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J. J. BULL and ERIC. L. CHARNOV

I would regard the problem of sex ratio as solved.
G. C. Williams (1966)

1. INTRODUCTION

It has been known for centuries that species tend to produce sons and daughters in equal numbers (Darwin 1871, pp. 300–20; Parkes 1926), but the first plausible evolutionary explanation of this fact was provided by Fisher in 1930. Fisher's explanation for 1:1 population sex ratios was based on the seemingly trivial fact that every zygote has one mother and one father; the reproductive value of all males must therefore equal the reproductive value of all females. If sons and daughters are not equally numerous in the population, then the per capita reproductive success is higher for individuals of the rare sex than for the common sex, and genes overproducing the rare sex may increase in frequency until the sex ratio equalizes.

Various animals are known to have sex ratios near 1:1, but the empirical success of Fisher's theory remains in doubt for two reasons. First, many observations of 1:1 sex ratios are based on sex chromosome systems (XX/XY), and serious doubts have arisen about the degree to which sex ratios can evolve in these systems. Second, there has not been a widespread attempt to evaluate Fisher's theory in species lacking XX/XY systems. This lack of attention to Fisher's sex ratio theory is surprising because sex ratio theory has grown immensely since Fisher and is one of the most successful quantitative branches in the study of evolution; simple models have successfully predicted sex ratio variations ranging from nearly all males in some cases to nearly all females in others. However, these new developments in sex ratio theory have either deliberately dealt with special cases of non-Fisherian processes or dealt with sex ratio phenomena that do not bear directly on Fisher's prediction (sex ratio variance), and, while their successes have been spectacular, the question remains as to whether sex ratios in the bulk of sexually-reproducing species familiar to most biologists are explained by Fisher's model.

In this chapter we return to the basic phenomenon addressed by Fisher and consider how often population sex ratios are consistent with his theory.

However, it is difficult to address Fisher's theory in isolation from its bearing on the broader context of modern sex ratio theory. The chapter is therefore organized into two main parts. Section 2 reviews the main body of sex ratio models and their relationships with each other. Section 2.1 introduces the discipline of sex allocation theory, which includes sex ratio evolution and similar problems; section 2.2 reviews the assumptions underlying Fisher's theory and explains how these bear on the predicted sex ratio equilibria, and section 2.3 discusses the central place of Fisher's theory in the organization of sex ratio theory. Section 3 then focuses on the empirical side of sex ratio theory. Section 3.1 explains why the 1:1 sex ratios observed in many sex chromosome systems are not necessarily consistent with Fisher's theory, and sections 3.2 and 3.3 review the evidence on sex ratios from species with a variety of other sex-determining mechanisms. Throughout this chapter, sex ratio will usually be represented as the proportion male or as the number-of-males:number-of-females and will designate the 'primary' sex ratio (at conception) unless indicated otherwise.

2. MODELS AND THE STRUCTURE OF SEX RATIO THEORY

2.1 Sex allocation theory

The study of sex ratios and related phenomena presently occupies a major focus in evolutionary biology and is encompassed in the discipline known as sex allocation theory, which includes but is far broader than Fisher's sex ratio theory (Charnov 1982; Leigh *et al.* 1985). Sex allocation theory addresses topics ranging from sex ratio selection in dioecious species to the seemingly distant problems of the optimal time to change sex in a sequential hermaphrodite, the optimal allocation of resources to sperm versus eggs in a simultaneous hermaphrodite, to the very evolution of dioecy versus hermaphroditism. The unification of these problems into one framework is based on similarities in the underlying mathematics of models describing evolution in these systems, which in turn reflects common evolutionary principles underlying the problems. The sex allocation framework thus identifies parallels between different problems, indicates how tests of the models may be applied, and guides the search for new explanations when old ones have failed.

The objective of our study is an unusual one. We will attempt to isolate Fisher's model from sex allocation theory and evaluate its sufficiency as an explanation for population sex ratios. Although the goal in science is often to unify theories rather than divide them, it is of some interest to know how much can be expected of an individual theory as it stands by itself. Furthermore, the sex ratio literature is not clear on the extent to which observations agree with Fisher's theory, nor even on what is properly

regarded as Fisher's theory. We begin with an explanation of Fisher's sex ratio theory and its role in sex allocation theory.

2.2 Fisherian sex ratio evolution

This section explains our understanding of Fisher's sex ratio theory, first at an informal level (section 2.2.1) and then at a formal level. A scientific theory generally consists of a set of assumptions (a model) and a prediction that follows as a consequence of those assumptions. Fisher was explicit about the prediction of his model, but many of the assumptions were not explicit. Some reconstruction of his model is therefore necessary, and there is consequently no consensus on the complete set of models that should be attributed to Fisher (section 2.2.2). Violating any of the assumptions in Fisher's model may lead to a non-Fisherian sex ratio equilibrium, and the broader scope of sex allocation theory may thereby be developed as an outgrowth of systematic violations of the assumptions underlying Fisher's theory (section 2.2.3).

2.2.1 *Fisher's description and its implications*

Fisher's explanation of sex ratio evolution was characteristically terse (1930, p. 142), especially considering that his argument was unprecedented.

> In organisms of all kinds the young are launched upon their careers endowed with a certain amount of biological capital derived from their parents . . . If we consider the aggregate of an entire generation of such offspring it is clear that the total reproductive value of the males in this group is exactly equal to the total value of all the females, because each sex must supply half the ancestry of all future generations of the species. From this it follows that the sex ratio will so adjust itself, under the influence of Natural Selection, that the total parental expenditure incurred in respect of children of each sex, shall be equal; for if this were not so and the total expenditure incurred in producing males, for instance, were less than the total expenditure incurred in producing females, then since the total reproductive value of the males is equal to that of the females, it would follow that 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 than would parents having a congenital bias towards the production of females.

It is an understatement to suggest that Fisher's argument is cryptic; various models have since been published that merely attempted to elucidate Fisher's argument, attesting to the difficulty of the problem (e.g. Shaw and Mohler 1953; Leigh 1970; Kolman 1960; Bodmer and Edwards 1960). In the remainder of this section, therefore, we offer an intuitive explanation of Fisherian sex ratio evolution, reserving a formal reconstruction for section 2.2.2.

Natural selection operates in Fisher's sex ratio model as in any model of evolution by natural selection: if the average fitness of a male zygote is not

equal to the average fitness of a female zygote, natural selection favours an increase in the frequency of the sex whose per capita fitness is higher. Sex ratio models are special, however, because fitness is frequency-dependent; an individual's fitness is not simply determined by its phenotype (gender) and the environment, but rather its fitness depends on its gender in relation to the frequency of males and females in the population. The frequency dependence in sex ratio selection is fundamental yet renders the problem unintuitive. To illustrate, suppose that K daughters are conceived for every son in the population. Under random mating, it follows that, over its lifetime, the average fitness of a female relative to that of a male will be $1/K$. The fitness of a male zygote is thus equal to that of a female zygote only if $K=1$, i.e. only if the primary sex ratio is $\frac{1}{2}$, and this equation holds *regardless of the degree of polygamy, monogamy, or subsequent differential mortality*. If brood size is independent of sex ratio, then parents overproducing sons or daughters are afforded no difference in fitness at this equilibrium, because the average fitness of each son equals that of each daughter, and fecundity does not change with sex ratio.

The foregoing is a special case of Fisher's argument because we assumed that sex ratio and family size were independent. More generally, we need to allow sex ratio to influence family size, as when a son requires a different amount of parental expenditure than a daughter. Suppose that sons are twice as costly as daughters; hence a brood may consist of: ($2N$ daughters, 0 sons), of (0 daughters, N sons), of (N daughters, $N/2$ sons), and so forth. If the primary sex ratio is $\frac{1}{2}$, a son has the same expected fitness as a daughter, yet a parent can produce two daughters for every son. Parents producing $2N$ daughters would thus have 1.5 times the fitness as parents producing a 1:1 sex ratio (with fitness measured as the number of grandchildren); parents overproducing daughters would increase in frequency, and the sex ratio would shift from 1:1 toward an overproduction of daughters. Equilibrium would be reached at a primary sex ratio of 1 son:2 daughters, with each male zygote having twice the average fitness as each daughter. However, since two daughters must be given up to produce a son, there is no longer selection among parents producing different sex ratios—a brood of N sons would yield the same fitness as a brood of $2N$ daughters. Although the primary sex ratio at this equilibrium is not $\frac{1}{2}$, it does satisfy Fisher's prediction of 'equal expenditure': one-third of the progeny are sons, each at a cost of 2 units, and two-thirds are daughters, each at a cost of 1 unit, so the total expenditure on sons equals that on daughters. These examples may be generalized to show that, if a son costs s units and a daughter costs d units, the sex ratio satisfying Fisher's equilibrium is $d/(s+d)$ and always satisfies the principle of equal investment. As Fisher pointed out, a differential cost may arise if one sex experiences higher mortality than the other during the period of parental investment.

The selective neutrality of sex ratio at equilibrium. Fisherian sex ratio selection is stabilizing, frequency-dependent selection toward the equilibrium of equal investment. The magnitude of selection decreases as the population sex ratio approaches this equilibrium, and it vanishes when the population sex ratio reaches equilibrium—all family sex ratios are equally fit at the equilibrium. Factors that lie outside of Fisher's model and which have even a small impact on sex ratio selection can therefore shift the equilibrium substantially away from equal investment. Figure 1 illustrates the magnitude of selection on a genetic modifier of sex ratio as a function of the population sex ratio R. The curve shows the magnitude of Fisherian sex ratio selection (λ) on a gene that produces 100 per cent of the rare sex when the selected equilibrium is $\frac{1}{2}$. As indicated by the fact that λ is near unity for R in a broad range about $\frac{1}{2}$, selection is relatively weak near the value $R=\frac{1}{2}$.

Evolutionary implications. Fisher's model predicts that, genetic variation permitting, the primary sex ratio will be adjusted toward a unique equilibrium determined by the relative cost of producing a son versus a daughter whenever the population is not already at this equilibrium. The implications of his prediction depend in a curious way on the sex-determining mechanism, because under some mechanisms, the sex ratio automatically adjusts to the optimum *without any evolution*; in other systems the sex ratio can adjust only through gene frequency evolution.

Consider first a mechanism known as maternal monogeny, in which half the females conceived are destined to produce only sons, the other half are destined to produce only daughters (Bull 1983, Chapter 15). Differential cost of sons versus daughters translates into corresponding differences in lifetime

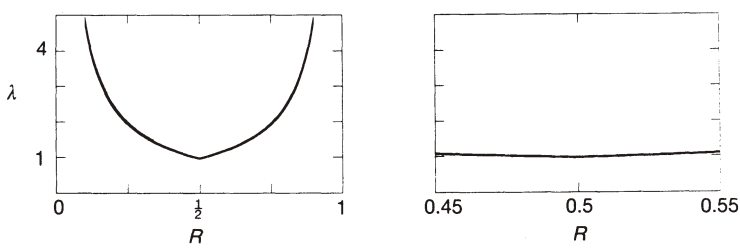

Fig. 1. The selective neutrality of sex ratio selection at equilibrium. The horizontal axis represents the average population sex ratio (R), and the vertical axis (λ) is the per-generation rate of change of the frequency of a rare gene producing 100 per cent of the rare sex. The value $\lambda=1$ (corresponding to $R=1/2$) means that the rare gene has no advantage or disadvantage, but all other values of λ exceed unity and indicate that genes overproducing the rare sex are selected. Importantly, the rare sex is selected more strongly as the sex ratio deviates further from 1/2. Right and left sides of the figure merely indicate different scales on the horizontal axis. (Calculated for the case in which a son and a daughter are equally costly.)

fecundities of the two types of mothers, and the primary sex ratio automatically produces the Fisherian equilibrium without any change in the genetics of sex determination or sex ratio; the total expenditure on offspring of each sex must be equal regardless of differential cost, since half the mothers produce only sons and the other half produces only daughters.

At the other extreme, consider heterogamety (XX/XY), and assume that, at each conception, the probability of producing a son is $\frac{1}{2}$ for all families. In this case, the primary sex ratio remains fixed at $\frac{1}{2}$ despite any magnitude of differential cost, and attainment of the Fisherian equilibrium would require selection of genetic variation in sex ratio. (The fact that the primary sex ratio does not immediately adjust to reflect the differential cost may not be obvious; families that, by chance, happen to overproduce the cheaper sex will be larger than families overproducing the expensive sex, and it may seem that the primary sex ratio would automatically deviate toward the cheaper sex. However, at each point in time, the probability of conceiving a son is $\frac{1}{2}$ for all families, so the primary sex ratio over the entire population must remain at $\frac{1}{2}$.) In comparing the two extremes of monogeny and heterogamety, therefore, we may anticipate that the amount of sex ratio evolution that is favoured in response to a differential cost depends on the prevailing sex ratio and on the magnitudes of within- and between-family variance in sex ratio.

2.2.2 A formal model consistent with Fisher's description

The need for reconstruction. Fisher's sex ratio theory was proposed at a time when the major phenomenon to be explained was the prevalence of 1:1 sex ratios. Fisher's theory is perceived at being so successful in this respect that virtually all new developments in sex ratio theory have focused on the evolution of sex ratios deviating from 1:1 (equal investment). This expansion of sex ratio theory has been accompanied by an increased awareness that various factors can influence sex ratio evolution, and there has been an inevitable emphasis on specific, algebraic models now used to explain sex ratio evolution. By present standards, therefore, Fisher's theory is based on a largely implicit model, and if one is to render it fully comparable to other models in sex allocation theory, it becomes necessary to reconstruct Fisher's model on an explicit level. Attempts at reconstruction have thus been based on Fisher's few explicit assumptions and on the predicted equilibrium of equal investment.

We think that the following assumptions would be regarded by most sex ratio theorists as being fundamental to Fisherian sex ratio evolution (authors first clarifying the importance of these assumptions are listed in parentheses):

1. Separate sexes (Fisher).

2. Biparentalism—every zygote has one mother and one father (Fisher).

3. Mendelian segregation of alleles influencing sex ratio (Lewis 1941; Howard 1942; Shaw 1958; Hamilton 1967).
4. Parental control of sex ratio; an individual's genotype influences the sex ratio of its progeny (Fisher; Trivers 1974).
5. Parents have a fixed amount of resources for producing offspring, and offspring costs are additive (Fisher; MacArthur 1965).
6. Random mating in an infinite population with no substructure (Hamilton 1967).
7. Sex ratio differences between families does not correlate with fitness differences within a sex (Bull 1981).

Considerable refinement of these assumptions is required to provide a specific model for mathematical analysis, but these seven assumptions are presently regarded as the ones most critical to Fisher's result of equal investment; violating any one of them leads to a profound and predictable alteration of sex ratio evolution. We postpone an elaboration of these points until section 2.2.3; the remainder of this section discusses the additional assumptions pertinent to Fisher's model.

Genetics. The above list of seven assumptions specifies inheritance only in bare outline, as provided by assumptions 2–4. Specific models of sex ratio evolution have typically assumed diploid males and females, with a single, diallelic locus controlling sex ratio. Other modes of inheritance have also been studied (polygenic sex ratio control, sexual haploids, and so forth), and these studies have collectively suggested that Fisher's equilibrium is not sensitive to these genetic details—the general impression has been that, given assumptions 1–7, Fisher's equilibrium applies so long as there is an adequate supply of 'additive' genetic variance in sex ratio and that sex-ratio genes do not pleiotropically affect other fitness traits. The Fisher equilibrium even holds for maternal control of sex ratio under haplo-diploidy. However, Karlin and Lessard (1986) recently challenged the robustness of Fisher's result under multiple-locus inheritance, suggesting that the nature of genetic interactions may profoundly alter the sex ratio equilibrium; this problem warrants further work because it is contrary to all previous findings and assertions.

The fact that many specific models are consistent with Fisher's description of sex ratio evolution or with the equilibrium of equal investment raises the philosophical question of which models should be attributed to Fisher. The tendency has been to attribute Fisher with all models yielding a unique sex ratio equilibrium of equal investment, even including haplo-diploidy (e.g. Trivers and Hare 1976). There does not appear to be a consensus on this matter, however.

Interaction of differential cost, parental control, and mating system. In the absence of a differential cost for a son versus a daughter, the Fisherian sex ratio equilibrium is $\frac{1}{2}$ regardless of whether sex ratio control resides in the parent or offspring. (Sex ratio 'control' refers to the individual in which genetic variation is expressed.) As Trivers (1974) noted, the Fisherian equilibrium applies under differential cost only if sex ratio control resides with the parent. However, parental control of sex ratio may not be sufficient to guarantee an equilibrium of equal investment. In a polygamous mating system, sex ratio control must reside with the same parent that determines family size to ensure the Fisher equilibrium. (This restriction does not apply under strict monogamy (Charnov 1982, p. 4).)

It should be evident from these few paragraphs, that Fisher's equilibrium of equal investment is based on an intricate and complicated set of assumptions, and considerable effort is required to tease apart the effects of these different assumptions. In section 2.2.3, we address a few of the most fundamental assumptions in Fisher's model and illustrate how they bear on the equilibrium of equal investment.

2.2.3 Implications of Fisher's main assumptions

Violating any of the above assumptions may cause the sex ratio equilibrium to deviate from equal investment, although some violations have a greater impact on the equilibrium than others (Fig. 2). Sex allocation theory has in fact grown by such a process—the progressive realization that many assumptions are specifically important to the evolution of equal investment. Thus, in showing the relevance of each of Fisher's assumptions to this equilibrium, one simultaneously constructs a theoretical framework for the explanation of non-Fisherian sex ratios (i.e. sex allocation theory).

At the outset, it should be reiterated that Fisherian sex ratio selection is frequency-dependent selection. The frequency dependence derives from the assumptions of Mendelian inheritance and biparentalism; hence the frequency-dependent component of selection is present in many non-Fisherian models as well. The frequency-dependent property of Fisherian sex ratio selection will thus serve as a focal point throughout this section.

Non-Mendelian inheritance and uniparentalism: A critical pair of assumptions in Fisher's argument are biparentalism (assumption 2) and Mendelian segregation of sex ratio genes (assumption 3). Although these properties are readily separable in some cases, the most common and important exceptions to one principle are also exceptions to the other. For example, one of the more renowned examples of non-Mendelian inheritance of sex ratio genes is provided by 'cytoplasmic' factors—transmitted to all offspring but transmitted to grandchildren only through daughters. (A cytoplasmic factor thus exhibits both non-Mendelian segregation and uniparental inheritance.) The extent to which a particular cytoplasmic factor is represented in future

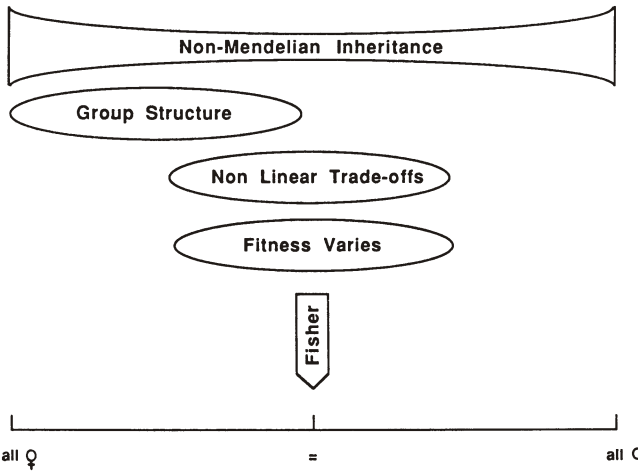

Fig. 2. Violating each of the critical assumptions in Fisher's model leads to deviations from equal investment at equilibrium, but the violation of some assumptions has more drastic effects on the sex ratio than the violation of others. For example, cytoplasmic inheritance of sex ratio variation is selected toward the production of all females, whereas extreme forms of non-linear trade-offs are required to select sex ratio extremes as great as, say, 0.2 or 0.8. The range of investment ratios that might be plausibly observed under each type of violation is represented by the portion of the horizontal axis overlapped.

generations thus increases directly with the proportion of females in the brood, and any cytoplasmic factor that somehow causes a mother to overproduce daughters is favoured. In a random-mating population, the equilibrium sex ratio under cytoplasmic inheritance is 100 per cent female—causing population extinction (Howard 1942; Shaw 1958; Hamilton 1967). Cytoplasmic factors lie outside Fisher's model because males do not 'supply half the ancestry of all future generations', and the frequency-dependence of sex ratio selection is thereby abolished.

Violations of Fisherian sex ratio evolution due to non-Mendelian inheritance and uniparentalism are important in sex allocation theory because the documented examples are spectacular. They are known for three specific cases of sex ratio modifiers: (1) cytoplasmic factors; (2) segregation distortion of the X or Y chromosome in the XY sex; (3) uniparental systems of parthenogenesis and gynogenesis (where females are produced uniparentally). Under random mating, the equilibrium sex ratio is 100 per cent female in all of these cases except for segregation distortion of the Y under male heterogamety or of the Z under female heterogamety, in which case the equilibrium is 100 per cent male. It is the extreme nature of these violations of

Mendelian inheritance and biparentalism that leads to the severe deviations from an equilibrium of equal investment.

The impact of violating Mendelian inheritance on Fisher's result can be demonstrated in a powerful and basic way without resorting to the extremes of sex linkage and uniparentalism. Consider the hypothetical case that, on average, each zygote receives a fraction k of its genome from its father and $(1-k)$ of its genome from its mother. The usual case is $k=\frac{1}{2}$, but we can imagine any value of k, as in the case that each zygote eliminates one of its two parental genomes shortly after fertilization (the paternal genome retained with probability k). Following similar lines as in Charnov (1982, pp. 230–1), one can establish that the sex ratio favoured by natural selection is k. Therefore, if we replace the assumption of biparental, Mendelian inheritance with the assumption of an arbitrary genetic contribution from one father and one mother, Fisher's result would be: natural selection favours an equilibrium investment ratio in sons which equals the proportion of the genome contributed by the father. The linear dependence of the sex ratio equilibrium on k illustrates that it is this component of inheritance underlying the frequency dependence of Fisherian sex ratio selection.

A population experiencing sex ratio distortion due to cytoplasmic sex ratio factors (or due to segregation distortion of the X or Y) selects Mendelian factors that suppress this non-Mendelian variation and restore the sex ratio to equal investment (Hamilton 1967; Charnov 1982). Deviations from equal investment that result from cytoplasmic factors may thus be unstable and, over long periods of time, may oscillate toward and away from the Fisherian equilibrium. (This conflict does not arise when the entire genome is inherited uniparentally, however.) In such cases, it is difficult to make any *a priori* predictions about sex ratio evolution, except that female excesses should be associated with at least partial cytoplasmic inheritance of sex (Uyenoyama and Felman, 1978).

Group-structured matings and inbreeding Hamilton (1967) first pointed out that Fisher's model was based on random mating (assumption 6), and that certain violations of this assumption have drastic effects on sex ratio evolution. Hamilton's examples were drawn from parasitoid wasps, fig wasps, mites, and a few other kinds of animals with life histories in which mating is confined to small groups consisting of one or a few families. The logic of Hamilton's argument can be seen by considering the extreme case in which each mother typically produces sons and daughters who mate entirely among themselves—all of a mother's daughters are inseminated by her sons, so her sons have no competition from other males. If one son can inseminate all of her daughters, she maximizes the number of her grandchildren by producing a brood consisting of one son and the remainder as daughters. An excess of daughters is also favoured if her progeny mingle with broods from one or a few other mothers, but the selected sex ratio progressively shifts toward $\frac{1}{2}$ with the number of competing broods.

The specific violation of random mating exhibited in this case has been designated 'local mate competition'. (Not all forms of non-random mating favour non-Fisherian sex ratios.) In comparing sex ratio evolution between local mate competition and random mating, the difference is seen to stem from the fact that the number of grandchildren produced by the sons in a family increases proportionately with the number of sons under random mating but not under local mate competition.

One interpretation of Hamilton's result is that sex ratio selection is operating at two levels, within and between groups, where a 'group' is the set of families to which mating is confined (Hamilton 1979; Bulmer and Taylor 1980*a*; Wilson and Colwell 1981). The within-group component of selection is Fisherian, and indeed, this component favours a sex ratio of $\frac{1}{2}$ (Wilson and Colwell 1981). The between-group component favours an excess of females, because a group's fitness (number of foundresses produced) increases with the fraction of daughters. The equilibrium sex ratio is thus balanced between the Fisherian force toward $\frac{1}{2}$ and the between-group force toward all females. In this manner, it is seen that the Fisherian component of frequency dependence is retained under group-structured mating, but that it is partly overridden by the force generated by the group structure.

In nature, group-structured mating systems often involve inbreeding, and in addition to the effect described above, inbreeding *per se* can be shown to alter sex ratio selection *when one sex is haploid* or at least transmits uniparentally (Herre 1985). Under male haploidy, inbreeding by itself favours a female excess, up to 2/3 daughters (Hamilton 1972; Herre 1985). In practice, it is often difficult to separate the effect of inbreeding from the effect of group-structured matings, because the biological circumstances that lead to one effect often lead to the other. None the less, Herre's study of fig wasps succeeded in showing the identities of both effects.

Non-linear trade-offs. One of Fisher's assumptions is that the set of possible family compositions is linear. Thus, if each son 'costs' twice what each daughter costs, then a family may consist of $2N$ daughters and 0 sons, $2N-2$ daughters and 1 son, and so forth to the extreme of 0 daughters and N sons. In conjunction with random mating, this assumption means that the number of grandchildren produced by the sons in a family is proportional to the investment in sons, and similarly for daughters. The frequency dependence of sex ratio selection is present regardless of the form of the trade-off, but non-linear trade-offs generally lead to the evolution of sex ratios that violate equal investment (MacArthur 1965; Charnov 1982; Frank 1987).

Non-linear trade-offs can enter sex ratio models in several ways: the set of possible family compositions may not be linear, or the fitness of a son (daughter) may depend on the family sex ratio (Clark 1978; Bulmer and Taylor 1980*b*; Toro 1982; Emlen *et al.* 1986; Seger and Charnov 1988). As an illustration of the latter type of non-linearity, the model of group-structured

mating considered above can be treated as a form of non-linear trade-offs: the number of grandchildren produced from a mother's brood increases linearly with the number of daughters in that brood but increases less than linearly with the number of sons; selection favours a female excess in this case (Charnov, 1982). Non-linearities of either sort can lead to the evolution of female excesses or male excesses; the equilibrium sex ratio favours the sex that would show the greater fitness gain per investment at a sex ratio of $\frac{1}{2}$.

A correlation between sex ratio and fitness. The primary sex ratio no longer evolves exactly according to Fisherian principles whenever the sex ratio covaries with some *environmental* effect on fitness (separate from any differential mortality based on gender; Bull 1981). The basis for this result can be seen in the hypothetical case that some offspring develop in patches of poor quality, to the point that they are almost completely sterile, while the others develop in patches of high quality. The sex ratio in the high-quality patch is selected to be near $\frac{1}{2}$, regardless of the sex ratio in the poor-quality patch. However, the population primary sex ratio is the average of the sex ratio in both patches and may deviate substantially from $\frac{1}{2}$ at equilibrium. There is an infinity of equilibria in this case, but only one satisfies equal investment. The frequency-dependent component of selection is retained.

A covariance between sex ratio and fitness is expected to evolve under such mechanisms as environmental sex determination and haplo-diploidy whenever some environmental parameter has a different effect on the relative fitness of males than of females (Trivers and Willard 1973; Charnov 1979; Bull 1981). The deviation from $\frac{1}{2}$ that is expected in the population depends on the male and female fitnesses associated with different patches and with the abundances of these patches. Except under extreme environmental effects on fitness, the population-wide sex ratio equilibrium is not expected to deviate greatly from $\frac{1}{2}$. Furthermore, the sex ratio should favour the sex that is overproduced in patches of poorer quality (Charnov 1982).

2.3 A central role for Fisher's model in the organization of sex allocation theory

Fisher is often heralded as the pioneer of modern sex ratio theory because his model was the first to point us in the direction we now stand. Yet it is important to separate his precedence from the role that his model currently holds in the discipline of sex ratio theory. The impact of Fisher's model can be evaluated from two perspectives—theoretical and empirical. In the first case, we observe that the Fisher model is central to the other major models in current sex ratio theory. The set of assumptions that underlies Fisher is one step removed from those of the other models, whereas no other model is so central (Fig. 3). Most importantly, Fisher's model incorporated what is perhaps the most important pair of principles in all of sex ratio evolution:

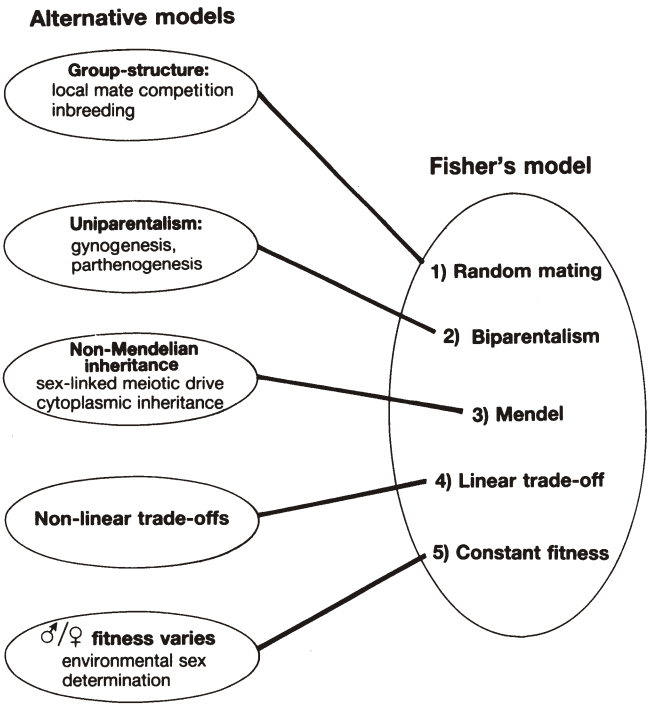

Fig. 3. The central role of Fisher's model in sex ratio theory is easily visualized because the set of assumptions in his model are one step removed from the assumptions that underly the major alternative models in sex allocation theory. These alternative models are discussed in section (2.2.3).

biparentalism and Mendelian inheritance. The fact that every zygote has one mother and one father with equal genetic contribution leads to the frequency dependence of sex ratio selection and is the principle that escaped Fisher's predecessors such as Darwin. This frequency dependence lies at the centre of so much of sex allocation theory that it virtually embodies the discipline, dominating sex ratio selection in most of the non-Fisherian models and governing the evolution of sex allocation in hermaphrodites (Charnov *et al.* 1976; Leigh *et al.* 1976; Charnov 1982).

The fundamental role of Fisher's theory in the realm of sex ratio models is perhaps evident only to the small audience that specializes in this discipline. The second role of his model, and the role undoubtedly evident to the wider audience, is as a possible explanation for the ubiquity of 1:1 sex ratios in nature. A long tradition of obsession with mammals, birds, and insects—species with sex chromosome systems—has led to a general impression that sex ratios in the overwhelming majority of species are $\frac{1}{2}$; in turn, Fisher's model has been upheld as the explanation of their ubiquity. It is to this latter point that the remainder of our chapter is addressed.

3. EMPIRICAL SEX RATIOS

The objective in this part of the chapter is to observe how often primary sex ratios in natural populations are consistent with Fisher's theory. There are two properties of his theory of interest: (1) do sex ratios satisfy equal investment (static tests)?, and (2) if perturbed from equilibrium, does the sex ratio evolve toward equal investment (dynamic tests)? Few dynamics tests have been conducted, so most of section 3 is concerned with static tests. Observing that a sex ratio satisfies equal investment does not, however, mean that it is necessarily consistent with Fisher's theory, as it is also necessary to consider whether Fisher's assumptions are satisfied in such cases. If observed sex ratios do not satisfy equal investment, Fisher's model is rejected, but it is again of interest to identify which assumptions are violated and to thereby determine whether some other model from sex allocation theory offers a more acceptable explanation.

One of the difficulties in considering whether sex ratios satisfy equal investment is that the relative cost of a son versus a daughter has virtually never been measured properly. For the most part, only qualitative estimates of differential cost have been proposed (reviewed in Charnov 1982, p. 35). For example, free living wasps often provide male larvae with fewer food reserves than female larvae, and the relative amounts of food have been used as the relative cost; in ants, the relative size of male versus female individuals has been used to measure cost (Nonacs 1986; Trivers and Hare 1976). Although these measures likely reflect the direction of differential cost, there is no assurance that they offer quantitative measures (cf. Charnov 1982). Recently, Bull and Pease (1988) proposed a procedure to estimate differential cost based on the association between brood size and sex ratio, but the method has limited applications.

In view of the uncertainty of how to estimate differential cost, it would seem that Fisher's theory was often quantitatively untestable, except that *equal* cost of sons and daughters is thought to be the rule. The basis for assuming equal cost in many groups is that parents make no investment in offspring beyond conception. If sex is not determined before conception (or before ovulation), as would be the case with most sex-determining mechanisms, it would be difficult to invest more in one sex than in the other sex, and thus it is doubted that many species do invest differentially in sons and daughters. The supposed ubiquity of equal cost of sons and daughters thus adds an important dimension to the apparent robustness of Fisher's theory; the selected primary sex ratio is often $\frac{1}{2}$ independent of differential mortality and degree of polygamy. Although various modifications of Fisher's model aside from unequal cost are known to select deviations from $\frac{1}{2}$, these cases have been regarded as exceptional, and the evolutionary stability of a primary sex ratio of $\frac{1}{2}$ has been treated as a rule in sex ratio theory.

Given that the relative cost of a son is assumed, the further question then

arises as to what sex ratios should be regarded as consistent with Fisher's model. The evidence from sex chromosome systems is that sex ratios are often indistinguishable from $\frac{1}{2}$ (see section 3.1). This invariance from $\frac{1}{2}$ may give the impression that tests of Fisher's model should always be based on statistically significant deviations from the predicted value. Yet it will be shown below that this apparent precision in sex chromosomes is misleading and that the sex ratio variance is substantial in systems without sex chromosomes. It is customary in the initial development of a science, when a set of alternative explanations is available, to identify those hypotheses which account for most of the variation, even though some of the variation remains unexplained. Agreement with equal investment will therefore be evaluated qualitatively, in the context of the data and the feasible alternative hypotheses. The following sections review sex ratios for different sex determining mechanisms and discuss the relevance of the observations to the different hypotheses. Section 3.1 argues that the 1:1 sex ratios typical of sex chromosome systems are not necessarily consistent with Fisher's theory or, in fact, with any theory of sex ratio adaptation. Section 3.2 then considers sex ratios when sex is determined by other, well-defined mechanisms, and section 3.3 briefly mentions studies of sex ratio dynamics.

It should be re-emphasized that, even when sex ratios are found to violate equal investment and we say that Fisher's model is rejected, the alternative model(s) supported by the data may be very similar to Fisher's model in predicting a sex ratio only slightly different from equal investment and in embodying many of Fisher's assumptions. To reject Fisher's model does not deny it an important role in the construction of the alternative theories nor does it imply that Fisher's model predicts a wildly different sex ratio than the one observed. Rather, the sex ratio of equal investment, and especially the sex ratio of $\frac{1}{2}$, has assumed the role of the premier null hypothesis in sex ratio theory, and the first goal in evaluating data is often to see whether the null hypothesis is rejected. In this sense, Fisher's theory is a label for a particular result and is consequently rejected whenever that result does not hold. The overall importance of Fisher's model and the foundation it provides for other sex ratio models is acknowledged in the organization of sex allocation theory (section 2) rather than from the language surrounding empirical tests of the models.

3.1 Sex chromosome systems

Inheritance of sex was discovered almost three decades before the publication of Fisher's theory. Many animals were shown to have *heterogametic* sex determination, whereby one sex was heterozygous (XY) and the other sex homozygous for sex factors (XX), the XX/XY difference ultimately controlling the development of sex (Wilson 1906). The earliest discoveries of heterogamety identified cytologically distinct sex chromosomes, but subse-

quent discoveries of heterogamety were also based on the detection of sex-linked markers and other methods (Aida 1921). When Fisher's theory was advanced, therefore, heterogamety and sex chromosomes were known in many insects and some vertebrates. Fisher's discussion of sex ratio evolution, however, was elevated above any consideration of particular sex-determining mechanisms.

3.1.1 *Are the 1:1 sex ratios adaptive?*

The long history of observed newborn sex ratios near $\frac{1}{2}$ that no doubt inspired Fisher to consider the matter were based chiefly on domestic birds and mammals, which are now known to have heteromorphic sex chromosomes (as are Darwin's 1871 examples of bird sex ratios). Ironically, even though these sex ratios are consistent with those predicted by Fisher's theory, they perhaps cannot be construed to support Fisher's theory. With just two sex factors, the primary sex ratio is determined by the relative proportion of XX and XY zygotes. Although genetic variation in sex ratio can arise in several ways (as variation in the segregation ratio of X and Y, as variation in the relative fertility of X and Y gametes, or as factors influencing the sex of XX and XY genotypes), the ubiquity of Mendelian segregation in diploids and the paucity of gene expression in the gametes of most animals leads one to doubt that sex ratios are free to evolve under heterogamety (Maynard Smith 1978, 1980; Williams 1979; Toro and Charlesworth 1982; Mrosovsky *et al.* 1984*a*).

The possibility that sex chromosome systems constrain sex ratio variation has been slow to be incorporated into considerations of sex ratio evolution. Yet the evidence so far accumulated from sex chromosome systems, based mostly on domestic and laboratory animals, suggests that genetic sex ratio variation, and even between-species sex ratio variation, is minor or non-existent in these systems, whether of male heterogamety (mammals: Clutton-Brock and Iason 1986; a dipteran: Toro and Charlesworth 1982) or female heterogamety (birds: Clutton-Brock 1986; Foster and McSherry 1980; snakes: Shine and Bull 1977). Major efforts by the agricultural industry to alter birth sex ratios in livestock and poultry have been almost complete failures (Kiddy and Hafs 1971), and, when heritable sex ratio variation has been detected in these systems, it is invariably of small magnitude and is often inseparable from differential mortality (e.g. Bar-Anan and Robertson 1975). The difficulty with which sex ratios can evolve in sex chromosome systems is highlighted by the fact that the most celebrated case of extreme sex ratio distortion in mammals occurs in lemmings and is due to a unique sex-determining mechanism in which the molecular-genetic basis of sex determination has been fundamentally altered from the state observed in other mammals (Fredga *et al.* 1976; Gileva 1980). Of course, one could argue that the absence of genetic sex ratio variation in sex chromosome systems is a constraint maintained as a consequence of Fisherian sex ratio selection and

that strong selection for a bias would eventually change the primary sex ratio, but there is yet little evidence to support this view.

It is usually impractical to measure primary sex ratios directly because such work requires chromosome analysis of cleavage embryos, unless egg and embryonic mortalities are known to be small; hence most studies have measured sex ratios at birth, hatching, or at even later stages and have consequently inferred the primary sex ratio. Even if one accepts the premise that variation of primary sex ratio is non-existent in these systems, it is surprising that these studies have failed to detect much variation in birth sex ratios. The few observations of deviations from $\frac{1}{2}$ in birth sex ratios from mammals appear to stem from differential mortality of embryos (e.g. Austad and Sunquist 1986; Gosling 1986; Clutton-Brock and Iason 1986; Hrdy 1987). Tests of Fisher's sex ratio theory are appropriate only in systems with genetic variation in primary sex ratio, although a more recent theory addressed the evolution of sex ratio through differential mortality of embryos (Maynard Smith 1980).

Evolution and maintenance of heterogamety. If heterogamety precludes genetic sex ratio variation and thereby prevents sex ratio evolution, one might take a broader perspective and consider whether the evolution of heterogamety itself provides some insight to sex ratio evolution: can heterogamety evolve and be maintained when selection favours a sex ratio other than $\frac{1}{2}$? Not surprisingly, the answer to this question is complicated (Bull 1983). Consider first the evolution of heterogamety amid genetic variation for alternative mechanisms such as polygenic or multiple-factor sex determination. Models of this process that incorporate Fisher's assumptions for equal cost of a son versus a daughter reveal that the sex ratio evolves to $\frac{1}{2}$, but heterogamety has no advantage over mechanisms with more than two factors.

More relevant to our problem, however, is whether heterogamety can evolve when selection favours a sex ratio different from $\frac{1}{2}$. Although some work suggests that a differential cost of sons versus daughters mitigates against the evolution of heterogamety (Bull 1983), it is also known that various factors can overcome selection against $\frac{1}{2}$ and lead to the evolution of heterogamety. One such effect is pleiotropy; an epistatic, male-determining gene with an intrinsic benefit to males (or a sex-determining gene closely linked to an allele with such a benefit) can increase in frequency and lead to the evolution of heterogamety despite selection against heterogamety *per se* (Rice 1986). Another factor that can lead to the evolution of heterogamety despite opposing selection is genetic drift in finite populations (although recurrent mutation would preclude long-term fixation of heterogamety). Therefore, the evolution of heterogamety may be facilitated by selection for a sex ratio of $\frac{1}{2}$, but various factors can lead to the evolution of heterogamety when a sex ratio other than $\frac{1}{2}$ is favoured. We can imagine that heterogamety

is more likely to evolve when the favoured sex ratio is close to $\frac{1}{2}$ than when it is far from $\frac{1}{2}$, however.

The preceding considerations apply to the origin of heterogamety. An even more important consideration is the maintenance of heterogamety long after it originated: if the favoured sex ratio differs from $\frac{1}{2}$ in a sex chromosome system, and if sex ratio variation is lacking, can a new sex-determining mechanism evolve that will allow sex ratio evolution? It appears that sex chromosome systems can persist despite strong selection against $\frac{1}{2}$, because, once present, they are progressively reinforced and become increasingly difficult to change. With male heterogamety, genes benefiting males are selected on the Y despite their effects on females, and a similar though less extreme process occurs on the X. The Y also reveals progressive degeneration over time. The longer an XX/XY system persists, the more likely it is to become irreversible, so that a new mechanism cannot readily evolve (Bull 1983; Bull and Charnov 1985). We conclude, therefore, that sex ratios in sex chromosome systems are largely uninformative of sex ratio selection; genetic sex ratio variation is often lacking, and these mechanisms may have little potential to evolve to an alternative mechanism, albeit that the origin of heterogamety probably occurs in conjunction with selection for a sex ratio near $\frac{1}{2}$. At present, the common occurrence of sex ratios near $\frac{1}{2}$ in sex chromosome systems provides no evidence for the precision of sex ratio adaptation.

Williams (1979) studied the between-family sex ratio variance in some vertebrates with sex chromosomes to identify possible constraints on sex ratio evolution. Rather than undertake a study of genetic sex ratio variation, Williams merely noted whether the between-family sex ratio variation deviated from the binomial expectation (which would result from random segregation of the sex chromosomes). The motivation for observing the between-family variance was that certain models predicted evolution of a large between-family sex ratio variance (e.g. Trivers and Willard 1973), and other models predicted evolution of a small between-family variance (Verner 1965). The family sex ratios from humans, five mammals, and one bird were each consistent with the binomial variance. Williams's observations are thus consistent with the more general suggestion that sex chromosomes prevent sex ratio evolution, although such observations might well be expected even if sex chromosomes allowed evolution of the *mean* sex ratio but simply restricted evolution of the variance.

If sex chromosomes and heterogametic mechanisms limit the opportunities for sex ratio evolution, perhaps theories of population sex ratio should be tested in species whose sex-determining mechanisms are not so confining. The population sex ratio of these mechanisms should reflect the selected sex ratio. However, it must be kept in mind that restricting a test of sex ratio theories to alternative sex-determining mechanisms may bias the observations against 1:1 sex ratios: these alternative sex-determining mechanisms

may have evolved and been maintained because sex ratios other than $\frac{1}{2}$ are favoured.

3.1.2 *The double standard*

As noted above, the sex ratios reported from species with sex chromosomes are typically near $\frac{1}{2}$, and the few known deviations are of small magnitude. Species with other sex-determining mechanisms show an order-of-magnitude more sex ratio variation than species with heterogamety and sex chromosomes (see section 3.2). However, the same evolutionary models have been tested in both kinds of systems. The inevitable result has been that sex ratio variation in the second decimal place has been the fulfilment of models applied to sex chromosome systems, yet this magnitude of variation has been ignored amid the greater variety of alternative mechanisms. An example of the preoccupation with small deviations from $\frac{1}{2}$ is revealed even in Fisher's discussion of the human data, in which the sex ratio at birth is close to 0.53; Fisher discussed the excess of male birth as having adaptive significance along the lines of his equal-investment theory.

There is no clear resolution of this double standard. If the close fit to a sex ratio of $\frac{1}{2}$ in many sex chromosome systems reflects constraints unique to these systems, it might be appropriate to attribute adaptational significance to small deviations from $\frac{1}{2}$. The difficulty in using sex chromosome systems to test sex ratio theory, however, is not only the small magnitude of deviations from $\frac{1}{2}$, but the *inconsistency* of the deviations and the difficulty in repeating them (Schlager and Roderick 1968; Casida 1971; Hrdy 1986, 1987; Clutton-Brock and Iason 1986). It is this inconsistency that has led to the situation that nearly all empirically-based advances in sex allocation theory have come from mechanisms other than sex chromosome systems (Charnov 1982). At the same time it must be realized that these advances are qualitative, based on broad patterns of variation.

It does not seem appropriate to abandon studies of sex ratios in sex chromosome systems. Instead, it should be possible to apply data from sex chromosome systems to models that specifically incorporate the constraints of sex chromosomes. For example, models of sex-linked segregation distortion are tailored to sex chromosome systems (Hamilton 1967; Lyttle 1979), and Maynard Smith's (1980) theory of sexual investment (which applies to species with sex chromosomes) was based on the premise that the primary sex ratio is fixed at $\frac{1}{2}$. In Maynard Smith's model, parents could manipulate the sex ratio by reducing brood size at the expense of sons or daughters, and parents could alter the investment per surviving offspring. We think that studies of sex ratio that incorporate the specific constraints of sex chromosomes will greatly enrich sex allocation theory and provide a critical dimension that is not readily accessible from studies on alternative mechanisms.

3.2 Sex ratios under alternative sex-determining mechanisms

We offer a brief overview of sex ratios from four sex determining mechanisms that appear to provide enough genetic variation in sex ratio to be useful for testing theories of sex ratio adaptation: arrhenotoky, paternal genome loss, environmental sex determination, and sex change. Following a brief introduction to each mechanism, sex ratios from species with that mechanism will be reviewed. We are concerned both with the fraction of cases conforming to equal investment and, of those cases that deviate, the evolutionary basis for the deviations. As will be pointed out, these mechanisms vary in the ease with which Fisher's assumptions can be violated, so that some mechanisms will *a priori* be more likely to yield equal-investment sex ratios than other mechanisms. Unfortunately, studies of sex ratio have often been predisposed toward demonstrating deviations from $\frac{1}{2}$, so the reported proportion of non-Fisherian sex ratios may exceed the true proportion. Furthermore, only a small fraction of species in any one group have been studied, and many groups are not represented at all.

The mechanisms considered below omit cytoplasmic factors and parthenogenetic or gynogenetic production of daughters, for which sex allocation theory predicts female excesses and for which extreme female excesses are often observed (examples in Bell 1982; Bull 1983). Cytoplasmic factors and uniparental mechanisms are defined on the basis of a violation of Fisher's assumptions so, even if sex ratios under those mechanisms did satisfy equal investment, Fisher's theory could not provide the explanation. (Arrhenotoky may be said to violate the 'letter' of Fisher's assumptions, but it does not obviously violate the spirit of his assumptions, hence it is considered below.)

3.2.1 *Arrhenotoky: Hymenoptera*

In this mechanism, which is also known as haplo-diploidy, males arise from unfertilized eggs and females arise from fertilized eggs. Arrhenotoky is found throughout the sexually-reproducing Hymenoptera, Thysanoptera (thrips), and in various mites, ticks, and scale insects. Fisher may not have been considering that his theory applied to haplo-diploidy when he assumed that 'each sex must supply half the ancestry of all future generations', but it has since been shown that a sex ratio equilibrium of equal investment applies to sex ratio variation acting in the mother; if sex ratio variation is manifested in the father, however, the sex ratio is selected toward production of all daughters (Hamilton 1967). In the Hymenoptera, at least, the mother controls whether each egg is fertilized or not—sperm are released individually for each egg by a valve separating the sperm-reservoir from the oviduct. Based on this anatomical evidence and the diversity of sex ratio behaviours observed in Hymenoptera, it is presumed that genetic variation in maternal control of sex ratio is not limiting; heritable variation in the sex ratio has been demonstrated in a few species (reviewed in King 1987). Thus, arrheno-

toky is potentially an appropriate mechanism for testing theories of sex ratio evolution.

Sex allocation theory offers two reasons why Fisher's model may not apply to arrhenotoky exactly, although the discrepancies may often be minor. The first reason is that arrhenotoky provides the mother with easy facultative control of the sex ratio, and selection of the sex ratio may thereby be influenced by various environmental effects on progeny fitness. Although such factors probably do not select large deviations from the Fisher equilibrium, they can easily select small but significant deviations from $\frac{1}{2}$ (recall section 2.2.3). The second reason is that arrhenotoky generates asymmetries in the genetic relationships between brothers and sisters and between parents and sons versus daughters. For example, in a monogamous population, sisters have identical paternal genomes, whereas brothers lack a paternal genome entirely; sisters thus share more genes with each other than they do with brothers (cf. Trivers and Hare 1976). These asymmetries can influence sex ratio selection, particularly with inbreeding (recall section 2.2.3).

We now consider sex ratios from three hymenopteran life history modes: grouped progeny parasitoids, colonial species, and solitary species.

Grouped-progeny parasitoids. Certain wasps and bees experience a life history involving group-structured matings: all of a female's offspring are raised in a single host (e.g. an insect pupa or a fruit), and on emerging as adults, they mate amongst themselves and perhaps a few other families. The sex ratios of these species range from heavy female excesses to sex ratios near $\frac{1}{2}$ (e.g. Fig. 4). Hamilton (1967) added a new dimension to sex ratio theory in observing these female excesses and explaining why Fisher's model does not apply (section 2.2.3). This area of sex ratio theory has been expanded considerably since 1967 and offers one of the most satisfying blends of empirical and mathematical approaches to be found in evolutionary biology (Hamilton 1967, 1979; Werren 1980; Wilson and Colwell 1981; Herre 1985). It is thus an area where Fisher's model is usually incorrect but where sex ratios are explained by an alternative model of sex allocation theory.

Hamilton's (1967) examples of female excesses were taken from various parasitoid and parasitic wasps and bees, a few parasitic haplo-diploid mites, and a haplo-diploid bark beetle. Most of these parasitoids are not expected to exhibit Fisherian sex ratios because their life-cycle encourages selection of female excesses through group-structured matings. However, observations have been made for one case of group-structured matings in which sex allocation theory predicts Fisherian sex ratios. As described in section 2.2.3, the selected sex ratio deviates from the Fisherian value according to the group size (the number of competing families at the mating site); the selected sex ratio is for a single son if a mother's progeny mate entirely among themselves, but the predicted sex ratio may approach equal investment if

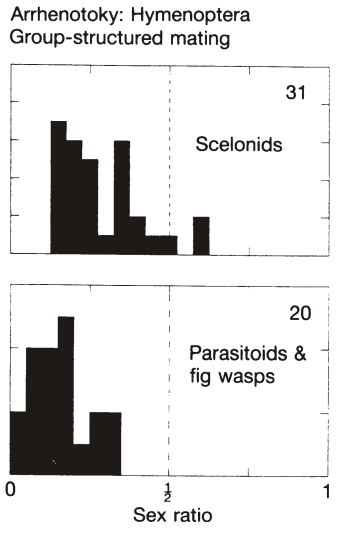

Fig. 4. Sex ratios in wasp species experiencing local mate competition. Lower: parasitoid and fig wasps (Hamilton 1967). Upper: scelonid wasps (Waage 1982). There is a pronounced excess of females in most species. In these two studies, the sex ratio is of newly-emerged adults and is thought to represent the primary sex ratio. The numbers 31 and 20 refer to the number of data (species) in the respective histograms.

group size is large. Thus if group-structured mating is the sole reason for the female excesses in these species, we should observe sex ratios of $\frac{1}{2}$ when group size is large.

Two options exist in studying large group sizes. We may investigate average sex ratios in species that always mate in large groups, or we may study sex ratios under the occasional conditions of large group size in species that typically mate in small groups. Sex ratios have been studied in only a few species that typically mate in large groups, and the sex ratios range from approximately 0.35 to 0.58 (Waage 1982). Over the range from small group sizes to large group sizes, however, the sex ratio changes in the expected direction.

Two patterns have been observed when large group size has been studied in species that typically mate in small groups: (1) the sex ratio is not affected by group size (hence there is apparently no facultative control of sex ratio), or (2) the sex ratio progresses from a strong female excess at small group sizes toward a greater fraction of males at large group size; the sex ratios at large group size often lie within the range 0.4–0.6, and in some cases they do not differ significantly from 0.5 (reviewed in Charnov 1982, pp. 78–87). One complication with this latter kind of analysis has recently been discovered by Herre (1985); the expected sex ratio at large group sizes is not necessarily $\frac{1}{2}$ but depends on the usual level of inbreeding (hence depends on the usual group size). In highly inbred species, the expected sex ratio at large group size

is only $\frac{1}{3}$; only in outbred species is the expected sex ratio $\frac{1}{2}$ for large groups. Herre's study of fig wasps demonstrated the existence of the inbreeding effect, but these corrections for inbreeding have not been incorporated into most of the above studies, and so the apparent fit with $\frac{1}{2}$ at large group sizes may actually be contrary to the sex ratio predicted by sex allocation theory.

In summary, sex ratios are usually non-Fisherian in group-structured matings—they tend to female excesses. The female excesses are in accord with other models of sex ratio theory. The one case in which sex allocation theory predicts Fisherian sex ratios is when group size is typically large; observations are consistent with this prediction on a coarse scale of analysis.

Colonial species. Colonial existence is defined by the mutual endeavours of individuals toward the maintenance of a common home and the co-operative rearing of offspring. Colony members are often related (e.g. sisters), and they may exhibit extensive division of labour, as with distinct reproductive and non-reproductive castes in ants and many bees. Mating usually occurs away from the nest, so group-structured mating is not obviously applicable. Female reproductives are typically larger than males; hence investment ratios may be more appropriate than actual sex ratios. Investment has typically been calculated from dry weights of the reproductive individuals at the time of emergence.

Despite satisfying many of the assumptions of the Fisher model, the investment ratios of many ants favour females (Fig. 5). The apparent basis for the female excesses was proposed by Trivers and Hare (1976): under monogamous mating and single-queen colonies ('monogyny'), workers are more closely related to female reproductives than to male reproductives. Selection favours an average colony sex ratio (investment ratio) of $\frac{1}{4}$ if genetic variation is expressed among the workers; the Fisherian investment ratio of $\frac{1}{2}$ is favoured if the genetic variation is expressed among queens. These calculations do not apply to two other types of colony structure, polygyny (multiple queens per colony) and slave-making. The prediction for slave-making species is that the sex ratio should satisfy equal investment: slaves rear the reproductives, so inherited variation in worker control of sex ratio cannot be expressed. It is difficult to anticipate the possible complications of sex ratio selection in polygynous populations, but if the multiple queens are close relatives, the investment ratio favoured under worker control is nearer to equality than under monogyny because the disparity in the relatedness of a worker to male versus female reproductives is reduced (Trivers and Hare 1976; Nonacs 1986).

The latest summary of data on ant sex ratios was provided by Nonacs (1986) (Fig. 5). For monogynous populations, the average investment ratio across species was 0.28, whereas the average sex ratio was 0.58. For slave-making ants, the investment ratio was 0.52 (sex ratio 0.63). For polygynous populations, the average investment ratio was 0.52 (sex ratio 0.69). Thus

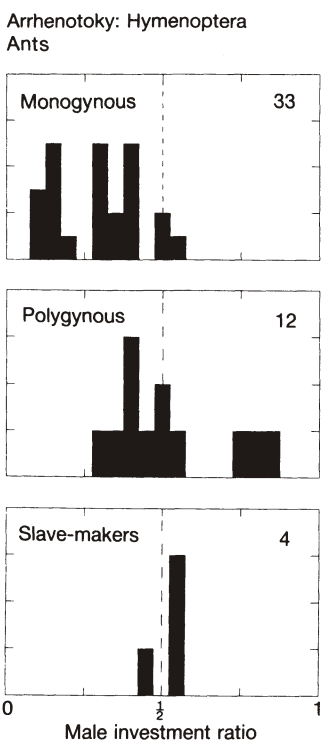

Fig. 5. Male investment ratios in various species of ants (data from Nonacs 1986). As predicted by Trivers and Hare (1976), investment ratios are near 1/2 in slave-makers but favour females in monogynous colonies. Predictions for polygynous colonies are sensitive to various parameters that have not been empirically determined, but many of the investment ratios appear to be superficially consistent with Fisher's model.

investment ratios in two of the three categories of ants are broadly consistent with equality, whereas investment ratios in the third category of ants are consistent with Trivers and Hare's theory. There is considerable variance in sex ratio within each of these classes that is not yet explained.

Noonan (1978) studied a primitively social paper wasp to test the Trivers and Hare hypothesis for a species on the threshold of eusociality. The sex ratio measured at the end of the summer was not significantly different from $\frac{1}{2}$, in accord with equal investment.

Solitary species. Many bees and wasps nest in isolation and lack sib mating; hence these species would seem *a priori* to be likely to exhibit equal investment sex ratios. Unfortunately, estimates of the sex ratios in these species are difficult to obtain. The sex ratio of emerging adults often changes seasonally, and the sex ratio may be affected by the nest site: some bees and

wasps nest in narrow tunnels in wood that are located by the mother (not constructed by her); tunnel diameter places an upper limit on offspring size, and in many species males are smaller than females so the mother alters sex ratio according to tunnel diameter. Werren and Charnov (1978) and Seger (1982) have further shown that the sex ratio in bivoltine species is selected to change between the first and second generations of each year.

Despite problems in estimating differential costs and in estimating the sex ratio, these species should allow tests of the simple 'Fisher inequality' that the cheaper sex should be overproduced. The evidence supports this prediction (Fig. 6; Trivers and Hare 1976; Charnov 1982). Yet, other analyses of these data are not so consistent with the equal-investment theory (see the legend to Fig. 6).

Overview. Many, if not most, of the sex ratios reported from Hymenoptera do not satisfy equal investment, although many species remain to be studied. The data are obviously biased in that the most thoroughly studied sex ratio phenomena are exceptions to equal investment and were studied in detail *because* they violate equal investment. Sex allocation theory seems to explain

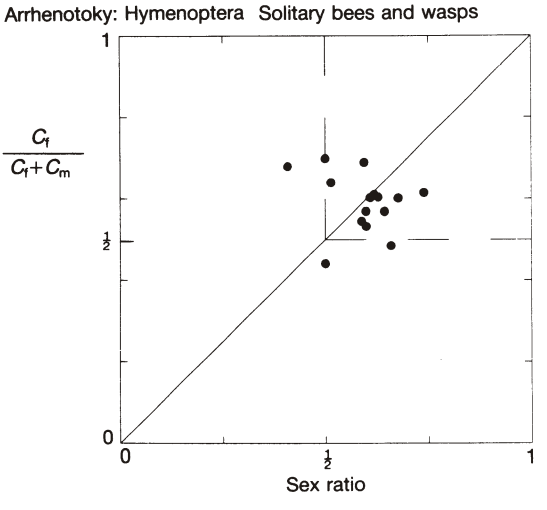

Fig. 6. Sex ratios (not investment ratios) versus the ratio $c_f/(c_f+c_m)$, where c_f is the estimated cost of a daughter (c_m for a son). The diagonal line is the relation expected under Fisher's equilibrium of equal investment. The upper right quadrant encloses points for which sons are cheaper to produce than are daughters and sons are overproduced—a weak test of the equal-investment theory. Alternatively, the data may be inspected to observe whether the sex ratio increases or decreases with the value of $c_f/(c_f+c_m)$. The observed regression is negative rather than positive although not significantly so. Therefore, it is not clear how well the data support the equal-investment theory. Data are from Trivers and Hare (1976).

at least the major deviations from $\frac{1}{2}$, usually by modifying a single assumption of the Fisher model—group matings, asymmetric sib–sib relatedness, and so forth, which for the most part, is evident on casual inspection. The central role of Fisher's model in the organization of sex allocation theory is thus highlighted in the empirical analysis of these sex ratios.

3.2.2 *Paternal genome loss: coccoids and mites*

In this genetic system, eggs of both sexes are fertilized by sperm, but a male's paternal genome is not transmitted (reviewed in Bull 1983). With maternal control, the sex ratio evolves under paternal genome loss (PGL) just as under arrhenotoky, and the equilibrium of equal investment applies, given no other violations of the Fisherian assumptions. This genetic system is observed in many coccoid insects (scales, armoured scales, mealybugs), in phytoseid mites, and in fungal gnats, but the details vary as to how males eliminate their paternal chromosomes. In various coccoids, for example, the paternal genome is eliminated in sons during development; in fungal gnats and in some other coccoids, it is retained by the male throughout its life but then is destroyed during spermatogenesis. PGL can, in principle, operate under various kinds of sex-determining mechanisms, including female but not male heterogamety, although the inheritance of sex is known or suspected in only a few cases. PGL systems have been found to show considerable maternal effects on sex ratio, so it seems justified to anticipate genetic variation in sex ratio. The equilibrium does not satisfy equal investment if sex ratio is controlled by the father or the zygote, and various violations of Fisher's assumptions may arise even with maternal control of the sex ratio, due to the asymmetric relatedness between sons, daughters, and parents (as also applies under arrhenotoky).

The sex ratios in at least some strains of *Sciara* apparently satisfy equal investment, because sex is inherited according to maternal monogeny: one genotype of mother (aa) produces all sons and the other genotype of mother (Aa) produces all daughters, half of which will be son-producers, half daughter-producers (Metz 1938). As explained in section 2.2.1, the sex ratio of such a system necessarily satisfies equal investment even when the cost of a son differs from the cost of a daughter.

Scattered evidence suggests that sex ratio variation exists in coccoid insects and that the sex ratio is controlled by the mother. For example, Nur and Brett (unpublished) observed significant variation in primary sex ratio among strains of a single species (see below and Fig. 7). Evidence of maternal control of the sex ratio is provided by widespread observations of maternal age effects on sex ratio and by apparent predetermination of sex in the egg (as indicated by egg colour; Nur 1988). Even with this much information, however, the estimation of primary sex ratios in coccoids faces several complications. Males either eliminate the paternal genome in cleavage or inactivate it, so they are either haploid or effectively haploid. Consequently,

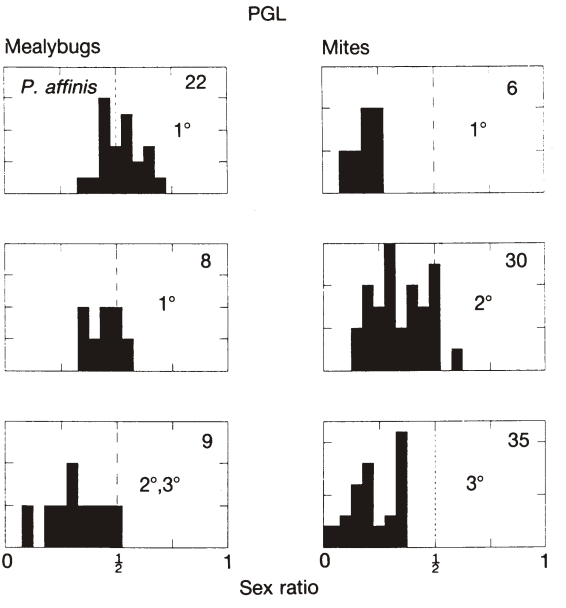

Fig. 7. Sex ratios in two taxa with paternal genome loss. The primary sex ratios appear to be Fisherian for mealybugs but not for phytoseid mites. **Left:** Sex ratios in mealy bugs. Top: primary sex ratios in 22 strains of *Pseudococcus affinis*. Middle: primary sex ratios in three species from the wild and five species in the lab (*Pseudococcus affinis* being omitted). Bottom: secondary and tertiary sex ratios reported in the literature (nine data representing seven species). **Right:** sex ratios in phytoseid mites. Top: six observations of primary sex ratios in three species. Middle: secondary sex ratios. Bottom: tertiary sex ratios. References are U. Nur and B.L.H. Brett (unpublished) (left top and left middle); James (1937), Nelson-Rees (1960), Hausermann (1966), and Sinha (1972) (left bottom); Amano and Chant (1978), Schulten *et al.* (1978), Helle *et al.* (1978) (right top); Sabelis (1985) (right middle); Dyer (1975) (right bottom).

sex ratios observed even as early as hatching could be affected by appreciable male mortality (Smith and Shaw 1980). Furthermore, various environmental factors (including maternal age) have been reported to influence sex ratio, so any samples must avoid these biases. These difficulties are emphasized by Nur (1988) in a review and discussion of sex ratios in armoured scales. Sex ratios are known from second-instar larvae and later stages in several species, and many sex ratios are reported to be approximately $\frac{1}{2}$, but some extreme deviations from $\frac{1}{2}$ are also known. Interpretation of these data are difficult because the primary sex ratios are unknown. The primary sex ratio was studied in one species and favoured males, but it is not clear that the samples were free of maternal-age biases (cf. Nur 1988).

A set of unpublished studies has identified the primary sex ratio in various species of mealybugs (U. Nur and Brett, unpublished personal communica-

tion; e.g. Fig. 7). The primary sex ratios cluster about $\frac{1}{2}$, even though reported adult sex ratios have been heavily female-biased. Nur and Brett have documented strong maternal age effects on the primary sex ratio. (The sex ratio increases with maternal age in some species, decreases in others, and shows no evidence of an age effect in yet others.) Weak temperature effects are also known in some cases. Overall, therefore, the data largely support Fisher (equal investment) despite these complications to measuring the primary sex ratio.

In contrast to the observations on mealybugs, sex ratios in phytoseid mites appear to favour females (Fig. 7). PGL is suspected in phytoseids on the basis of widespread male haploidy, but it has been demonstrated convincingly in only a few species. Adult sex ratios have been studied in various species and nearly always favour females. Primary and secondary sex ratios are also female-biased. Various authors have noted that the female excesses may be selected through group-structured mating (Charnov 1982; Sabelis 1985; Crozier 1985), but the evidence is somewhat equivocal, and the female excesses are potentially explained by group-structured mating, by inbreeding (with or without group-structured mating), or through sex ratio variation expressed in zygotes.

3.2.3 *Environmental sex determination (ESD)*

In this mechanism, sex is determined largely in response to some environmental effect experienced early in life. For example, in many reptiles and in silverside fish, the incubation temperature of the embryo or larva determines its sex; in parasitic nematodes of the family Mermithidae, sex is determined according to the nutrition derived from the host; and in the marine worm *Bonellia*, sex is determined in the larva according to the proximity of an adult female (reviewed in Bull 1983).

In many systems of ESD, there can be no more than a small inherited component to sex, but it is not unreasonable to assume that sufficient genetic variation in sex ratio occurs so that sex ratio evolution is virtually unconstrained in the long term. In silversides, Conover and Heins (1987*b*) observed wide between-family sex ratio variation due to sire, indicating genetic variation in sex ratio. Bull *et al.* (1982) reported significant between-clutch variation in sex ratio from turtle eggs incubated under similar conditions, which is again consistent with genetic variation, but maternal effects could not be ruled out in this study and the evidence is best regarded as equivocal.

Sex ratios have been studied in two groups with ESD—reptiles and fish. The sex ratios of many reptiles with ESD are not Fisherian, and they in fact appear to be unexplained even in the larger realm of sex allocation theory. In the one study of a fish with ESD, sex ratios were essentially Fisherian.

Testing sex ratio theories under ESD requires a clear understanding of the appropriate life history stages to measure sex ratio. Even though sex is not determined when eggs are laid, the primary sex ratio is calculated as in any

other system: it is the sex ratio that would be observed after the period of sex determination if all zygotes survived to be counted. The secondary sex ratio is the sex ratio among surviving hatchlings/newborn.

Fisher's model predicts a primary sex ratio of $\frac{1}{2}$ under ESD, assuming no postlaying parental care. However, ESD is generally expected to introduce a covariance between progeny sex ratio and fitness (which are assumed to be zero in the Fisher model), and these in turn may affect the sex ratio equilibrium (recall section 2.2.3). In the special case that all fitness effects occur before hatching as viability effects, selection favours a *hatchling* sex ratio of $\frac{1}{2}$, and the primary sex ratio will favour the sex that inadvertently experiences the higher mortality. (We refer to this model as the ESD sex ratio model.) A hatchling sex ratio of $\frac{1}{2}$ is not generally predicted if the fitness effects that covary with sex ratio are manifested beyond hatching, but in the case of reptiles, the major fitness effects of nest site may well occur before hatching (see Bull 1983, for some additional but unimportant restrictions on these calculations).

ESD offers the special problem that the sex ratio may vary seasonally and may vary between years because of environmental fluctuations. The predicted sex ratio equilibria (as above) have assumed the absence of environmental fluctuations, and although they should be approximately correct even when the environment fluctuates, the fluctuations necessarily limit the precision with which the models may be tested. For example, primary and hatchling sex ratios observed over a single year may offer little insight to the long-term, average sex ratio.

Reptiles. Nest temperature is thought to be the major determinant of sex in reptiles with ESD (reviewed in Bull 1983), but three patterns of ESD are known: (1) males develop at high temperatures, female at low ones (alligators and caimans, some lizards); (2) females develop at high temperatures, males at low ones (many turtles); (3) males develop at intermediate temperatures, females at high and low extremes (most *Crocodylus*, some turtles). Incubation temperature has direct effects on embryo survival, and it also correlates with various environmental factors that influence nest survival (e.g. predation, flooding). Although it is possible for mothers to differentially provision sons versus daughters, it seems more likely that investment per son is the same as per daughter. The Fisher model therefore predicts a primary sex ratio of $\frac{1}{2}$. The ESD models predict a sex ratio of $\frac{1}{2}$ at hatching if all correlations between fitness and sex ratio occur as effects on egg viability.

Primary sex ratios and hatchling sex ratios in the wild have been observed for three crocodilians and five kinds of turtles (Table 1). Nest sex ratios and hatchling sex ratios range from $\frac{1}{2}$ in some species to heavy female excesses in others. If the data reflect population-wide sex ratios, some cases support Fisher, some support the ESD model, and some seem to have such extreme female excesses as to deviate from both models. Unfortunately, many of

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Table 1
Sex ratios reported in reptiles with ESD

Reptile	Sex ratio	Age*	Reference
Crocodilians			
Alligator	0.2	N, H	Ferguson and Joanen (1983)
Fresh-water crocodile	0.2–0.6†	H	Webb and Smith (1984)
	0.5†	H	Smith, unpublished (1983–5)
Mugger crocodile	0.25, 0.43‡	N, H	Lang and Whitaker (1988)
Turtles			
Loggerhead sea	0.5	N	Mrosovsky *et al.* (1984*b*)
Green sea	0.3	N	Standora and Spotila (1985)
	0.5	N	Mrosovsky *et al.* (1984*a*)
Leatherback sea	0.5	N	Mrosovsky *et al.* (1984*a*)
Snapping	0.3§	N	Brooks and Nancekivell (1984)
Map	0.24**	H	Vogt and Bull 1984
	0.45**	H	Bull unpublished (1982)

* Stage of life history at which the sex ratio was measured: N, nests (primary sex ratio); H, hatchlings (secondary sex ratio). The sex ratio here is the proportion male, rounded to the nearest 0.1.

† Hatchling sex ratios from different river systems reported by Webb and Smith (1984) were: 0.31, 0.29, 0.23, 0.40*, 0.20, 0.34*, 0.36, 0.61 (considering only those samples with more than 100 individuals; the sex ratios with asterisks contained more than 1000 individuals). Few of the deviations are thus in the male direction. A.M.A. Smith has provided us with unpublished sex ratios of hatchlings from approximately 20 nests in each of three years from the McKinlay river system: 0.55, 0.44, and 0.44, with sample sizes ranging from 114 to 201. The overall sex ratio is 0.49; the hatchling sex ratio reported for this same river system in Webb and Smith (1984) is 0.25, based on 60 animals. Webb and Smith pointed out that cool nests, which produce females, were subject to greater mortality than warm nests because the cool nests took longer to hatch. The primary sex ratio thus favours females more than the hatchling sex ratio.

‡ A sex ratio of 0.25 was observed over 1977–83 for 52 nests (734 hatchlings); a sex ratio of 0.43 was observed in 1986 (20 nests). The study site was a field enclosure and may not represent a natural situation (Lang, personal communication).

§ The study site is at the northernmost extent of the snapping turtle's range, and this population may not have existed prior to civilization, as most turtle nests are constructed in man-made earthen structures (R. Brooks, personal communication). This population may therefore not represent a natural or equilibrium state.

** Data are from a population of map turtles studied on the Mississippi River in 1980 (Vogt and Bull 1984) and again in 1982 (sex ratios unpublished, but the study was described in Bull 1985). The sex ratio of hatchlings gathered from known nests on several beaches of a single island was approximately 0.24 in 1980 and was approximately 0.45 in 1982, the difference being statistically significant. The summer of 1982 was in fact cooler than the summer of 1980, and cool temperatures produce males in map turtles, so perhaps climatic variation caused this change in sex ratios between years. Possible sources of bias in sampling these sex ratios include: (1) nests are easier to locate in the sites that produce females than in the sites that produce males; (2) only a small fraction of the nesting beaches have been studied; and (3) for many years before the study, the nesting beaches were maintained in an artificial state by dredging operations, the anticipated effect of which is an overproduction of females (discussed further in Vogt and Bull 1984).

these studies admit the possibility of serious collecting biases or man-made influences on the population (see the legend to Table 1). For example, primary and hatchling sex ratios are virtually never calculated from the distribution of nests over the entire population. Yet, sex ratio depends highly on nest site, so the failure to locate one or more general nesting areas for the population or even the failure to locate nests in specific types of microsites can generate a drastic bias in the sex ratio estimate (e.g. Vogt and Bull 1984; Ferguson and Joanen 1982, 1983; Mrosovsky *et al.* 1984*a*). Overall, the studies of crocodilians are probably the most successful in avoiding these pitfalls, yet crocodilians also have the most extreme female excesses reported. The sex allocation literature offers several alternative models to consider as an explanation for the female excesses, but as yet, none seems especially plausible (cf. Bull 1983).

Fish. Conover and Kynard (1981) and Conover and Heins (1987*a*,*b*) have reported moderate temperature effects on sex determination in the Atlantic silverside fish, although inherited effects on sex determination are also evident. The observed sex ratios in populations with ESD and in one population that lacks ESD are very close to $\frac{1}{2}$. Thus, in contrast to ESD in reptiles, these fish seem to agree with Fisher's prediction and with the basic ESD model.

3.2.4 *Sex change*

Sequential hermaphroditism may seem to be a strange mechanism to test sex ratio theories, but Charnov (1979, 1982) showed that the same principles underlying sex ratio theory underlie the time spent as male and as female in organisms that change sex. The sex ratio in a population of hermaphrodites is not generally expected to obey equal investment, but certain kinds of populations with sequential hermaphrodites are effectively dioecious (separate sexes), and the sex ratios in these are expected to obey equal investment at least approximately. These quasi-dioecious populations consist of two morphs—a pure sex that does not change sex and a hermaphrodite that starts life as the sex opposite that of the pure sex. In populations where the hermaphrodite realizes the vast majority of its reproductive success through its first sex phase, the ratio of pure sex to hermaphrodites conceived should satisfy equal investment.

Charnov (1982) presented sex ratio data from relevant populations of pandalid shrimp and wrasses. In some populations, the frequencies of the two morphs were close to the expected value of $\frac{1}{2}$, whereas in others there was considerable deviation from $\frac{1}{2}$ (Fig. 8). The mean of the data is consistent with the Fisher model, but we do not yet understand the significance of the appreciable variance around the mean—whether the variance arose from sampling problems or represents substantial deviations from $\frac{1}{2}$.

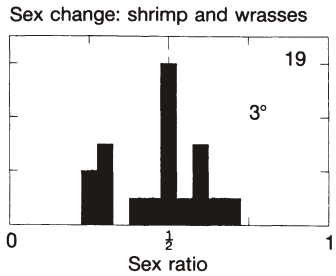

Fig. 8. Sex ratios in sequential hermaphrodites—pandalid shrimp and wrasses. Sex ratios are taken among adults (tertiary) in populations polymorphic for a pure-sex morph and a morph that changes sex but which experiences nearly all of its fitness through its first sex phase. The sex ratios are variable but centre on 1/2. (Data from Charnov 1982: 12 points for shrimp, pp. 150, 157, 159, 160; 7 points for fish, pp. 166, 172.)

3.2.5 *Female excess in a polychaete: differential cost?*

Polychaete marine worms display a variety of sexual systems, from sequential hermaphroditism, to simultaneous hermaphroditism, to dioecy. A striking and persistent female excess was reported by Akesson (1970) in a dioecious strain of *Ophryotrocha labronica* from Naples (since designated 'Naples I'). Bull and Pease (1988) suggested that a female excess was consistent with equal investment, but the problem is complicated. Consider the following observations reported by Akesson (1970, 1972*a*):

1. The sex ratio was approximately 0.31.
2. All sex ratios were evaluated after birth, possibly in the adult stage. Differential mortality was apparently too low to account for the excess of females, as Akesson (1970) stated that the total of animals scored for each family was close to the original number of eggs. At least 26 per cent of the brood must die to generate a sex ratio of 0.32 from $\frac{1}{2}$, and the suggestion is that mortality was much lower.
3. Selection for increased sex ratio eventually yielded $\frac{1}{2}$, but selection for a decrease failed to change it.
4. Brood size was negatively correlated with sex ratio (correlation $= -0.43$; $p < 0.01$).

Why is the sex ratio not $\frac{1}{2}$, especially when genetic variation for $\frac{1}{2}$ exists? Differential mortality seems unlikely (although the data on this point need clarification). Bull and Pease developed an estimation procedure which showed that the negative correlation between brood size and sex ratio was consistent with a greater cost per son than per daughter—brood size is expected to decrease with sex ratio if a son costs more than a daughter. The

equilibrium sex ratio predicted from their analysis was in fact significantly *lower* than 0.31, in which case the shortage of males is not surprising; rather males are more common than expected. The sex ratios of some other strains were also significantly less than $\frac{1}{2}$ (Akesson 1975), but family size/sex ratio data were not reported for these cases, so the differential cost could not be estimated. What is interesting and puzzling about this case is that there is no suggested biological basis for a greater cost of sons than daughters; the evidence of differential cost simply emerges from the relationship between brood size and sex ratio. This method of estimating differential cost is potentially applicable to other species, and it has the advantage that it addresses the direct fitness consequences of a differential cost rather than relying on proximate causes of differential cost.

3.3 Evolutionary dynamics of sex ratio

A fruitful means of testing a sex ratio theory is by observing the dynamic process of sex ratio evolution. As most natural populations are thought to be at or near equilibrium, dynamical studies are best done through artificial manipulations, and they require species with genetic variation in sex ratio. Although various organisms are available for such work, few studies have been conducted. Perhaps the most appropriate studies of this nature were conducted on the polychaete *Ophryotrocha labronica* by Akesson (1972*a*,*b*, 1975, 1977). This species reveals considerable between-strain sex ratio variation, ranging from 0.5 to 0.3. Akesson performed six crosses among three of these strains (strain sex ratios of 0.31, 0.37, 0.46) and followed sex ratios for two or three generations. F_1 sex ratios exceeded 0.65 in two of these crosses, yet in the third generation the sex ratios had dropped to at least 0.37, both drops being significant. F_1 sex ratios in the other four crosses were between 0.46 and 0.52; only one of the F_2 sex ratios was significantly lower than its initial value, and F_3 generations were not reported. These results are interesting, but our present perspective requires that they be conducted over additional generations before we can identify the final equilibria and determine whether the approach to equilibrium is monotonic or oscillatory. We also require some understanding of the evolutionary basis of the differences in strain sex ratio (see section 3.2.5).

Anomalous sex ratio trajectories have been reported for houseflies and for *Ophryotrocha* in which strains progressively overproduced males until extinction from a lack of females (Akesson 1977; Milani 1971). Intense inbreeding was involved in at least the *Ophryotrocha* study, but only one of eight *Ophryotrocha* strains treated similarly exhibited such an anomaly. Obviously, neither of these observations is consistent with Fisher's model, but no plausible explanation is apparent.

Lyttle (1979) studied sex ratio evolution in a strain of *Drosophila* in which an autosome with segregation distortion was translocated to the Y chromo-

some. Polygenic, autosomal suppressors of the distortion accumulated in some of the caged populations, this evolution being consistent with Fisherian sex ratio selection. In addition, enhancers of the distortion also evolved (these in linkage with the distorter); evolution of linked enhancers is consistent with non-Fisherian models of sex ratio evolution through Y-linked segregation distortion. In summary, although the study of sex ratio trajectories can offer appropriate tests of Fisher's sex ratio theory, this area is currently a void in sex allocation theory.

4. EVOLUTIONARY CONSTRAINTS AND SEX ALLOCATION THEORY

The blend of models and data in modern sex ratio theory, combined with knowledge of sex-determining mechanisms, presents an unusual opportunity to evaluate the nature and importance of constraints on evolution. It is widely recognized that constraints have important effects on evolutionary processes, yet it is often difficult to identify constraints as specifically as has been done in sex ratio theory. The foregoing presentation has offered several examples in which sex ratio evolution depends on the sex-determining mechanism:

1. Sex chromosome systems often lack genetic variation in primary sex ratio and thus preclude sex ratio evolution (section 3.1).
2. Many mechanisms prevent differential parental investment in sons versus daughters before conception, because offspring gender is determined independently of parental genotype or parental behaviour (recall the opening paragraphs of section 3).
3. Arrhenotoky (haplo-diploidy) generates asymmetric brother-sister relatedness, which in turn influences sex ratio evolution in particular cases (sections 2.2.3 and 3.2.1).
4. Under environmental sex determination, the sex ratio varies according to environmental fluctuations (section 3.2.3).

This list could be continued, but these examples are sufficient to illustrate three lessons about constraints. First, the patterns of sex ratio variation between and within species would often be impossible to interpret without knowledge of the sex-determining mechanism, yet when the mechanism is known, these patterns are explicable. For example, without knowledge of the sex determining mechanism, it would indeed be very puzzling to understand why hymenopterans (with arrhenotoky) exhibit so many interesting sex ratio phenomena, whereas dipterans (with heterogamety) do not (Charnov 1982). Second, the constraints do not fall into any obvious pattern. In some cases a

constraint is of the form that sex ratio evolution is prevented, in others the constraint influences the sex ratio equilibrium that is selected, and so on. Each example is so peculiar that it does not readily lead to the identification of other constraints. Third, the constraints imposed by the sex-determining mechanism may constitute the most important constraints on sex ratio evolution. If a variety of additional constraints were also important, it is difficult to imagine that current sex ratio theory would be as successful as it appears to be. We must caution, however, that constraints may render sex ratios consistent with a model for erroneous reasons, as observed in the apparent concordance between Fisher's model and the sex ratios of sex chromosome systems. It is difficult to know what parallels will be found between these lessons and the lessons from evolutionary constraints observed outside the realm of sex ratio evolution.

5. CONCLUSIONS

The mathematical basis of sex ratio theory consists of a well-integrated set of models that apply to specific, alternative biological settings. Fisher's model is at the core of these models because it provides a nucleus from which to consider the entire variety of sex ratio models. On an empirical level, Fisher's model appears to be a sufficient explanation of population sex ratios in many cases, but there are also many exceptions to equal investment that are explained by other models in sex allocation theory. The sex ratios of sex chromosome systems may not support any adaptive theory.

The success of sex ratio theory is exceptional when compared to other evolutionary disciplines: a variety of simple models have met with qualitative empirical success in predicting the directions of and magnitudes of sex ratio deviations from $\frac{1}{2}$. The work is still at the stage of explaining major patterns, and many small variations in sex ratio remain unexplained. The success of sex ratio theory seems to be due to the ease of measuring sex ratio and to the overriding importance of frequency-dependent selection that can be predicted from simple observations—the rules of inheritance (Mendelism and biparentalism) and simple aspects of population structure. In short, sex ratio equilibria can often be predicted without measuring fitnesses.

One of the more interesting possible directions for future work in sex ratio and sex allocation theory concerns the nature of constraints on evolution. Although it is widely acknowledged that constraints are important in evolution, little progress has been made outside of sex allocation theory in understanding exactly how and when constraints are important. The many empirical successes of sex allocation theory provide a logical foundation for a theory that is modelled around constraints imposed by the organism's physiology and genetics. Some progress has been observed in understanding the importance of constraints in sex ratio evolution, but further progress can

be expected as empirical studies begin addressing the second-order discrepancies (and some of the remaining first-order discrepancies) between observations and predictions.

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