain error rate in recognising offspring genotype, whereas the explicit genetic model assumes that only females with the right genotype can recognise offspring genotype at all, with an error rate that I set to zero for simplicity.

Adjustment of sex in response to genotype has been previously used to explain the evolution of genetic sex determination (see papers by Charnov and Bull between 1978 and 1982).

In the ensuing discussion, it is assumed that males are the sex with greater variability in reproductive success. The conclusions are reversed when female variance is greater.

It has been shown that sex can speed up rates of adaptation (Colegrave 2002). The mechanism here described doubtless contributes to the maintenance of sex in all species that manipulate sex in the way described above, as can sexual selection on its own (Siller 2001, Agrawal 2001).

It also merits noting that with condition-dependent sex allocation, there is a considerable delay before the allele finally gets fixed. This delay may help in avoiding interference between genetic linkage groups (sometimes referred to as clonal interference) - yet another mechanism that has already been suggested to be useful in maintaining sex (Crow and Kimura 1965).

When deleterious mutations arise, the same process can similarly help to remove these, by preferentially giving birth to daughters when mated to a male carrying the harmful allele.

What is still unrealistic about this model is its failure to account for repeated or concurrent sweeps. Note that one sweep of a beneficial allele is not enough to also drive the condition-dependent sex allocation allele to fixation. The effect of repeated sweeps under most parameter combinations will be to drive U to fixation, and hence establish condition-dependent sex allocation as a population-wide strategy. However, it is not obvious what effect clonal interference will have on the system - the situation where several beneficial mutations are present in the population simultaneously, and, due to their respective loci being physically linked together, may interfere with each other's growth. Our intuition from previous results should be that U will still be fixed, and that the usual results for clonal interference will apply to the remaining alleles. It is interesting, however, that since CSE partitions a selective sweep into a male and female component as seen in Figure 4.5, it stabilises selection somewhat, and it may be possible for this to reduce clonal interference by offering alleles that intermediate stage. However, the overall slowing of fixation may also increase clonal interference, as alleles take longer to "get out of the way".

What's most interesting is that in most of the runs of the model that I've tried (and there at least four parameters to consider, plus the starting frequencies and linkage disequilibria of the alleles), due to the longer time taken for the female part of the sweep, during which time the females have greater fitness variance and sex ratios are as a result male-biased, the average sex ratio over the course of a sweep is also male-biased. It stands to reason that a female-biased sex ratio as observed in many studies, can only be maintained through a continuous supply of new, favourable mutations with sufficiently large effects.

At a glance, there is a qualitative agreement between Figure 4.13 and the expected performance of the algorithm: females are allocated when female phenotype is more favourable, and males when male phenotype is more favourable (Fig. 4.13).

Note also that departures from 50/50 sex ratios force departure from monogamy (although even with 50/50 ratios, individuals may still be promiscuous), with implications for the spread of sexually transmitted disease.

Chapter 5

Detecting condition-dependent sex allocation in natural populations

5.1 Introduction

In the introductory chapter, I discussed that condition-dependent sex allocation arises from a difference in reproductive variance between males and females. In chapter 2 I showed that while this difference exists before sex expression, it may not always be preserved after sex expression, and its suitability as a diagnostic therefore depends on exactly how fitness is distributed with condition. In terms of finding out whether condition-dependent sex allocation is a strategy used by a given population, this simply shifts the problem to determining the fitness distribution, a task itself fraught with difficulties. Female fitness can be readily determined from observational studies, and roughly estimated in mammals by looking at scars that pregnancies leave in the uterus (although this count will include some stillbirths and individuals deceased in early infancy). Male fitness, for the same reasons that paternity is difficult for males to assess, can be more difficult to get a grip on. Observational methods are used, but it's desirable to confirm these with DNA fingerprinting. When there are many candidate fathers and not many polymorphic markers available, or few linkage groups, such paternity tests may only give probabilistic results, with the added problem that the real father may be a marauding male not included in the study.

While many studies establish the action of condition-dependent sex allocation by determining correlations between some feature of one or both parents, and the sex of the offspring, this suffers the chief problem of all empirical studies, that correlation is not causation. It is therefore of interest to see if condition-dependent sex allocation leaves other traces upon the population that we may detect. This is what I hope to achieve in this chapter. It is convenient that this will also help me summarise some of the evidence and themes presented in previous chapters.

For those who have not read the previous chapters, here follows a brief introduction of condition-dependent sex allocation. The Trivers Willard hypothesis (Trivers and Willard 1973), which can be more broadly described as a case of condition-dependent sex allocation (Frank and Swingland 1988), is one of a number of situations where theory predicts that sex ratios, and also sex allocation, will deviate from equality (where sex allocation is the allocation of resources to the production of individuals of either sex). It generates a number of testable predictions that we can use to easily detect its presence in existing datasets or *de novo* studies, even in situations where sex was not one of the recorded parameters.

So what is condition-dependent sex allocation? It is the situation where the fitness of one sex is greatest when produced in one particular set of conditions. Typically, the fitness of both sexes will be greatest in similar conditions, so they can be thought of competing for these conditions. Evolutionary theory predicts that the sex with greatest variance in reproductive success wins (Trivers and Willard 1973), and is hence generally produced when conditions are favourable. Taking body mass as an example (as did Trivers and Willard), for condition-dependent sex allocation to arise, body mass must have a greater positive effect on fitness in one sex than in another. For instance, female fecundity could be a linear function of body size, and male fertilisation success could be an power law where the exponent is greater than 1. Remember that half the genetic material in the next generation is contributed by males, and half by females, and that males fight among themselves for the privilege to contribute to their half (as, more indirectly, do females). Now, if individuals were created male or female regardless of body size, there would be some males who according to this power function of fertilisation success, would contribute less to the next generation than some females. Hence, these males could contribute more to the next generation by being females instead, and natural selection would favour this. In essence, in this example, males benefit more from the good circumstances than do females, and would therefore be produced in these conditions, and the benefit is in terms of fitness.

A significant amount of data now suggests that, particularly in polygynous mammals, males are generally produced when conditions are favourable (Hewison and Gaillard 1999).

In this chapter, I will first show present two views of fitness, one where fitness is a continuous variable, and one where it is discrete, and discuss their utility and applicability. I will then go on to explore scenarios where different kinds of data are available to determine whether condition-dependent sex allocation is taking place in a population.

There are a number of different ways in which researchers have sought to provide evidence that there is condition-dependent sex allocation in any given system. Studies differ in the strength of evidence. Some studies use correlations between offspring sex and fitness and parental trait values, such as maternal dominance (Ostner *et al.* 2005), timing of conception (Holand *et al.* 2006), and food availability (Holand *et al.* 2006). Bereczkei and Dunbar (1997) criticised that previous anthropological studies had only demonstrated either that the allocation of resources was skewed towards one sex, or that one sex benefited more from a given level of investment than the other, but never both for the same study population. The former alone would only prove the differential allocation hypothesis, whereas the latter will show that there is the basis for condition-dependent sex allocation. There is no theory in existence that would not predict the sex whose fitness benefits more from investment to be born into a less favourable circumstance.

Several predictions can be made that should be met by populations affected by condition-dependent sex allocation where there are no sexually antagonistic alleles, that is, there are no loci having major effects where one allele increases the fitness only of males, whereas another allele increases the fitness only of females (note that when there are two alleles, it is not possible to say whether one allele increases fitness or the other causes a fitness detriment; these statements are equivalent). I will go into three of these below; they are (a) the weak prediction that sex ratio and investment of resources in the two sexes deviate from equality in opposite directions from each other, (b) the strong prediction of particular trajectories in frequency through time of selected alleles, and (c) the strong prediction that a cross-sectional allele frequency spectrum would have three modes, at allele frequencies 0, 1, and one near the population sex ratio. The prediction that variance in reproductive success will differ has been sufficiently discussed in chapter 2 and 3.

It is also known that in condition-dependent sex allocation, the sex that "requires" more resources to produce (to be evolutionarily competitive) is also produced in smaller numbers. This was made clear by Frank and Swingland (1988), following earlier, less complete treatments by Bull (1981) and Charnov (1982 p. 40). To be clear, this means that the population as a whole may devote more resources to producing males than females, if males are the more variable

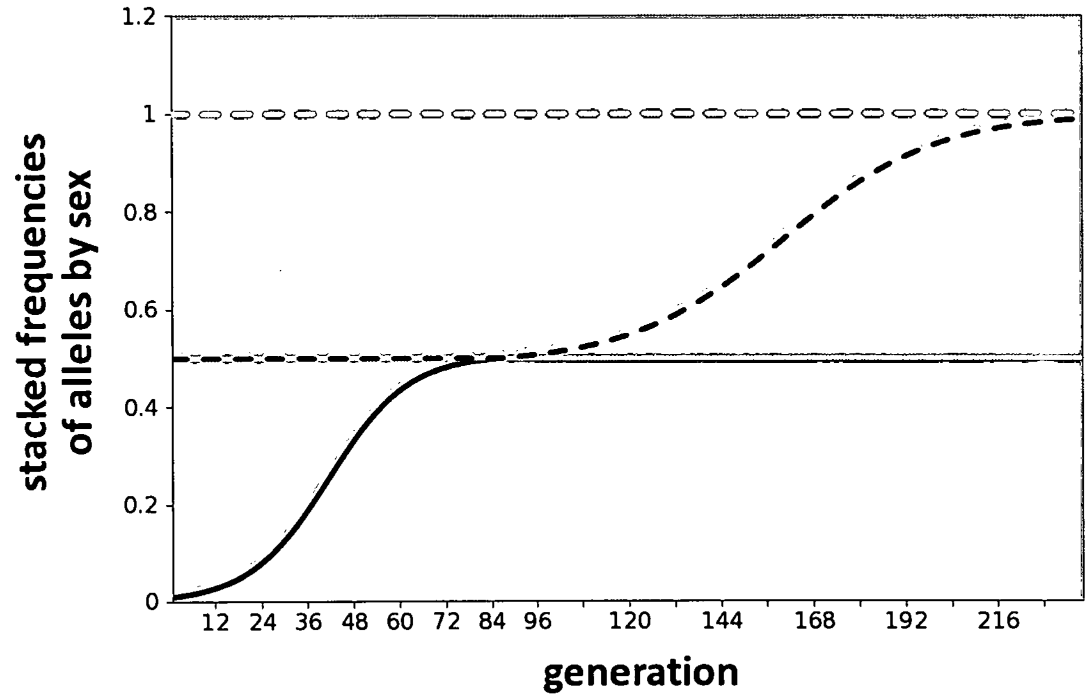


Figure 5.1: A characteristic double sigmoid curve arises from the spread of favourable alleles under condition-dependent sex allocation. Data is from chapter 4.

sex; so the fewer males individual females may be compelled to produce as a result of female choice, the more costly they become. Note that this is a greater excess investment than predicted by Darwin and Fisher for the case of fewer males being produced. Darwin and Fisher saw, retrospectively exposing their hidden assumption, when the fitness variance of the two sexes is the same, equal resources would be invested in both sexes, such that if fewer males were produced, natural selection would favour greater investment to make them competitive; however, the sum of resources used on all of the males in a population would still equal the sum expended in producing females. Thus, if expenditure on each sex as well as the sex ratio (excluding stochastic sources of mortality that may affect sexes differentially) could be assessed for a population, this would be strong evidence of condition-dependent sex allocation.

5.2 Allele frequencies

As I have shown in chapter 3, there are clear examples where females choose to allocate sex according to a male's phenotype, and there is some evidence that this can reflect aspects of a male's genotype. For instance, zebra finch females overproduce the sex of the more attractive parent as determined by leg band colour (Burley 1981). Females also overproduce sons when mated to males with high survival prospects in blue tits (Svensson and Nilsson 1996). Polyandrous female side-blotched lizards *(Uta stansburiana)* produce more sons when mated to large males (Calsbeek and Sinervo 2004).

The existing theory on sex allocation suggests that there will be a tendency for carriers of advantageous alleles to be born the more reproductively variable sex - often males. This means that the frequency of such an allele will increase faster initially, as it sweeps through the male "half" of the population. In extreme cases, this can lead to a graph that has two sigmoid curves, one for the male sweep, and a shallower one for the female sweep. The fact that this is not the fastest way for an allele to reach fixation will be discussed in a separate paper. What is important here is that, depending on the degree of sex ratio control exercised (which includes error introduced by a female's incomplete judgement of the quality of a male's genes - if there is a new allele, as I could be said to assume here, we should not expect her to have a ready-made response to it), we can detect two waves of advance; in an extreme case, two separate sigmoid curves.

Essentially, we would predict that favourable alleles are initially more abundant in males than in females. This throws up two questions: could any other cause have produced this pattern, and, equivalently, what other conclusions might we arrive at if we encountered such data?

Condition-dependent sex allocation generates a very specific shape of allele frequency curves through time, which, if sex allocation is sufficiently accurate, can be detected in plots of allele frequencies from real data. The curve is composed of two separate sigmoid curves, as shown in Figure 5.1. The clarity with which this pattern stands out will depend not only on effective population size (because stochastic variation in allele frequency (genetic drift) will obscure the pattern), and on whether there are other polymorphic loci under selection that are linked to the locus in question, but also on the rigour with which the sex expression decision can be made. With greater error rates, the pattern becomes difficult to pick out by eye, and a more rigorous statistical test would have to be devised for such cases.

When an allele is kept at an intermediate frequency by selection for a long time, we say it is under balancing selection. This can be due to overdominance (heterozygote advantage) or some other form of frequency dependent selection. Its effect is, of course, to keep the allele at an intermediate frequency. However, as the data so far presented show, condition-dependent sex allocation is also

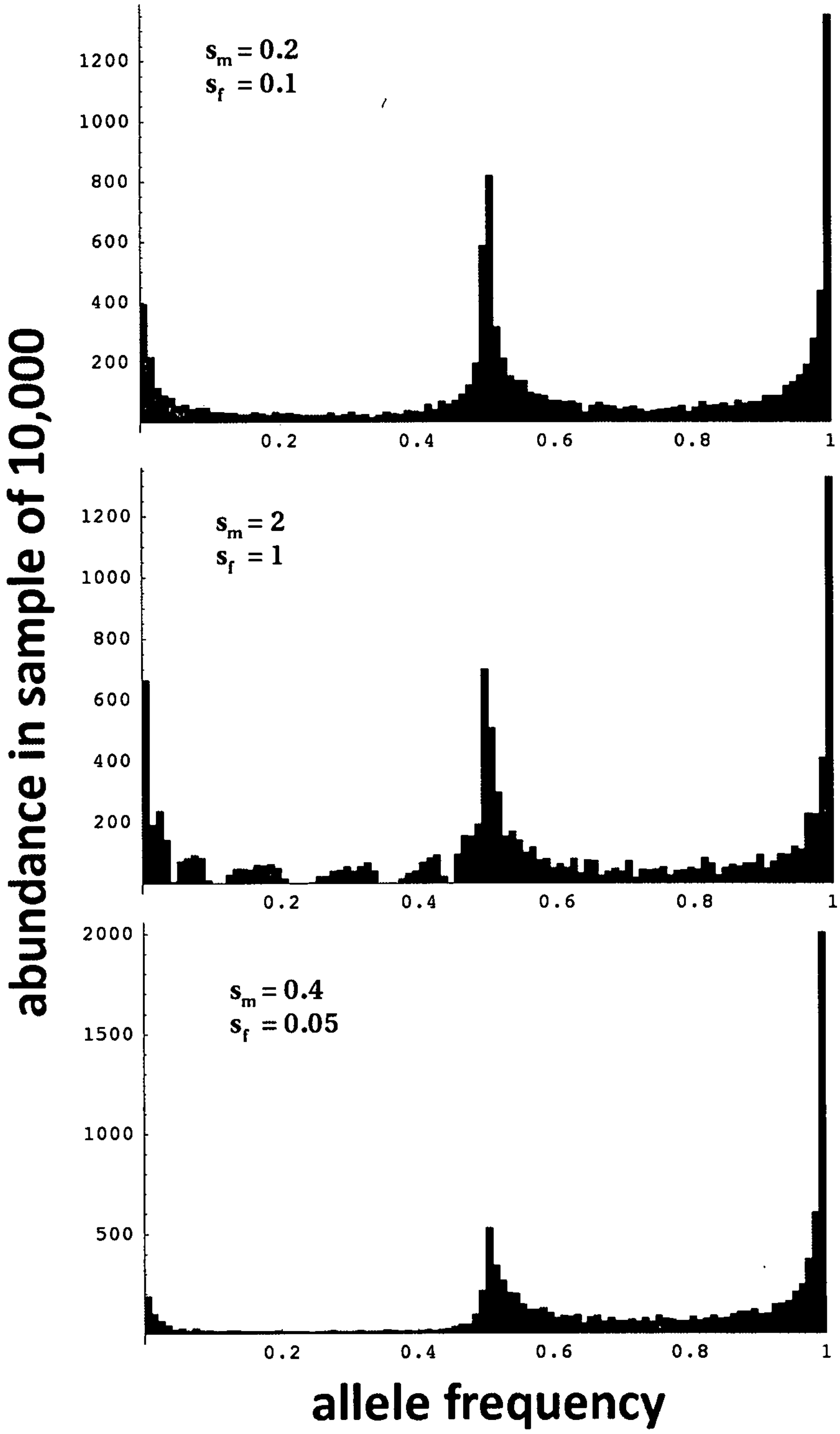


Figure 5.2: The allele frequency distribution changes only with variation in the ratio of s_m and s_f. Each histogram based on 10,000 replicates.

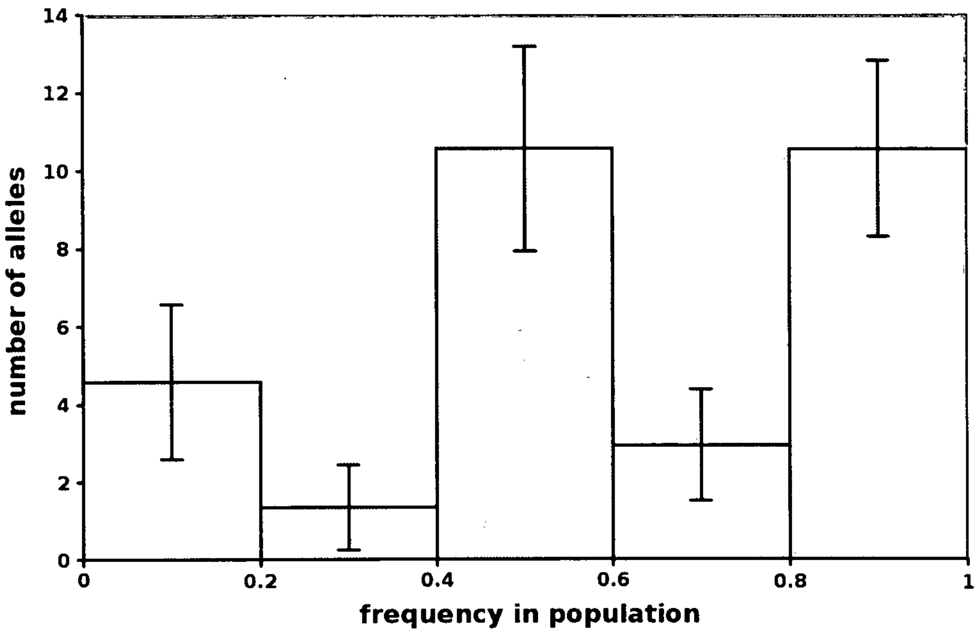


Figure 5.3: Sampling thirty loci that are under sexual selection may be enough to detect condition-dependent sex allocation. Parameter values are $s_m = 0.2, s_f = 0.1$, and the population size assumed infinite. Error bars represent estimated standard deviations of the distribution.

conducive to keeping alleles at intermediate frequencies for several generations, leading to the possibility of false positives when considering only time series of allele frequency data to identify loci under balancing selection (as is of widespread interest in immunology research, where many loci are under balancing selection due to being able to withstand different parasite strains, or because loss of the associated protein prevents pathogen entry, at some fitness cost). However the other characteristic of balancing selection - preservation of ancient polymorphisms - is not shared with condition-dependent sex allocation, and can be used to reliably distinguish the two. In addition, as the number of generations through which a population is being studied increases, so does the statistical power to distinguish between condition-dependent sex allocation and balancing selection.

I should reiterate at this point that because allele frequencies can be estimated cheaply from pools of DNA (Barcellos *et al.* 1997, Johnson 2005), allele frequency spectra are actually much easier to deal with than phenotypic measurements - this is the real, deeper benefit of the allele frequency based methods proposed here.

5.3 Methods

Allele frequencies were drawn from a sample produced by many runs of the single locus, deterministic model illustrated in chapter 3, to virtual fixation (having started with a beneficial allele at low frequency). These values are representative of any population that is large with respect to the number of loci under consideration, so that no clonal interference is observed, as this could potentially distort the distribution of allele frequencies; to avoid clonal interference, it must also be assumed that the loci are on separate chromosomes so that they may segregate entirely independently (or that recombination frequencies happen to be around 0.5).

To obtain this sample from which allele frequencies could be drawn, the simulation was run a large number of times (n=10,000) with initial frequencies uniformly distributed in the range 0.002-0.004, and runs ended when the frequency exceeded 0.999. Generations were discrete, and only one frequency value was retained for each initial frequency. Due to the uniform sampling of initial low frequencies, the values obtained are explicitly not representative of very large populations.

Just to make it explicitly clear - all offspring's sex is determined by condition-dependent sex allocation with an error rate of 0 (the parameter ϵ in the previous chapter).

The idea is that if we can gather data from a large number of loci believed to be under positive as opposed to balancing selection, we can construct a distribution of their frequencies, as shown in Figure 5.2. The data obtained from simulation shows very clearly that a trimodal allele frequency distribution is expected, and can be taken as evidence of condition-dependent sex allocation. On the down side, the strength of this test will be negatively affected by any of the assessed loci being in genetic linkage, and therefore prone to clonal interference effects. However, on the up side, a pronounced mode at a frequency that coincides with the sex ratio of the population, and is likely to be close to 0.5 for many of taxa commonly used for such studies (vertebrates), is quite unlikely to arise from balancing selection, since different loci under independent balancing will have different equilibrium allele frequencies, and so no peak in the frequency of allele frequencies would be observed. Therefore the presence of any such peak, especially when coinciding with the sex ratio, is strongly suggestive of condition-dependent sex allocation. Because of the match between this peak and the sex ratio, the sampling strategy should be adjusted to the sex ratio of the population. When the sex ratio deviates strongly from 0.5, a larger sample will be required to detect this peak, as it will be closer to the two larger peaks caused by alleles that are at, or close to, fixation within your sample. Fig. 5.2 illustrates the fine shape of the distribution. However, Fig. 5.3 illustrates a more realistic sample from a real population, and shows that it is realistic to test for condition-dependent sex allocation using this method.

That is the prediction in its most useful form. However, another related and testable prediction is possible. This next one is relatively trivial, and derives directly from the observation, also stated in chapter 3, that, where a_0 represents the sex ratio among carriers of the lesser allele, and a_1 the sex ratio among carriers of the favourable allele, an ideal strategy would be setting $a_1 = 1$ when $a_0 > 0$, and equally $a_0 = 0$ when $a_1 < 1$. Reciprocally, therefore, the frequency of the preferable allele will always be greater among (in this example) males, representing the sex with greater fitness variance. Even if this strategy cannot be perfectly followed, there may be a significant difference in allele frequency between the sexes in the expected direction (that is, higher frequency of beneficial allele in most birds and mammals, and the opposite prediction where females are more variable in fitness, such as certain sex-changing fish). This prediction is clearly related to the existing tests of phenotypic correlations, and helps to show that the new tests here proposed are straightforward extensions from existing theory. This is a less useful prediction than the previous one in that it requires us to know the sex of each individual whose genotype is being assessed.

5.4 Discussion

I have alluded to several theoretical difficulties with ascertaining conditional sex allocation. Whether conditional sex allocation arises depends on the properties of what I have called the prior distributions of female fecundity and male attractiveness. These prior distributions are not measurable in real populations, so it is important to understand how the properties of real populations may help us to determine relevant properties of the priors; specifically, in order to learn whether sexes might be allocated conditional on some variable, we must determine the ratio of the variances of the two priors.

This may be a suitable place to offer some concluding remarks about variance. We saw in chapter 3 that positively skewed fitness distributions lead to the sex with the higher trait variance also having higher fitness variance at the evolutionarily stable equilibrium. However, we also saw in chapter 2 that in species in whom fertilisations occur quite freely, either through polygamy, or through dispersal of gametes into a pool that many individuals contribute to, a significant amount of fitness variance may be added to the fitness variance of the sex that lacks fertilisation insurance - usually, males. Hence, not only is fitness variance misleading in cases where the fitness distribution is negatively skewed, but it's also likely to mislead when females are the more variable sex through sexual selection, but males gain additional variance from the nature of the mating system - variance that has no adaptive causes and does not contribute to the maintenance of conditional sex allocation. Furthermore, as Figure 2.1 nicely illustrates, fitness variance must be adjusted by the sex ratio to yield any meaningful information, adding another source of data necessary to collect; finally, the applicable sex ratio is one that includes any members of the populations that died after contributing their fertilisation efforts - counting these individuals will prove difficult in the majority of field research situations. My conclusion is that any empirically measured fitness variance should not be trusted, and use of the concept restricted to theoretical discussions.

Chapter 6

Conclusions

6.1 Sex ratios and conservation of species

I have shown that deviations in a species' sex ratio, even as a result of different fitness variances in males and females (chapter 3), can lead to a reduction in the reproductive rate, especially when males are being produced in excess, with females usually the limiting factor of this rate. In the long run, reductions in reproductive rate can lead to extinction - they are the proximal cause of extinction. Sex determining mechanisms that have evolved to produce the correct sex ratio under natural conditions can also produce perilously skewed sex ratios when conditions change - as happened in the very rare New Zealand endemic flightless parrot, the kakapo *(Strigops habroptilus)*. This bird seems to be a case of sex being conditioned on food availability, with males produced when more food is available (Clout *et al.* 2002). Clout *et al.* speculate that males may benefit more from additional food (i.e. males have greater *a priori* reproductive variance) and are thus overproduced when food is artificially supplemented. The male-biased clutches thus produced were an unexpected setback to the conservation programme.

Another speculated case of condition-dependent sex allocation gone wrong is a study of two species of turtle in the Mediterranean, the green *(Chelonia mydas)* and loggerhead *(Caretta caretta)* turtles. This is a case of environmental sex determination in which females are being preferentially produced at higher temperatures. Reece (2003) found that study populations in Northern Cyprus produced a strongly female-biased sex ratio as opposed to other locations.

Moorhouse and McDonald (2005) found that radio-collared populations of the endangered water vole *(Arvicola terrestris)* produced male-biased sex ratios through non-birth of female foetuses. Previous studies had shown that expecting vole mothers that were food-deprived or gained little weight during pregnancy had male-biased litters (Bazhan *et al.* 1996, Nazarova and Evsikov 2000). As Moorhouse and McDonald note, the observed deviation from the normal sex ratio can be equally well explained by local resource competition (Clark 1978) as by condition-dependent sex allocation (Trivers and Willard 1973).

These studies not only show that species - those of the kakapo and water voles - of the impact of conservation and research methods have additional value in that they can be regarded as experimental manipulations of the environment of these animals. In particular, the fact that these animals are not able to produce the correct sex ratio when conditions change - instead markedly overproducing one sex - is interesting with respect to understanding their sex determination mechanisms: particularly, it tells us what sorts of queues they respond to.

However, these studies also show that a correct understanding of how individuals make sex ratio decisions is crucial to conservation biology. To this end, I have presented data showing that in species that engage in condition-dependent sex allocation may be less or more at risk from extinction than randomly sex-allocating species, depending on which is the sex whose fitness increases more with additional investment. Where reliable data on sex allocation patterns is available, this could be used as one of a range of criteria to assess conservation urgency.

6.2 Sex ratio as a measure of male to female variance ratio

Sex ratios can sometimes be inferred, or used to estimate other biologically interesting variables. Nee *et al.* (2002) propose using the sex ratio of protozoan endoparasites as an estimator the Wright's coefficient of inbreeding (Wright 1969; more simply known as f). This sets an important precedent for indirect estimators. However, in chapter 3 I've provided a great deal of evidence to suggest that inferring male to female fitness variance ratio from observed sex ratios is not a reliable estimation even for species known to follow condition-dependent sex allocation. There is interesting further work to be done on this, particularly the question of what information can be gained from partial distributions such as the actual fitness values of individuals in real populations, who generally remain of one sex for all their life, allowing no complete picture of the fitness distribution of males and females.

In the same vein, I have to conclude that fitness variance is not a useful indicator of condition-dependent sex allocation.

6.3 Fitness variance from neutral causes

An interesting collateral result, presented in chapter 2, is that fitness variance can also be increased by a discrepancy in gamete number, particularly when the fertilisation of gametes of one sex is certain. The exact details depend on the number of male and female individuals, and the number of gametes.

6.4 Method to parameterise individual-based simulations with several phenotypes

In chapter 3, I have also suggested a new way to parameterise individual-based simulations of the sex ratio by using inverse distribution functions. This is an elegant solution that will generalise to other cases where two or more phenotypic distributions are highly correlated, and it is desirable for these distributions to remain mathematically well-defined after transformation. To the best of my knowledge, the method is new.

6.5 Population genetics insights

In chapter 4, I introduced the first known model incorporating the effects of both sexual selection (a pre-requisite of condition-dependent sex allocation) and condition-dependent sex allocation itself. I showed that condition-dependent sex allocation has a variety of benefits over random allocation. A modifier model showed that adaptive evolution through selective sweeps with sex-biased selection coefficients also selects for condition-dependent sex allocation under a wide range of parameter combinations. This finding proved fairly robust against variation in recombination rates and linkage disequilibrium, and thus should be robust for finite populations as well.

In the same chapter, I showed that condition-dependent sex allocation has the potential to increase population longevity, and may additionally spread through group selection. This gives an advantage to sexually selected species and could lead to increasing spread. However, as described above, this crucially depends either on the presence of sexually antagonistic alleles, or on females being the sex whose fitness increases more steeply with increasing investment.

6.6 Detecting condition-dependent sex allocation

An important issue concerns whether we are actually able to detect condition-dependent sex allocation. Correlational studies are intensive, and fitness variance, although receiving much attention in the early literature on condition-dependent sex allocation, has proved unreliable.

Condition-dependent sex allocation can, however, be reliably detected using a combination of allele frequency trajectories through time and cross-sectional allele frequency data from polymorphic loci; this combination is necessary to avoid false positives from loci under balancing selection.

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