Sex Ratios

Concepts and Research Methods

Edited by

Ian C.W. Hardy

School of Biosciences, University of Nottingham, United Kingdom

CAMBRIDGE
UNIVERSITY PRESS

PUBLISHED BY THE PRESS SYNDICATE OF THE UNIVERSITY OF CAMBRIDGE
The Pitt Building, Trumpington Street, Cambridge, United Kingdom

CAMBRIDGE UNIVERSITY PRESS
The Edinburgh Building, Cambridge CB2 2RU, UK
40 West 20th Street, New York, NY 10011-4211, USA
477 Williamstown Road, Port Melbourne, VIC 3207, Australia
Ruiz de Alarcón 13, 28014 Madrid, Spain
Dock House, The Waterfront, Cape Town 8001, South Africa

http://www.cambridge.org

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First published 2002

Printed in the United Kingdom at the University Press, Cambridge

Typefaces Swift 9.5/12.25 pt. and Gill Sans *System* LaTeX 2ε [TB]

A catalogue record for this book is available from the British Library

Library of Congress Cataloguing in Publication data
Sex ratios: concepts and research methods / edited by Ian C.W. Hardy.
p. cm.
Includes bibliographical references and index.
ISBN 0 521 66578 7
1. Sex ratio. I. Hardy, Ian.
QH481 .S47 2002
576.8′55–dc21 2001037938

ISBN 0 521 66578 7 paperback
ISBN 0 521 81896 6 hardback

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Chapter 1

Models of sex ratio evolution

Jon Seger & J. William Stubblefield

1.1 Summary

Our understanding of sex ratio evolution depends strongly on models that identify: (1) constraints on the production of male and female offspring, and (2) fitness consequences entailed by the production of different attainable brood sex ratios. Verbal and mathematical arguments by, among others, Darwin, Düsing, Fisher, and Shaw and Mohler established the fundamental principle that members of the minority sex tend to have higher fitness than members of the majority sex. They also outlined how various ecological, demographic and genetic variables might affect the details of sex-allocation strategies by modifying both the constraints and the fitness functions. Modern sex-allocation research is devoted largely to the exploration of such effects, which connect sex ratios to many other aspects of the biologies of many species. The models used in this work are of two general kinds: (1) expected-future-fitness or tracer-gene models that ask how a given sex allocation will affect the future frequencies of neutral genes carried by the allocating parent, and (2) explicit population-genetic models that consider the dynamics of alleles that determine alternative parental sex allocation phenotypes. Each kind of model has different strengths and weaknesses, and both are often essential to the full elucidation of a given problem.

1.2 Introduction

Males and females are produced in approximately equal numbers in most species with separate sexes, regardless of the mechanism of sex determination, and in most hermaphroditic species individuals expend approximately equal effort on male and female reproductive functions. Why should this be so? Sex allocation is a frequency-dependent evolutionary game (Charnov 1982, Maynard Smith 1982, Bulmer 1994). The basic principle that explains why balanced sex ratios evolve so often was described in a limited and tentative way by Darwin (1871), further developed by Karl Düsing (1883, 1884) and several early twentieth century authors, and then summarized concisely by RA Fisher in *The Genetical Theory of Natural Selection* (1930) (Edwards 1998, 2000). Subsequent work has generalized the principle and extended it to cover a great variety of special circumstances to which Fisher's elegant but elementary account does not apply.

Sex allocation is now remarkably well understood, and this understanding is often hailed as a triumph of evolutionary theory. However, to say that the fundamentals may be well understood is not to say that all of the interesting and important discoveries have been made. Despite its focus on a seemingly simple and singular phenomenon, sex-allocation research has become a rich and diverse enterprise that makes contact

with many aspects of biology in a wide range of taxa. The field has continued to yield surprising phenomena and novel insights, and the pace of discovery shows no signs of slowing. The book you are now holding illustrates the field's amazing richness and describes many of the current research frontiers. But because the field is so large, not even a multi-authored volume can cover it all. Reviews of varying emphasis and scope have been provided by Williams (1979), Charnov (1982, 1993), Trivers (1985), Clutton-Brock (1986, 1991), Clutton-Brock and Iason (1986), Karlin and Lessard (1986), Nonacs (1986), Bull and Charnov (1988), Frank (1990, 1998), Wrensch and Ebbert (1993), Bulmer (1994), Godfray (1994), Bourke and Franks (1995), Crozier and Pamilo (1996), Herre *et al.* (1997), Klinkhamer *et al.* (1997), Hewison and Gaillard (1999) and West *et al.* (2000), among many other authors.

In this chapter we introduce the central principle of sex ratio evolution and some of the techniques used to model it. We emphasize basic concepts and issues that appear (at least implicitly) in all models, and we attempt to place these ideas in their historical context.

1.3 Models have always been central

Mathematical models are, and always have been, central to the study of sex ratios. Indeed, it is hard to think of any biological field, associated with specific phenotypes, that is more thoroughly model-driven. Population genetics is also model-driven in this sense, of course, but its models concern genes *in general*; the genes of population genetics are abstracted, intentionally, from any particular class of phenotypic effects.

The sex ratio, by contrast, could hardly be more concrete. This is sometimes forgotten, because every sexual species has a sex ratio (or at least allocation to male and female functions). But in fact the phenotype at issue (the relative numbers of two reproductive morphs) is in many ways an extremely particular and mundane fact of life. Even so, biologists from Darwin to the present have sensed an underlying generality of principle. They have spoken of 'the' sex ratio (singular), as if to understand the sex ratio of any *one* species would be (obviously) to understand the sex ratios of many *others*. Today we have good reasons to view 'the' sex ratio in this way, but most of these were unknown to Darwin. Nonetheless, he initiated the modern discussion of sex ratios, in *The Descent of Man and Selection in Relation to Sex* (1871), by describing the outlines of a quantitative, dynamical model that includes most of the essential features of everything that would follow. Formal mathematical analysis came later, as did direct connections to genetics, and these developments gave rise to a richness that Darwin could not have anticipated. Even so, he saw that there must be a simple underlying principle to be elucidated and then (by implication) applied to a broad diversity of special cases. We still see the subject in this way.

The principle emerges from an analysis of the reproductive consequences of an elementary but generic model of reproduction. The principle is then applied and extended by specifying details that may be left vague in the generic model, which is to say by modifying various implicit and explicit assumptions of the model.

Sex ratio modelling has been an extremely successful enterprise. This success can be attributed to three features of the relationship between the models and reality. First, the relevant biological factors can be specified and represented appropriately in simple mathematical expressions. Second, these factors can be observed and measured in nature, and many of them vary both within and among species in ways that are predicted to change the sex ratios produced by different individuals or species. Third, the fitness differences arising from sex ratio behaviours are often large, so real organisms are expected to show sex ratio modifications at least qualitatively like those predicted by theory, and in fact they often do. In this chapter we focus mainly on the first of these three features of sex ratio research: how biology is represented in models, and how the models are then analysed to uncover

predictions that might (at least in principle) be tested in nature. Other chapters more thoroughly explore the variations that have been incorporated into sex ratio models, and the ways in which experimental and observational data have been used to test these models.

1.4 Darwin's argument

As its title implies, *The Descent of Man and Selection in Relation to Sex* (1871) is really two books merged into one. The book on human origins begins with Chapter I, 'The Evidence of the Descent of Man from some Lower Form', and the book on sexual selection begins with Chapter VIII, 'Principles of Sexual Selection'. Darwin opens the chapter by explaining that sexual selection is 'that kind of selection' that 'depends on the advantage which certain individuals have over other individuals of the same sex and species, in exclusive relation to reproduction' (page 256). Sexual selection is about *relative* advantage in the competition for mates, not about survival or absolute competence to reproduce.

> When the two sexes follow exactly the same habits of life, and the male has more highly developed sense or locomotive organs than the female, it may be that these in their perfected state are indispensable to the male for finding the female; but in the vast majority of cases, they serve only to give one male an advantage over another, for the less well-endowed males, if time were allowed them, would succeed in pairing with the females; and they would in all other respects, judging from the structure of the female, be equally well adapted for their ordinary habits of life.
>
> *(page 257)*

Darwin then describes several kinds of sex differences that seem to make sense on this principle; for example, the generally earlier emergence of male insects. He notes that the intensity of the competition for mates will be a function of the sex ratio and then opens a section titled 'Numerical Proportion of the Two Sexes' (page 263).

> I have remarked that sexual selection would be a simple affair if the males considerably exceeded in number the females. Hence I was led to investigate, as far as I could, the proportions between the two sexes of as many animals as possible; but the materials are scanty. I will here give only a brief abstract of the results, retaining the details for a supplementary discussion, so as not to interfere with the course of my argument. Domesticated animals alone afford the opportunity of ascertaining the proportional numbers at birth; but no records have been specially kept for this purpose. By indirect means, however, I have collected a considerable body of statistical data, from which it appears that with most of our domestic animals the sexes are nearly equal at birth.

Darwin's numbers show rough equality or modest male excesses at birth for various domestic species and for humans. He then points out that 'we are concerned with the proportion of the sexes, not at birth, but at maturity,' because that is when the competition for mates will occur. His data here are less definite, but they suggest greater male mortality and thus a relative *deficit* of males at maturity. However, 'The practice of polygamy leads to the same results as . . . an actual inequality . . . for if each male secures two or more females, many males will not be able to pair; and the latter assuredly will be the weaker or less attractive individuals.' Pages 266–279 then review patterns of polygamy and sexual dimorphism, and pages 279–300 discuss the 'laws of inheritance' of secondary sexual characters.

The chapter then returns to the problem of the sex ratio. Pages 300–315 present a detailed 'Supplement on the proportional numbers of the two sexes in animals belonging to various classes' (humans, horses, sheep, birds, fish and insects). A final short section 'On the Power of Natural Selection to regulate the proportional Numbers of the Sexes, and General Fertility' (pages 315–320) lays out the evolutionary argument. Its second paragraph (page 316) begins as follows:

> Let us now take the case of a species producing . . . an excess of one sex–we will say of males–these being superfluous and useless, or nearly useless. Could the sexes be equalized 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 equalization of the sexes would be brought about. . . . The same train of reasoning is applicable . . . 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.

Parents that produce an excess of the minority sex will be 'more productive' because fewer of their offspring will be 'superfluous'. The paragraph says more of these offspring will 'survive', but this is illogical. Perhaps Darwin meant 'reproduce', or perhaps he was confused about the cause of the differential productivity. The paragraph asserts that parents of the minority sex will enjoy a productivity advantage, no matter which sex is 'produced in excess', and it indicates that the sex in excess will suffer increased failure to mate ('not uniting'). But does the paragraph show *how* these effects modulate parental fitness? It certainly contains all the elements and reaches the right conclusion, but it does not clearly explain why, or in what sense, parents of the minority sex are 'more productive'. In retrospect it comes extremely close (see Sober 1984, Bulmer 1994, Edwards 1998), but it does not explain what will happen in the generation of the parents' grandprogeny.

The next paragraph (pp. 317–318) presents both an advance and a retreat. The advance is an overt anticipation of the concept of parental expenditure or investment (as 'force'). In the previous paragraph, Darwin had explicitly noted the trade-off between numbers of male and numbers of female offspring; in this paragraph he explicitly notes the trade-off between offspring number and offspring quality. Parents that produce fewer 'superfluous males' but 'an equal number of productive females' would probably benefit, as a consequence, from 'larger and finer' ova or embryos, and 'their young [would be] better nurtured in the womb and afterwards.' In support of this idea, Darwin notes that inverse relationships between seed number and seed size can be seen both among and within species of plants. 'Hence the offspring of the parents which had wasted least force in producing superfluous males would be the most likely to survive, and would inherit the same tendency not to produce superfluous males, whilst retaining their full fertility in the production of females. So it would be with the converse case of [an excess of] the female sex.'

The retreat is a muddled explanation of the disadvantages experienced by 'superfluous' offspring. For purposes of argument, Darwin had begun the paragraph assuming that there was an excess of males, and that some parents produced fewer of them but a typical number of females. 'When the offspring from the more and the less male-productive parents were all mingled together, none would have any direct advantage over the others.' This is not true in the sense that he seems to intend. The offspring might be equivalent *individually* (leaving aside the 'indirect' benefits noted above), but not *collectively*; parents that produced more males would have more descendants *through males* than those that produced fewer males, given that the offspring 'were all mingled together'. In this sense sons are not 'superfluous' even when produced in excess. Darwin seems to be imagining that parents that contribute to the male excess will have no more grandoffspring through their sons (collectively) than those parents that refrain from producing excess males.

In the second edition of the *Descent* (1874), most of Chapter VIII is similar to that of the first edition, but the final section is completely different. It is renamed 'The proportion of the sexes in relation to natural selection', and it consists mainly of an inconclusive discussion of the relationship between sex-biased infanticide and the primary sex ratio. It concludes:

> In no case, as far as we can see, would an inherited tendency to produce both sexes in equal numbers or to produce one sex in excess, be a direct advantage or disadvantage to certain individuals more than to others; for instance, an individual with a tendency to produce more

> males than females would not succeed better in the battle for life than an individual with an opposite tendency; and therefore a tendency of this kind could not be gained through natural selection. . . . I formerly thought that when a tendency to produce the two sexes in equal numbers was advantageous to the species, it would follow from natural selection, but I now see that the whole problem is so intricate that it is safer to leave its solution for the future.

Why did Darwin abandon his own previous argument which was close to the 'solution' and clearly moving in the right direction? On one reading of the 1874 retraction, he considers the 1871 argument to be flawed by a reliance on species-benefit reasoning. Consistent with such an interpretation, the paragraph laying out the evolutionary argument (1871, p 316) includes an extraneous and confused aside on the adjustment of fertility, which we deleted from our earlier quotation.

> . . . 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 equalized 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.

Why Darwin should invoke, here, the concept of species' benefit (or need!) seems baffling. Two pages later, as promised, he describes in two paragraphs how reduced fertility (offspring number) could evolve by ordinary natural selection, given trade-offs between maintenance and reproduction, and between offspring number and quality. These two paragraphs end the chapter and brilliantly anticipate late-twentieth-century developments in life-history theory. They contain no species-benefit reasoning that we can detect. Darwin credits Herbert Spencer's *Principles of Biology* (1867) for inspiration on this subject.

It seems odd that Darwin should have lost his nerve and failed to correct confusions that were probably no worse than hundreds that he must have surmounted in other contexts. His decision to remove the entire argument from the second edition of the *Descent* (1874) can be taken to support the view that he never really understood the principle as well as a generous reading might suggest he did at the time he wrote it. He sees a close connection between sexual selection and the sex ratio: as the number of males competing for each productive mating increases, their average reproductive success must decrease. But he does not seem to recognize that he should directly compare the average fitnesses of males and females, and that he should evaluate the fitnesses of parents by counting their grandprogeny. In any case, his decision to remove the evolutionary argument from the second edition undoubtedly changed the history of behavioural ecology. The second edition was reprinted far more extensively than the first and became the edition read by almost everyone, including RA Fisher (Edwards 1998).

The recognition that sex ratios evolve through negatively frequency-dependent selection on the relative reproductive success of male and female offspring is traditionally attributed to Fisher (1930). His two-page verbal argument is informed by a knowledge of genes and it is far more lucid than Darwin's, but otherwise it is very similar in spirit. Why does Fisher not credit Darwin? One explanation is that, like most of his contemporaries, Fisher had read the second edition of the *Descent* and understandably failed to see any reason to persue Darwin's hint about what he 'formerly thought'. Edwards (1998) has shown that Darwin's initial lead was picked up by Düsing and several early twentieth century authors who further clarified the argument, and that Fisher was almost certainly aware of at least some of these later works. Why does Fisher not cite them either? Edwards suggests that Fisher understood his own account of the principle to be derived from these sources, that he assumed his interested contemporaries also would have been aware of them, and that standards of scholarly attribution were not as strict in 1930 as they are today. These factors could

explain why Fisher (1930) presents the principle so casually.

1.5 The elements of a sex ratio model

A fully specified model of evolutionary adaptation can be viewed as a proposal showing how certain biological circumstances will give rise to a *fitness function* and a set of *constraints*. These relations are typically referred to as the *assumptions*, because the modeller is free, in principle, to change them in arbitrary ways. Models based on relatively 'realistic' assumptions are often considered more scientifically 'interesting' than those based on unrealistic assumptions, but, as Fisher himself points out in the preface to *The Genetical Theory of Natural Selection* (1930), models cannot really illuminate the natural world without also illuminating unnatural worlds. 'No practical biologist interested in sexual reproduction would be led to work out the detailed consequences experienced by organisms having three or more sexes; yet what else should he do if he wishes to understand why the sexes are, in fact, always two?' A model becomes explicitly mathematical when it embodies its assumptions ('three sexes', for example) in a set of formal quantitative relations that can be evaluated to reveal expected evolutionary outcomes. These deductions, following from the assumptions, can be interpreted as predictions about what would be expected to happen if the world actually worked as the assumptions propose it does. Such a derivation of expected consequences of the assumptions is often referred to as an *analysis* of the model.

There are two distinctive but complementary approaches to setting up and analysing explicit sex ratio models. The older, more intuitive and more expressive approach employs 'expected-future-fitness' calculations similar in spirit to those used in many inclusive-fitness and quantitative-genetic models. In this approach, sex-allocation strategies are evaluated with respect to the expected future frequencies of selectively neutral genes (tracers of descent) carried by an individual parent that exhibits a given sex ratio phenotype. The younger, more rigorous but less transparent approach employs dynamical population-genetic models to ask under what circumstances an allele that determines a specific parental sex ratio phenotype can invade (or fix) against an allele that determines a different phenotype. Both kinds of models can vary widely in sophistication and complexity. Neither is inherently 'better'; the choice of approach is largely a matter of taste and the nature of the problem being considered (see Bulmer 1994). We will illustrate both approaches.

Even in its original verbal form, the Darwin–Fisher argument is a legitimate (if primitive) sex ratio model. It is only marginally mathematical, but that does not disqualify it as a model. The relevant assumptions are clearly identified, most importantly: (1) that sex-specific fitness differences arise from an inevitable 'competition' for mates, which implies a *fitness function*, and (2) that parents that produce more sons (or daughters) must necessarily produce fewer daughters (or sons), which implies a *constraint*. The implicitly quantitative analysis proceeds as follows. Parents that overproduce the minority sex will have offspring that enjoy greater than average reproductive success, on average. Therefore, any heritable variants that tend to cause overproduction of the minority sex will increase in frequency, and as they do so the sex ratio imbalance will decrease. Because this is true no matter which sex is currently under-represented, there must be a stable evolutionary equilibrium at which male and female offspring are produced in approximately equal numbers. If all parents were to produce equal numbers of females and males, then no other sex ratio phenotype could increase under selection. Today we would call this unbeatable phenotype an 'evolutionarily stable strategy' or ESS (Maynard Smith & Price 1973).

1.6 Düsing's model

The first general mathematical treatment of sex ratio evolution has long been attributed to Shaw and Mohler (1953), who derived an elegant formalization of Fisher's argument. However,

Edwards (1998), in reconstructing Fisher's sources, discovered that a similar mathematical treatment had been published almost 70 years earlier by Karl Düsing. His Ph.D. dissertation (Düsing 1883, expanded to book length in 1884) is mainly a study of factors associated with variation in progeny sex ratios in 'man, animals and plants' (e.g. maternal age and parity). In the early pages of this work, Düsing poses and answers a question that leads him to construct what is undoubtedly the first formal sex ratio model and perhaps the first mathematical model in evolutionary biology.

Given that animals vary their sex ratios in response to particular conditions of life, why do we not see large overall sex ratio imbalances? The reason, Düsing says, is that deviations from a balanced sex ratio will tend to be self-correcting: an excess of one sex provides a reason to produce more of the other. To make the argument concrete, he assumes a population in which there is a lack of females, and points out that *all the males together have the same number of offspring as all the females*. Because the latter are (by assumption) in the minority, each will have on average more offspring. For example, if there are x females and nx males, and if they produce z offspring in all, then each female will produce z/x offspring and each male will produce z/nx (Düsing 1884 p. 10, see Edwards 2000 for a full translation of the argument). He points out that if a female produced more female offspring, these daughters would produce, collectively, a larger than average number of offspring. Suppose a female produces A sons and a daughters, and another produces the converse (A daughters and a sons). The first will have

$$A\frac{z}{nx} + a\frac{z}{x} \tag{1.1}$$

grandchildren and the second will have

$$a\frac{z}{nx} + A\frac{z}{x}. \tag{1.2}$$

If we assume that $A > a$, such that $A = ba$ (with $b > 1$), then the first female will contribute

$$\frac{az}{x}\left[\frac{b}{n} + 1\right] \tag{1.3}$$

individuals to the second generation, while the second female will contribute

$$\frac{az}{x}\left[\frac{1}{n} + b\right] \tag{1.4}$$

which is

$$\frac{1 + bn}{b + n} \tag{1.5}$$

times as many. Düsing notes that if the population sex ratio is balanced ($n = 1$), then this expression evaluates to 1 for any sex ratio. *No matter what progeny sex ratio a female produces, she will have the same number of descendants in the second generation.* But not so if the sex ratio is unbalanced. For example, if there are twice as many males as females, then the ratio of grandchildren will be

$$\frac{1 + 2b}{b + 2} \tag{1.6}$$

as a function of the difference in progeny sex ratios (b). Düsing contrasts the fitnesses of two females for which $b = 3$; the one producing a threefold female excess has 7/5 as many grandchildren as the one producing a threefold male excess.

In less than two pages, Düsing both clarifies Darwin's argument and quantifies it. He identifies the key underlying fact that total male and female fitnesses must be equal; he identifies relative numbers of grandchildren as the appropriate measure of fitness; he writes a general expression for fitness as a function of the parent's progeny sex ratio b given the population sex ratio n; and he discovers that fitness is unaffected by progeny sex ratios if and only if the population sex ratio is balanced (in effect, the ESS argument). Having given a general theoretical reason why progeny sex ratio adjustment might be advantageous to individuals, he then embarks on a massive empirical review of such adjustments and their correlates in many species. Apparently this subject was as interesting and controversial in Düsing's time as it is today; his analyses of the patterns were much discussed, and his evolutionary model was forgotten (Edwards 1998, 2000, SH Orzack, pers. comm.).

1.7 Fisher's equal-investment principle

Fisher's (1930) explanation of the sex ratio principle is as brief as Düsing's, but purely verbal and very well known, so we will not dwell on it here except to note that it presents a very important generalization of the earlier arguments. Fisher carefully considers the nature of the constraint on male and female offspring production, and discovers that the sex ratio equilibrium concerns the distribution of parental effort or 'expenditure' (later generalized by Trivers 1972 as 'investment'), not numbers *per se*. For example, suppose daughters are twice as costly to produce as sons. Then a parent with the resources to produce 12 sons might instead produce six daughters. What is the evolutionary equilibrium in this case? At a numerical sex ratio of 1:1 a typical parent could have four sons and four daughters. Males and females will have equal average reproductive success, so a rare male-specialist parent (with 12 sons) would have many more grandoffspring than an average parent (with eight offspring in all), and this advantage would increase the proportion of male-specialist parents (and males) in future generations. Only when males became twice as numerous as females (six sons and three daughters in a typical brood) would parents become evolutionarily indifferent to the sexes of their offspring. Sons would be only half as successful as daughters, but also only half as expensive. Thus, over the population as a whole, we expect to find equal expenditure or investment in the two sexes, not necessarily equal numbers.

This generalization leads immediately to testable predictions. In species where one sex is more costly (to parents) than the other, that sex should tend to be produced in correspondingly smaller numbers. This prediction has held up well in many recent studies of sexually dimorphic social and solitary Hymenoptera. Fisher was aware that human males suffer higher mortality rates in childhood than do females, rendering them less costly per infant born. He argues that the slight but conspicuous male excess at birth is plausibly an adjustment to equalize overall investment in the sexes (at least under patterns of mortality that would have existed in early human societies). This example illustrates the logic that has been used many times since then to connect sex allocation with other aspects of biology.

1.8 Genetic models I: tracer genes and the Shaw–Mohler equation

Shaw and Mohler (1953) set out to formalize Fisher's argument and connect it more closely to genetics. Their model is extremely simple and transparent, and it forms the basis of most subsequent sex ratio models. The key idea is to calculate the contribution that a parent in one generation (P) makes to the gene pool in the second descending generation (that of its offspring's offspring, G_2), if the parent produces a sex ratio x (proportion males) in the G_1 (offspring) generation where the average sex ratio is X. The focal parent produces n offspring in all, and the population at large produces N. In G_1 there will be NX males and 'all together they will supply half the genes which are transmitted from G_1 to G_2', so each male's share will be $1/2NX$. The focal parent's sons therefore contribute $nx/2NX$ of the genes in G_2, and its daughters contribute $n(1-x)/2N(1-X)$, for a total of

$$\frac{1}{2}\left[\frac{nx}{NX}+\frac{n(1-x)}{N(1-X)}\right]. \tag{1.7}$$

The parent contributes half of the genes carried by each of its nx sons and $n(1-x)$ daughters in G_1, so its net genetic contribution to G_2 is

$$C=\frac{1}{4}\left[\frac{nx}{NX}+\frac{n(1-x)}{N(1-X)}\right], \tag{1.8}$$

or

$$C=\frac{1}{4}\left(\frac{m}{M}+\frac{f}{F}\right), \tag{1.9}$$

'where m and f are the numbers of male and female zygotes in the [focal] progeny while M and F stand for the corresponding numbers in the entire G_1'. C is a measure of genetic fitness because it can be interpreted as the expected frequency in

future generations of a selectively neutral tracer allele that in generation P was carried only by the focal parent. If the population sex ratio is balanced ($X = 1/2$), then $C = n/2N$ independent of the focal parent's sex ratio (as long as n does not depend on x). But if X is any other value, then some sex ratios x will give rise to larger contributions than others. 'The gene or genes favored are always those whose increase will shift the population sex ratio (X) toward 0.5.'

The paper goes on to show that the equilibrium progeny sex ratio is not affected if male and female offspring, once produced, survive to adulthood with different probabilities; these probabilities cancel out of the expressions for m/M and f/F. Curiously, the paper does *not* extend the analysis to include sexually dimorphic mortality rates *during* the period of parental care, or other sources of differential offspring *costs*, even though Fisher considered this extension verbally and noted the implication that m, M, f and F can be interpreted more generally as net parental expenditures on behalf of male and female offspring. Bodmer and Edwards (1960) modelled Fisher's argument by writing an expression for the reproductive value produced by a unit of parental expenditure, given sex-specific intrinsic costs and probabilities of surviving the period of parental care. This *rate of return* measures 'the selective advantage attached to reproduction with particular sex and parental expenditure ratios'; it is independent of the focal parent's progeny sex ratio when 'the total parental expenditure incurred in respect of children of each sex is equal', confirming 'Fisher's Law'.

Because the *total* (population-wide) male and female investments M and F are directly proportional to the *average* (individual) investments, we are free to normalize the Shaw–Mohler equation to give an average fitness of 1, in keeping with modern conventions in other areas of population genetics

$$W = \frac{1}{2}\left(\frac{f}{F} + \frac{m}{M}\right), \tag{1.10}$$

where F is the average value of f in the population and M is the average value of m. A simple analysis that explicitly incorporates differential costs can then be carried out as follows. The constraint on allowable combinations of female and male offspring can be represented by an equation that specifies the number of daughters that a parent will produce if it also produces m sons. For example, assume the simplest kind of linear trade-off between male and female production, and let each daughter cost c times as much as a son. Then the constraint is $cf + m = r$, or

$$f = \frac{r - m}{c}, \tag{1.11}$$

where r is the total resource available for offspring production (in units of the cost of a son). Substituting the constraint (eq. 1.11) into the fitness function (eq. 1.10) we get

$$W = \frac{1}{2}\left(\frac{\frac{r-m}{c}}{\frac{R-M}{c}} + \frac{m}{M}\right). \tag{1.12}$$

Note that the relative cost of a female (c) cancels out, and that without any loss of generality we are free to set the average resource (R) equal to 1. Thus the constrained fitness function simplifies to

$$W = \frac{1}{2}\left(\frac{r - m}{1 - m} + \frac{m}{M}\right). \tag{1.13}$$

What sex ratio (m) will maximize our parent's fitness, given that it has r units of resource? Of course we already know what the answer is supposed to be: parents should expend equal amounts of resource on each sex. If that's what *typical* parents are doing, then $M = {}^1/_2$, and the fitness function further simplifies to

$$W = [(r - m) + (m)] = r. \tag{1.14}$$

Now the parent's sex allocation (m) also cancels out, and its fitness is simply equal to its resource pool. We have explicitly derived the result that each parent is indifferent to the sexes of its own offspring, even where male and female costs differ, as long as there is equal overall *investment* in the population at large ($M = {}^1/_2$).

Of course parents are far from indifferent when overall investment is not equal, and the Shaw–Mohler equation quantifies the fitness differences associated with atypical ('mutant') progeny sex ratios in populations that are away from the evolutionary equilibrium (Figure 1.1). Not only do parents that invest equally in sons and daughters do better than average when most other parents are investing unequally, but parents that over-compensate do even better, and

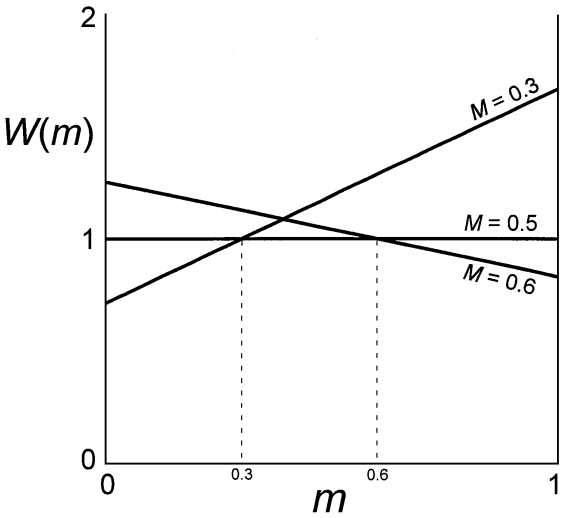

Fig 1.1 Fitness as a function of brood sex ratio in populations with different average sex ratios, as described by the Shaw–Mohler equation. $W(m)$ is the parent's expected genetic represention in the generation of its grandprogeny, given that it has one unit of resource ($r = 1$) which it uses to produce m male and $1 - m$ female offspring in a population where the average proportional allocation to males is M (0.3, 0.5, or 0.6).

those that produce the under-represented sex exclusively do best of all. As we mentioned earlier, the selection coefficients associated with sex ratio differences can be huge relative to those believed to account for much real adaptive evolution, and they can be large relative to those needed theoretically to overpower drift even in very small populations. For example, consider a population out of equilibrium by only 1% ($M = 0.49$). Then a parent with one unit of resource will have a fitness of

$$W = \frac{1}{2}\left(\frac{1-m}{0.51} + \frac{m}{0.49}\right). \tag{1.15}$$

A typical parent ($m = 0.49$) has a fitness of 1.0, while a nearly identical 'Fisherian' parent ($m = 0.5$) has a fitness of 1.0004 (0.04% above average) and a fully overcompensating parent ($m = 1$) has $W = 1.02$ (2% above average). The Fisherian ($m = {}^1/_2$) parent's advantage increases rapidly with the size of the population's deviation from equilibrium, reaching 1% when the deviation reaches 5% ($M = 0.45$ or $M = 0.55$).

In deriving this model we made some simplifying assumptions that do not always hold true. For example, we assumed that the population is effectively infinite and randomly mating, that generations are discrete and nonoverlapping, that the constraint on male and female offspring numbers is linear ($cf + m = r$) and that the fitnesses of individual female and male offspring are independent of brood sex ratios. With respect both to their production and to their reproductive values, positively and negatively synergistic interactions between sons and daughters can be imagined and, for certain taxa, documented. The Shaw–Mohler framework can be extended to allow for such nonlinearities, and two classic examples (local mate competition and hermaphroditic plants) are considered in section 1.10. The fitness function can be expanded to account for differences in ploidy (e.g. haplodiploidy), to account for differences in the focal parent's (or other caregiver's) relatedness to male and female offspring (e.g. workers in social Hymenoptera), to account for differences in situation-specific male and female fitnesses (e.g. offspring of high- and low-ranking mothers in some ungulate and primate species), and to account for overlapping generations (see Chapter 2).

Although straightforward in principle, these and other extensions may greatly complicate the analysis of the resulting model. Since both the evolutionary and analytical objectives are to maximize $W(m, f)$ subject to constraints, techniques from optimal control theory and other branches of applied mathematics are sometimes used to find the evolutionarily stable allocation strategies (e.g. Macevicz & Oster 1976, Oster & Wilson 1978). Probabilistic approaches may also be necessary, as in the small-population case where stochastic fluctuations of the sex ratio will be large and the total allocation to males and females (M, F) will not be effectively independent of the focal parent's allocation (m, f). Here parents are not indifferent to their own progeny sex ratios even when the population is at its evolutionary equilibrium, as first noted by Verner (1965).

MacArthur (1965) identified an interesting corollary of the Shaw–Mohler formulation that holds in many but not all models with nonoverlapping generations: at equilibrium the *product* of the *numbers* of females and males ($N_f N_m$) is maximized, even where individuals of one

sex cost more than the other (see Maynard Smith 1978, 1982, Charnov 1982, Karlin & Lessard 1986).

At this point it may be useful to review the core assumptions and logic of this historic neutral-gene-transmission model that still strongly influences the way we conceive and analyse selection on sex ratios. The evolutionary payoff associated with a given sex ratio phenotype is assumed to be proportional to the reproduction of the parent's offspring. Shaw and Mohler explicitly invoke the transmission of genes, and even refer to their paper as a discussion of the 'population genetics of autosomal genes affecting the primary sex ratio', although no alleles or gene frequencies appear anywhere in it. (Düsing knew nothing of genes, of course, but intuitively knew that maternal and paternal contributions would be of equal evolutionary importance.) Thus the sex ratio differences among parents are assumed to be caused at least in part by genetic variants that the parents will transmit to their offspring. Then, as Darwin almost argued, the offspring of parents that over-produce the under-represented sex will have more offspring, and their offspring will (as he did argue, but then doubted) 'inherit a tendency to procreate fewer' of the over-represented sex and more of the under-represented sex, so that 'a tendency towards the equalization of the sexes [will] be brought about'.

In the Shaw–Mohler formulation, this reasoning is embodied in an equation that expresses the total expected reproduction by offspring of parents that produce different sex ratios in a population with a given overall sex ratio. This measure of fitness is explicitly constructed to reflect the transmission to distant generations of selectively neutral genes carried by the parents. We find that overproducing the minority sex (more generally, the under-invested sex) always yields greater than average fitness. We interpret fitness (defined in this way) as a metric indicating the expected evolutionary fates of alleles that incline their bearers to produce different progeny sex ratios; such alleles are implicitly ones of small individual effect, possibly occurring at many different genetic loci scattered throughout the genome. We conclude that a population fixed for a 'Fisherian' genotype ($m = M = {}^1/_2$) should not be subject to invasion by male- or female-biasing alleles at any loci.

With the benefit of hindsight we may be tempted to view this argument as air-tight. After all, its conclusion is known to be correct. But it rests on some assumptions that could have proved troublesome. We glossed over all of the gritty mechanistic details that connect the phenotypes caused by particular alleles to the transmission of those same alleles. A moment's thought is all it takes to see that male-biasing alleles will tend to accumulate in males, while female-biasing alleles will tend to accumulate in females (Shaw 1958, Nur 1974). Thus the frequencies of alleles affecting sex ratios will differ between the sexes (as discussed in the next section and illustrated in Figure 1.3). Might this affect the evolutionary outcome in a species where sex ratios are determined by just one parent (say, the mother)? The answer is not obvious, so we need also to construct and analyse dynamic population-genetic models in which these potentially critical connections are represented explicitly.

1.9 Genetic models II: alleles that determine parental sex ratios

We were able to develop the logic of expected-future-fitness models along historical lines, because the history begins simply and then adds layers of complexity. By contrast, the history of models with alleles that specifically affect parental sex ratios is not so straightforward. The first models were, for the most part, relatively complex, opaque and lacking in generality, so they do not provide good examples with which to introduce the subject as presently understood. For this reason we will first describe a more highly derived but simple and generic model, and then look back briefly at some pioneering models from the literature.

Genetic evolution will not occur unless the genome includes at least one locus with two or more different genotypes that tend to produce different phenotypes. Often we can reasonably assume that what's true for one locus will be true (qualitatively) for others, in which case the

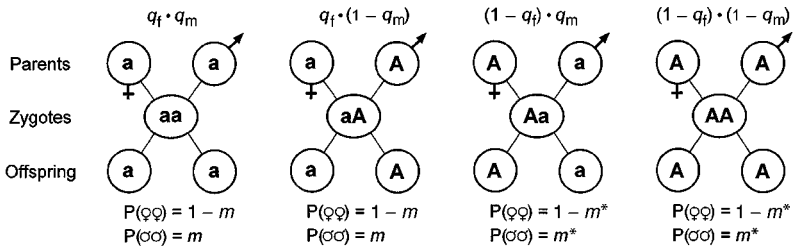

Fig 1.2 Mating types and offspring productions in a haploid genetic model. Alleles **a** and **A** occur at frequencies q_f and $1 - q_f$ in females and q_m and $1 - q_m$ in males. Male and female parents mate at random, so the four mating types occur in the proportions indicated above the pedigrees. Diploid zygotes form briefly and then undergo meiosis to form haploid spores that develop into the next generation of adults. Brood sex ratios are determined by the mother's genotype (m for mothers of genotype **a**, and m^* for mothers of genotype **A**).

problem can be represented adequately by just one locus with two alleles. Often it is also reasonable to let the species be haploid, so as to reduce the number of distinct genotypes to an absolute minimum. If we assume, in addition, that progeny sex ratios are determined by the mother's phenotype, and that parents mate randomly with respect to their sex ratio genotypes, then we have defined the very simple model that is shown in Figure 1.2 and summarized algebraically in Table 1.1. Each row in the table represents one of the four mating types illustrated in the figure. Alleles **a** and **A** act in the mother to determine her expected progeny sex ratio, m or m^*, respectively. The frequency of **a** is q_f in females and q_m in males. The entries under 'daughters' and 'sons' are the expected proportions of each progeny resulting from matings of a given type (row) that will be females or males of genotypes **a** or **A** (columns).

The *state variables* in this model are the genotype frequencies q_f and q_m. We want to know whether these gene frequencies will change and, if so, in which direction. To do this we need to write equations for q'_f and q'_m (the allele frequencies *next* generation) as functions of q_f and q_m (the allele frequencies *this* generation). This may sound difficult, but in fact it is easy given the preliminary calculations we have already placed in the table.

By definition, q'_f is the *proportion* of all daughters that will be of genotype **a**. The *total* production of daughters is $q_f(1-m)+(1-q_f)(1-m^*)$. This expression goes in the denominator. For the numerator we need the total production of daughters *of genotype* **a**. This can be read directly from the table, as the sum of the products formed by multiplying each term in the 'frequency' column by the term in the 'daughters/**a**' column of the same row. Thus the *recurrence equation* for the female genotype frequency is

$$q'_{\mathrm{f}} = \frac{q_{\mathrm{f}}q_{\mathrm{m}}(1-m)+\frac{1}{2}q_{\mathrm{f}}(1-q_{\mathrm{m}})(1-m)+\frac{1}{2}(1-q_{\mathrm{f}})q_{\mathrm{m}}(1-m^*)}{q_{\mathrm{f}}(1-m)+(1-q_{\mathrm{f}})(1-m^*)}. \tag{1.16}$$

To make the origin and meaning of each term as easy to see as possible, we have written the equation without any further algebraic simplifications. By a similar train of reasoning, we obtain

Table 1.1 Frequencies and outputs of the four mating types in a haploid model

		Daughters		Sons	
Mating	Frequency	a	A	a	A
a × a	$q_f q_m$	$(1-m)$		m	
a × A	$q_f(1-q_m)$	$\frac{1}{2}(1-m)$	$\frac{1}{2}(1-m)$	$\frac{1}{2}m$	$\frac{1}{2}m$
A × a	$(1-q_f)q_m$	$\frac{1}{2}(1-m^*)$	$\frac{1}{2}(1-m^*)$	$\frac{1}{2}m^*$	$\frac{1}{2}m^*$
A × A	$(1-q_f)(1-q_m)$		$(1-m^*)$		m^*

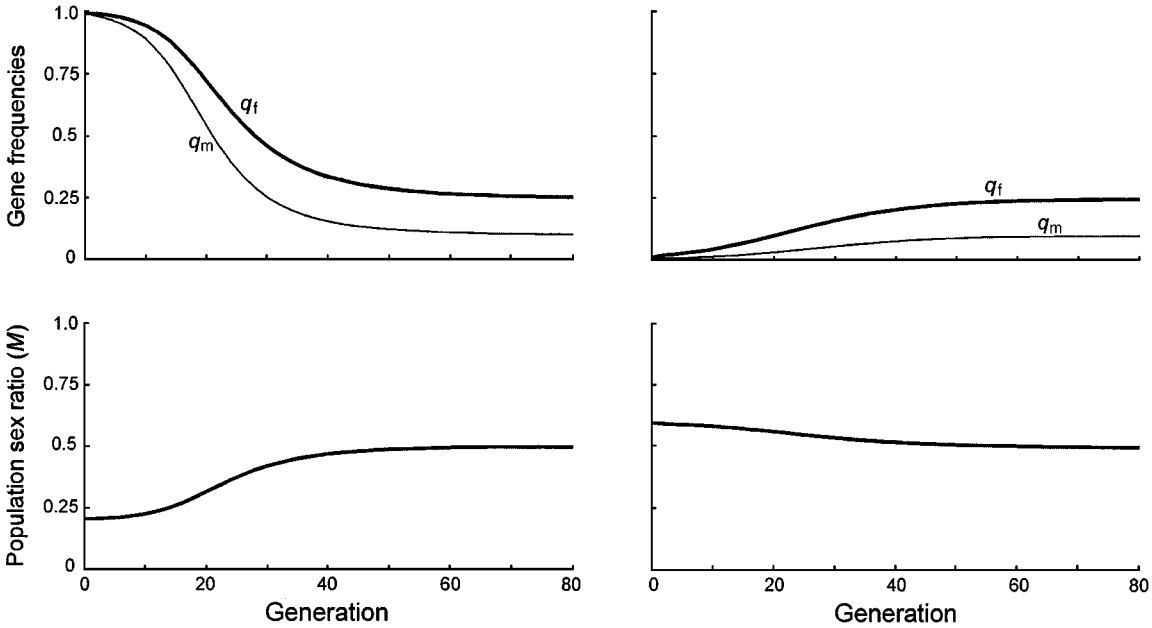

Fig 1.3 Allele frequency and population sex ratio trajectories for the two-allele haploid model. Mothers of genotype **a** produce broods with 20% males ($m = 0.2$) regardless of their mate's genotype, and mothers of genotype **A** produce 60% males ($m^* = 0.6$). The population illustrated in the panels on the left begins from an allele frequency of $q = 0.99$, and the population on the right begins from $q = 0.01$.

the corresponding recurrence equation for the male allele frequency

$$q'_{\mathrm{m}} = \frac{q_{\mathrm{f}} q_{\mathrm{m}}(m) + \frac{1}{2} q_{\mathrm{f}}(1 - q_{\mathrm{m}})(m) + \frac{1}{2}(1 - q_{\mathrm{f}}) q_{\mathrm{m}}(m^*)}{q_{\mathrm{f}}(m) + (1 - q_{\mathrm{f}})(m^*)}. \tag{1.17}$$

Everything on both right-hand sides is known, so by evaluating this pair of equations we obtain the genotype frequencies for the next generation; these can then be used to obtain the genotype frequencies for the generation after that, and so on for as long as we care to *iterate* this dynamical system. Doing so by hand would be tedious, but it is easy by computer. Figure 1.3 illustrates two such calculations. Given alleles with phenotypic values flanking $^1/_2$ (in this case, $m = 0.2$ and $m^* = 0.6$), the system always converges to genotype frequencies that give $M = {}^1/_2$ (in this case, $q_f = 0.25$). Males have a much lower frequency of the female-biasing allele **a** ($q_m = 0.1$ at equilibrium) because a disproportionate number of their mothers carry the male-biasing allele **A**. Despite this complication, our conclusion from the expected-future-fitness approach is supported, at least for this genetic system.

Any population-genetic model that can be written down in this way (as a set of recurrence equations) can be studied by iteration in the manner illustrated in Figure 1.3. For example, Shaw (1958) did this for a diploid model (without the benefit of a computer!), and Hartl and Brown (1970) did so for a haplodiploid model. But each set of parameters and initial conditions considered in this way is just an anecdote. Even in the present very simple model, there are infinitely many combinations of m and m^*. Since not all of them can be considered, how are we to be confident of any general conclusions we might want to draw about the model's behaviour throughout certain regions of its parameter space?

Fortunately, we can often obtain rigorous general results by restricting our attention to the dynamics of *invasion* and *fixation*. We consider the system's behaviour at the 'boundary' of the state space, where one allele (**A**) is nearly fixed and the other (**a**) is vanishingly rare. Here the system can be represented as a set of linear equations far simpler than the full system. In

matrix form, the system can be written $\mathbf{q}' = \mathbf{M}\mathbf{q}$ where $\mathbf{q}'$ and $\mathbf{q}$ are vectors of the (infinitesimal) genotype frequencies (here, q_f and q_m) and $\mathbf{M}$ is a matrix of coefficients representing the system's dynamics in the immediate neighbourhood of the boundary. The elements of $\mathbf{M}$ are partial derivatives of the full recurrence equations evaluated at $\mathbf{q} = 0$, where allele **a** is imagined to have just entered the population as a very rare migrant or a new mutation. As long as **a** remains very rare, the linearized system accurately represents the behaviour of the full system. Standard techniques of linear algebra allow us to determine under what conditions a small 'disturbance' of the equilibrium (i.e. tiny positive allele frequencies) will grow. Typically this is all we really care about because we need only show that an allele with a certain value of m^* will not be invaded by any allele with an m different from m^*. This is usually a straightforward task.

In the present case, as in many, the matrix $\mathbf{M}$ takes a simple and illuminating form. Here we write it out explicitly, with its associated vectors $\mathbf{q}'$ and $\mathbf{q}$, and the result of the multiplication, so as to make the meanings of all the terms easy to grasp.

$$\begin{bmatrix} q'_{\mathrm{f}} \\ q'_{\mathrm{m}} \end{bmatrix} = \begin{bmatrix} \frac{1}{2}\frac{1-m}{1-m^*} & \frac{1}{2} \\ \frac{1}{2}\frac{m}{m^*} & \frac{1}{2} \end{bmatrix} \begin{bmatrix} q_{\mathrm{f}} \\ q_{\mathrm{m}} \end{bmatrix}$$

$$= \begin{bmatrix} \frac{1}{2}\frac{1-m}{1-m^*}\, q_{\mathrm{f}} + \frac{1}{2} q_{\mathrm{m}} \\ \frac{1}{2}\frac{m}{1-m^*}\, q_{\mathrm{f}} + \frac{1}{2} q_{\mathrm{m}} \end{bmatrix} \tag{1.18}$$

Note that the phenotypic values (m, m^*) of the genotypes (**a**, **A**) appear only in the first column of the matrix, while nothing but a simple constant ($^1/_2$) appears in the second column. The coefficients in the first column are multiplied by q_f, the frequency of the mutant genotype in *females*. When m is smaller than m^*, the upper left-hand coefficient is greater than one-half and the lower left-hand coefficient is less than one-half. As a consequence, increases in q_f will tend to cause larger increases of q'_f than of q'_m and the relatively female-biasing mutant allele **a** will tend to concentrate itself in females. The opposite happens when m is larger than m^*. Males transmit their genotypes without bias (passively) to daughters and sons, because males do not influence the sexes of their offspring.

The vector $\mathbf{q}$ (which is near zero) will tend to grow when the largest eigenvalue of $\mathbf{M}$ is greater than 1, and it will shrink towards zero when the eigenvalue is less than 1. (The eigenvalues λ are solutions of the characteristic equation $\det(\mathbf{M} - \lambda\mathbf{I}) = 0$, where $\mathbf{I}$ is the identity matrix.) The overall magnitude of $\mathbf{q}$ will not change when the largest eigenvalue is exactly 1, and this will obviously be the case at least whenever $m = m^*$, even if m^* is far from its evolutionary equilibrium. (When $m = m^*$, all four elements of $\mathbf{M}$ are $^1/_2$ exactly.) The eigenvalue is easy to calculate, but it takes a rather messy and unrevealing form involving nonintegral powers of m and m^*. However, since we are really most interested to know what conditions other than $m = m^*$ will give an eigenvalue of 1, we can greatly simplify the problem by *setting* $\lambda = 1$ and expanding the resulting characteristic equation $\det(\mathbf{M} - \mathbf{I}) = 0$ which usually takes an understandable and revealing form. In the present case we get

$$\frac{1}{2} - \frac{1}{4}\frac{1-m}{1-m^*} - \frac{1}{4}\frac{m}{m^*} = 0, \tag{1.19}$$

which easily rearranges to give

$$1 = \frac{1}{2}\left[\frac{1-m}{1-m^*} + \frac{m}{m^*}\right]. \tag{1.20}$$

This should look familiar: it is the Shaw–Mohler equation with the parent's fitness (the left-hand side) set equal to 1. This equality will hold, for arbitrary values of m, if and only if $m^* = {}^1/_2$, consistent with our previous analysis.

It may seem that we have worked through a great deal of 'intricate' genetics and mathematics (as Darwin predicted), only to return to a place very close to the one he reached 130 years ago without the benefit of genes or calculations. In fact, the theory has been greatly augmented and strengthened. We understand that the equilibrium entails equal net investment in the sexes, which will not mean equal numbers if female offspring cost more or less than males. We understand the basic principle much more deeply, having seen it implemented in two different

genetic frameworks, and this gives us both the understanding and the confidence to construct complex and sophisticated tracer-gene models (e.g. Frank 1998). In short, we now have tools with which to attack a nearly unlimited range of problems that could not be solved securely (or in some cases, at all) without formal methods such as these. The next section considers how such methods have been used to extend the central principle to situations not encompassed by the original model.

1.10 Themes and variations

In nature, the biological circumstances surrounding sex allocation are as variable as the ecologies and life histories of real organisms. This variation motivates a seemingly endless diversity of sex ratio models. Does this diversity of models undermine the supposed generality of sex ratio theory? Not really, because the variations play on just a few underlying themes, if often in combinations with each other.

1. *Differential costs of the sexes.* This theme was clearly identified by Fisher. It arises from differential rates of mortality during the period of parental care, from differential resource needs of male and female offspring (reflected for example in different sizes at weaning), and from other ways in which male and female offspring may differentially affect a parent's future reproduction (for example, by compromising the parent's future growth, or by exposing the parent to increased risks of mortality).

2. *Condition-dependent benefits and reproductive values of the sexes.* Both the constraints and the genetic payoffs associated with different progeny sex ratios may change with a variety of environmental factors including seasonality, resource availability, parental size (competitive ability, fecundity) and aspects of local population structure. In some social species, for example, members of the less-dispersing sex may help their parents to defend a territory or to feed subsequent offspring, thereby providing direct reproductive benefits that partially offset the costs of their own production. Alternatively, under certain circumstances the members of one sex may be more likely to reproduce or to benefit from increased parental investment than the other (as first argued by Trivers & Willard 1973). Finally, the offspring of one sex may be more likely to compete with each other for the same matings, in which case they are partially reproductively redundant from the parent's point of view (local mate competition, section 1.10.1). Some of these effects can be subtle, giving rise to selection pressures far weaker than those associated with the population's mean allocation ratio.

3. *Mode of inheritance and locus of control.* Genes with unusual patterns of inheritance, such as those on Y chromosomes and mitochondrial chromosomes, sometimes have different ESS sex ratios than do those on autosomes, giving rise to evolutionary 'conflicts' over the sex ratio (Hamilton 1967). In haplodiploid species where males transmit their genes only to daughters, mates 'disagree' profoundly as to what their progeny sex ratio should be (e.g. Brockmann & Grafen 1989), and in some social Hymenoptera, workers and queens disagree as to what sex ratio their colony should produce (section 1.10.2). In principle, embryonic offspring may disagree with their parents over what sex they themselves should become; for example, if one sex is more costly and therefore produced in smaller numbers, all offspring, given the choice, would prefer to be of that sex.

We now briefly describe three classic extensions of the basic Darwin–Fisher model to show how it has been adapted to such special circumstances.

1.10.1 Local mate competition

The current 'Golden Age' of sex ratio research could be said to have begun with W.D. Hamilton's (1967) paper 'Extraordinary sex ratios'. Hamilton reviews Fisher's argument and then relaxes two of Fisher's implicit assumptions. The paper's first section relaxes the assumption that sex allocation is controlled by autosomal genes (sex-linked meiotic drive, an instance of theme 3, above). The second section relaxes the assumption of random mating in a large population and considers 'Local mate competition' or LMC (theme 2). Hamilton considers species such as fig wasps, where mating

takes place in small aggregations representing the offspring of just a few females, followed by dispersal of the mated females. Under this population structure, brothers compete relatively directly with each other for matings, but sisters do not compete directly for the resources on which their own reproductive success depends. This asymmetry requires a modified *fitness function*.

Under the LMC scenario, a mother's fitness rises linearly with the number of dispersing females she produces, as in the Darwin–Fisher model, but *not* with the number of sons. Because her sons tend to compete with each other, each *additional* son yields a *smaller increase* in the *total number of inseminations* achieved by all her sons together, which is the other source of her fitness. In other words, male production obeys a law of diminishing reproductive returns that does not apply to daughters. Hamilton writes expected-future-fitness expressions for females producing sex ratios x_A and x_B in a population where each mating aggregation contains the offspring of n randomly chosen females, and he finds that the 'unbeatable' (ESS) sex ratio is

$$x^* = \frac{n-1}{2n}. \tag{1.21}$$

In an aggregation containing the offspring of two unrelated mothers, the ESS allocation is extremely unequal: 25% effort to sons, 75% to daughters. However, as n increases beyond just a few mothers contributing offspring to each aggregation, brothers compete less directly with each other (so they are less redundant from their mother's point of view), and the optimal male investment rises toward 50%. In an aggregation founded by just one female, the theoretical optimum sex ratio is 0% males, which is interpreted as '[no] more males than are necessary to ensure the fertilization of all her daughters'. Hamilton reviews sex ratio data from wasps, beetles, mites and thrips that mate in small aggregations and that have haplodiploid genetic systems permitting females to freely control their progeny sex ratios. Broods are strongly female-biased in almost every case but tend to include at least one male.

To test the model's logic, Hamilton constructs an explicit dynamical haplodiploid genetic model and iterates it on the computer for the case $n = 2$. Surprisingly, the unbeatable sex ratio turns out to be approximately 0.21 rather than 0.25 as predicted by the general analytical model. This discrepancy was later confirmed to be a real difference between the ESSs for diploid (biparental) and haplodiploid (arrhenotokous) genetic systems, through the analysis of more sophisticated expected-future-fitness models and explicit genetic models (Hamilton 1979, Taylor & Bulmer 1980, Uyenoyama & Bengtsson 1982, Frank 1985, Herre 1985, Taylor 1988, Stubblefield & Seger 1990). The exact ESS for haplodiplody is

$$m^* = \frac{(n-1)(2n-1)}{n(4n-1)}. \tag{1.22}$$

The downward deviation of m^* (for a given n), relative to Hamilton's original solution, is a consequence of arrhenotoky (males developing from unfertilized eggs), not haplodiploidy *per se*. The same ESS (eq. 1.22) holds for hypothetical haplo-haploid and diplodiploid genetic systems under which, as in ordinary haplodiploidy, females arise from biparentally produced zygotes while males arise from unfertilized eggs (Stubblefield & Seger 1990). Under arrhenotoky, but not under biparental genetic systems (regardless of the ploidys of males and females), inbreeding has unequal effects on genetic transmission through the two sexes, and this leads to the difference between eqs. 1.21 and 1.22.

Under the assumptions of the original Darwin–Fisher model, equal investment in the sexes is a 'weak-form' ESS, not an optimum: if the population as a whole invests equally, then all individual allocations (from 100% sons through to 100% daughters) are equally fit. But under LMC, the ESS is 'strong-form' (Uyenoyama & Bengtsson 1982): even if the population is at equilibrium, individuals suffer reduced fitness if their own progeny sex allocations depart from the ESS, because fitness is determined by the sex ratios within local aggregations.

During the last two decades of the twentieth century, local mate competition became a centrepiece of sex ratio research both through experimental studies of several species of parasitoid wasps and through field studies of entire communities of fig wasps (Chapters 6, 10, 19 and 20). An

important theoretical and empirical issue running through much of this work concerns sex ratio adjustments made by individual mothers in response to information about the numbers and fecundities of other females likely to have contributed offspring to the same mating aggregation. What cues might females use to gather information about other females contributing to their aggregation? What responses should they try to make? How accurate might their responses be? Both theoretically and empirically, the answers depend in interesting ways on a variety of biological details. Given his interest in sex ratio variation among families, Karl Düsing clearly would have enjoyed the current state of LMC research.

1.10.2 Sex ratio conflict in ants

The theory of inclusive fitness (Hamilton 1964) was conceived with social insects in mind, and in his 1972 paper Hamilton considers them at length. Focusing on the Hymenoptera, he points out that haplodiploidy gives rise to a peculiar pattern of relatedness among family members. Owing to their father's haploidy, outbred full sisters are related by $r = 3/4$, but a mother is related to offspring of both sexes by the usual $r = 1/2$. (Coefficients of relatedness are reviewed by Bulmer 1994.) Thus a female would transmit more of her genes to future generations by rearing a sister than by rearing a daughter. Hamilton proposes that as a consequence, a hymenopteran female will 'easily [evolve] an inclination to work in the maternal nest rather than start her own.' However, a female is related to her brothers by only half the usual amount ($r = 1/4$), so she is *not* more related to her mother's offspring *as a whole* than to her own, unless 'the sex ratio or some ability to discriminate allows the worker to work mainly in rearing sisters.' Hamilton suggests that inbreeding might lead to female-biased sex ratios and thereby to eusociality, but a female's average r to her siblings remains the same as her average r to offspring under inbreeding, if mothers control their own sex ratios (Trivers & Hare 1976), so inbreeding does not of itself favour the evolution of eusocial workers. Trivers and Hare (1976) argue that Hamilton's suggestion will work only if daughters actively promote their own reproductive interests at the expense of their mother's.

> The asymmetrical degrees of relatedness in haplodiploid species predispose daughters to the evolution of eusocial behavior, provided that they are able to capitalize on the asymmetries, either by producing more females than the queen would prefer, or by gaining partial or complete control of the genetics of male production.
>
> *(p. 250)*

Trivers and Hare then outline several different steps that workers could take to 'capitalize' on their closer relationships to sisters, sons and nephews than to daughters and brothers. The most important for our purposes (and the most famous) is 'Skewing the colony's investment toward reproductive females and away from males.'

In a colony with just one queen who is singly mated, and who lays all the reproductive eggs, females will be three times as related to their sisters as to their brothers ($3/4 : 1/4$). If the ratio of investment were 1:1 over the population as a whole, then workers would gain three times as much fitness from rearing sisters as from rearing brothers and might therefore benefit from biasing their investment towards sisters, as pointed out by Hamilton. Trivers and Hare argue: (1) that there is little to stop the workers from doing this, counter to their mother's interests, since they do all the work, and (2) that at the resulting evolutionary equilibrium

> we expect three times as much to be invested in females as in males, for at this ratio of investment [3:1] the expected [reproductive success] of a male is three times that of a female, per unit investment, exactly canceling out the workers' greater relatedness to their sisters. Were the mother to control the ratio of investment, it would equilibrate at 1:1, so that in eusocial species in which all reproductives are produced by the queen but reared by their sisters, strong mother-daughter conflict is expected regarding the ratio of investment, and a measurement of the ratio of investment is a measure of the relative power of the two parties.

The paper presents an extensive analysis of data on investment ratios in ants, bees and wasps with different kinds of social structures, and these are

broadly in agreement with the sex ratio arguments. In particular, an average allocation ratio of roughly 3:1 is found for 20 species of monogynous ants, in agreement with the model on the assumptions: (1) that the queens in most of these species are singly mated, (2) that the relative dry weights of males and females indicate their relative costs to the colony, and (3) that workers tend to control the sex ratio. In 1976 there was very little evidence about the mating frequencies of queen ants. Subsequent work has shown that mate numbers can vary within as well as between species, and has exploited this fact to produce some very clean and elegant tests of the model (see below). Subsequent work also has shown that the dry weights of females may tend to overrepresent their costs relative to males, such that the average allocation ratio of Trivers and Hare's 20 monogynous ants may actually be closer to 2:1 than 3:1 (see Boomsma 1989, Bourke & Franks 1995, Crozier & Pamilo 1996), as might be expected if multiple mating is common in some of these species. Trivers and Hare estimate allocation ratios for a number of polygynous ants (those with several to many queens per colony) and 'slave makers' (in which the queen's offspring are reared by workers of another species); as predicted, the apparent allocation ratios of these species are less female biased than those of the monogynous species, on average.

Having introduced the concept of an irreducible conflict over the sex ratio, Trivers and Hare go on to dissect it in some detail. For example, if the queen lays only a fraction (p) of the male eggs, with unmated workers laying the remainder ($1 - p$), then the equilibrium ratio of investment (F/M) for workers is

$$\frac{3(3+p)}{(3-p)^2}. \tag{1.23}$$

This declines from 3:1 when the queen lays all the male eggs ($p = 1$) to 1:1 when workers lay all the male eggs ($p = 0$). From the queen's point of view, the corresponding ESS is

$$\frac{(3+p)}{(3-p)(1+p)}, \tag{1.24}$$

which is 1:1 at both endpoints and slightly lower in the middle. The conflict over the *sex ratio* disappears when workers produce all the males, but it is replaced by a conflict over *male production*, since the queen's inclusive fitness is reduced by worker laying.

Like other models in the paper, this one is derived within an expected-future-fitness framework that takes Fisher's principle as an axiom and that extends it, using Hamilton's inclusive-fitness theory, to account for unequal coefficients of relatedness and indirect parentage. By 1976 this framework seemed so obvious and secure to the authors that they could present expressions such as (1.23) and (1.24) without derivation and with little or no comment. Other theorists, not so readily persuaded by the logic of Trivers and Hare's novel and intricate arguments, soon started testing these arguments by analysing explicit genetic models (e.g. Oster *et al.* 1977, Benford 1978, Charnov 1978, Oster & Wilson 1978, Craig 1980, Taylor 1981, Pamilo 1982, Bulmer 1983). Trivers and Hare's central conclusions were all upheld, although a number of previously unsuspected complications were uncovered by these models; for example, the way in which workers with different sex-allocation phenotypes interact behaviourally to determine the colony's sex ratio can affect the nature of the ESS (Charnov 1978, Craig 1980, Pamilo 1982, Bulmer 1983).

Social insect colonies differ in many relevant ways among and even within species. For example, as mentioned above they may have little or no worker production of males; they may have one or several queens (or no queen); and the queen or queens may mate with one or several males. Models incorporating each of these contingencies have been analysed. Variation in the queen's mate number within species is especially interesting because it affects the workers' but not the queen's equilibrium sex allocation, with potentially dramatic effects on the outcome of the worker–queen conflict (Boomsma & Grafen 1990, 1991).

Consider a species where there is always one queen but she may have mated with one or two males, and suppose that colonies with once- and twice-mated queens are equally frequent and equally productive. Workers in the once-mated colonies would be indifferent to their

colony's allocation ratio if the population-wide ratio were 3:1, as in the simplest model considered above. But workers in the twice-mated colonies would be indifferent if the population ratio were 2:1. (Their average relatedness to sisters is $r = {}^1/_2({}^3/_4 + {}^1/_4) = {}^1/_2$.) Thus both colony types cannot be indifferent simultaneously. If the population-wide allocation ratio is more female-biased than 2:1, then workers in twice-mated colonies would do best to produce males (brothers) exclusively, because males would be the under-represented sex from their point of view. Similarly, if the population-wide ratio is less female-biased than 3:1, then workers in once-mated colonies should produce females (sisters) exclusively.

Suppose that workers can assess the queen's mate number (for example, by perceiving the genetic diversity of their sisters). Then, given our assumptions that the two colony types are about equally frequent and that workers can 'assume' a roughly equal mixture of colony types, the evolutionarily stable outcome should be a polymorphism among colonies, with the singly mated colonies specializing in exclusive female production and the twice-mated colonies producing (at least on average) a 1:2 *male* bias such that the combined population-wide investment ratio is 2:1. These divergent allocations by colonies with different patterns of relatedness asymmetry give rise to a 'split sex ratio' distribution over colonies. Neither type of colony can improve its fitness by making a different sex ratio, and neither actually *produces* its own ESS, although in our example the population average is the ESS for twice-mated colonies.

Colony sex ratios are distributed bimodally in many ant species, and some recent genetic studies of mate numbers in such species are qualitatively consistent with the predictions of this split sex ratio model. Colonies with once-mated queens tend to produce sex ratios that are more strongly female-biased than those of colonies with twice-mated queens (Sundström 1994, Sundström *et al.* 1996). These findings support the idea that sex ratio 'imbalances' (from any actor's point of view) create significant opportunities to increase fitness by making adjustments that exploit the imbalance, and they suggest that worker ants can in fact assess levels of relatedness within their own colonies. Other sources of among-colony variation in relatedness asymmetry, with expected or observed effects on among-colony sex ratio variation, have been considered by Trivers and Hare (1976), Ward (1983), Yanega (1989), Boomsma (1991), Mueller (1991), Chan and Bourke (1994), Evans (1995) and others.

1.10.3 Hermaphrodites

Most plants are simultaneous hermaphrodites (Chapter 16), as are some animals. In such species, sex allocation is a matter of relative effort devoted to male and female functions (for example, to pollen and seed production). Given a linear trade-off between male and female functions, the ESS in an outbreeding population is to invest equally in each kind of function (Maynard Smith 1971). Empirically, relative investment in male and female functions is more difficult to estimate than the numbers of male and female offspring in a dioecious species, and there are additional reasons why hermaphroditism tends to strike us as complicated, even messy. But the hermaphroditic model is actually much easier to solve than the Darwin–Fisher model, because the fitness differences associated with different relative male investments appear sooner (in the first, offspring generation) rather than later (in the second, grandoffspring generation). Perhaps, if human beings were outbreeding simultaneous hermaphrodites, sex ratio evolution would not have baffled Darwin to the extent that it did.

Why are some species hermaphroditic rather than dioecious? Various reasons have been suggested, and most are plausible. For example, some hermaphrodites self-fertilize, and this permits colonization of unoccupied habitats by single immature individuals (e.g. seeds). Self-fertilization also shifts the ESS sex allocation strongly toward investment in female functions, through a principle closely related to that of local mate competition. By economizing on male function, individuals can increase their genetic contributions to future generations; in effect, they escape part of the 'cost of sex' (e.g. Maynard Smith 1978).

However, many plants are self-incompatible (outcrossing) simultaneous hermaphrodites. They pay the full cost of sex, and they need unrelated mates. What are the benefits in this

case? One popular and well supported idea is that owing to their immobility and their reliance on animal vectors for pollination and/or seed dispersal, many plants may experience diminishing returns on investment in one or both sex functions. In addition, the temporal separation of male and female functions may reduce the degree to which those functions draw from the same pool of resources. Under such conditions, an individual may be able to achieve greater net reproduction by being partly male and partly female than it could by devoting all of its resources to just one sex function. In other words, the *constraint* on possible combinations of male and female reproduction may be nonlinear in a way that makes hermaphroditism more efficient than dioecy.

This idea is often modelled by representing an individual's realized or effective male and female reproductive *outputs* as arbitrary powers of its internal resource *allocations:* $m = x^a$ and $f = (1 - x)^b$. The exponent a controls the shape of the function that scales reproductive returns on investment in male function, and the exponent b scales returns on female investment. If a or b is less than 1, then the corresponding sex function shows diminishing returns to scale, but if a or b is greater than 1, then the corresponding function shows increasing returns to scale. For example, suppose $a = 0.25$ because pollinators are easily saturated, but $b = 1$ because fruits and their seeds will be eaten by birds in direct proportion to their abundance and then dispersed widely. The *fitness set* representing possible combinations of realized male and female outputs (m and f, corresponding to values of x between 0 and 1) bends outward with respect to the origin, as shown in Figure 1.4. This graph makes it easy to see that hermaphrodites (individuals with some degree of mixed sex expression) will tend to have larger total reproductive outputs than pure males or pure females, because $m + f$ is clearly greater for intermediate points on the fitness set than it is for points at the ends where m or f is zero. But where exactly is the ESS? The unbeatable allocation is not obvious, because the fitness set is not symmetrical.

We can find the ESS by writing a Shaw–Mohler equation for the fitness of a focal individual that allocates x to male function in a population

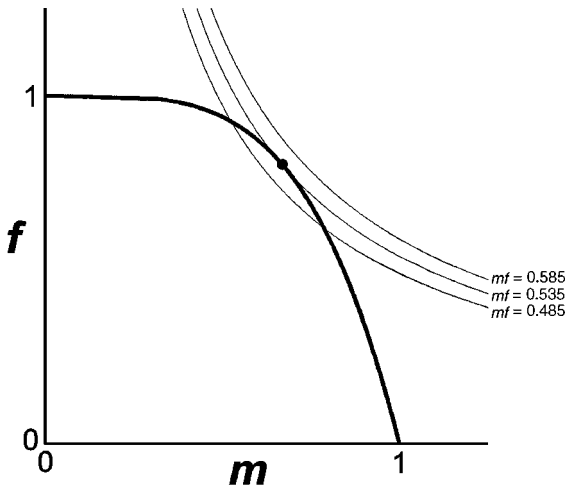

Fig 1.4 Fitness set for a simple model of hermaphroditic sex allocation. The heavy curve shows possible combinations of male (m) and female (f) reproductive outputs for a plant whose male gain exponent ($a = 0.25$) provides diminishing returns on investment in male function, while its female gain exponent ($b = 1$) provides linear returns. The equilibrium resource allocation ($X^* = 0.2$) is highly female biased, as is the realized output of male and female reproductive functions ($m^* = 0.67$, $f^* = 0.80$), indicated by the filled circle. The light curves are hyperbolas representing constant values of the product mf. They can be viewed as a contour map, and they demonstrate that MacArthur's (1965) principle applies to this model; the evolutionarily stable strategy (ESS) coincides with the highest value of mf attainable on the fitness set.

where the average allocation is X

$$W = \frac{1}{2}\left[\frac{x^a}{X^a} + \frac{(1-x)^b}{(1-X)^b}\right]. \tag{1.25}$$

When the population-wide average allocation X is at the evolutionary equilibrium, our focal individual should be unable to increase its fitness W by choosing an allocation x that differs from X. In other words, $W(x)$ should be maximized when $x = X$. To find this unbeatable allocation we differentiate the Shaw–Mohler equation with respect to x, set the derivative equal to zero, set $x = X$, and then solve for X. Doing so gives the solution

$$X^* = \frac{a}{a+b}. \tag{1.26}$$

On substituting the exponents discussed above ($a = 0.25, b = 1$) into this general solution we get $X^* = 0.20$, which is highly female-biased with respect to resources invested and substantially biased even with respect to the resulting

reproductive outputs ($m^* = 0.67$, $f^* = 0.80$) (Figure 1.4). The hermaphrodite's fitness ($W = 1$) is substantially greater than that achieved by a male ($x = 1, W = 0.75$) or a female ($x = 0, W = 0.63$), so hermaphroditism is clearly stable against invasion by the pure sexes (Charnov *et al.* 1976).

If the scaling exponents a and b both exceed 1, then the fitness set bends inward towards the origin and dioecy (with equal numbers of males and females) is evolutionarily stable. If one exponent is less than 1 and the other is greater than 1, then either androdioecy (males and hermaphrodites) or gynodioecy (females and hermaphrodites) may be stable, depending on the exact values of a and b (Charnov *et al.* 1976). This simple model has been extended in many ways to reflect potentially important aspects of plant physiology, development and ecology. For example, models have been studied that incorporate vegetative growth between bouts of reproduction, with trade-offs between investment in growth (for future reproduction) and investment in reproduction (for current fitness); in some such models the regions of parameter space supporting gynodioecy and dioecy expand while those supporting androdioecy shrink, in ways that may help to explain why androdioecy is very rare in nature (Seger & Eckhart 1996, Eckhart & Seger 1999). Plant sex-allocation strategies are further complicated by several other features of plant biologies including strong spatial population structures, mating systems that involve mixed selfing and outcrossing, and cytoplasmically determined 'male sterility' (femaleness), which gives rise to an evolutionarily unstable but widespread form of gynodioecy.

In 1941, the botanist D. Lewis published an explicit population-genetic model for the relative frequencies and fecundities of females and hermaphrodites in populations where male-sterile individuals (females) are determined by genotypes at a nuclear locus. He discovered that females cannot invade an outbreeding hermaphroditic population unless they set at least twice as many seeds as a typical hermaphrodite, that the equilibrium frequency of females will approach one-half as hermaphrodites increase their male function and decrease their female function (thereby becoming male-like) and that none of this is affected by the dominance or recessiveness of the alleles that convert hermaphrodites to females. Lewis does not cite Darwin, Düsing, Fisher or any other previous sex ratio theorist, and he does not seem to realize that his model illuminates general issues in sex allocation and represents a major methodological advance. The paper seems not to have been noticed by subsequent sex ratio theorists until much later, after explicit genetic models had been reinvented.

1.11 Conclusion: diversity in unity

Sex ratios evolve according to simple, aesthetically beautiful principles, but they often affect and respond to many particular and even idiosyncratic aspects of a species' biology. Sex ratio theory therefore establishes concrete links between some of the most specific and some of the most general phenomena in biology, and it does so in a rich and productive way. The theory also accommodates a wide variety of styles and techniques of analysis that continue to grow in sophistication and rigour. Yet despite the field's ever-growing diversity in these respects, its theoretical structure is becoming simpler and more transparent. The field as a whole is much larger than it once was because more is known, and in more detail. But the central ideas and principles seem more coherent, more clearly articulated, and therefore easier to master than they were a few decades ago. As we stressed at the outset, there is still a great deal to be done, both empirically and theoretically, and no one can predict what will turn up next. Sex touches almost everything, so the study of its allocation will lead us to many new problems, and a large fraction of these seem certain to be interdisciplinary.

For example, as genetics becomes increasingly genomic in scale, we are encouraged to think in increasingly concrete terms about the possibilities for intragenomic conflict over sex ratios. When Hamilton introduced this subject in the first half of his 1967 paper, it was little more than an abstract possibility supported by a few observations of sex-chromosome meiotic drive. Now, only 35 years later, we know the exact chromosomal locations of all the genes in

several species' genomes, and soon we will know when and where these genes are expressed and at least something about the physiological properties of their products. There are many situations in which sex-linked and autosomal genes might 'disagree' about their carrier's parental investments or other sex-biased interactions with kin. How are these conflicts 'settled'? Our growing ability to observe both the expression and the evolution of arbitrarily large sets of genes should bring new life to both the theoretical and empirical aspects of this very interesting problem.

On the theoretical side, expected-future-fitness and population-genetic models will again play complementary roles. Obviously, with specific genes in mind, it will be natural and necessary to construct explicit multi-locus dynamical models that show how the interactions of alleles with various different phenotypic effects might lead to various alternative outcomes. Perhaps less obviously, it will also be necessary to ask how neutral alleles at typical loci, elsewhere in the genome, might be affected by the possible outcomes of the conflict. If different outcomes are better and worse for large numbers of genes throughout the genome, then such genes might be recruited into the conflict as modifiers of small effect. Because fitness-based models describe how particular sex-allocation phenotypes affect the future frequencies of neutral genes associated with those phenotypes, such models will be used to study how the genetic background at large might be expected to respond to particular genetic conflicts.

Acknowledgments

We thank Steve Frank, Ian Hardy, Mike Mesterton-Gibbons, Steve Orzack and Franjo Weissing for encouragement and for many helpful criticisms and suggestions.

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