

A HIERARCHICAL VIEW OF SEX-RATIO PATTERNS

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"Is it a boy or a girl?" (Todo el mundo)

Most organisms have two sexes; thus it is reasonable to ask what is the ratio of the sexes, and does this ratio fluctuate in a predictable manner. Basic researchers have long recognized that the sex ratio is a phenotypic trait of an organism, and therefore may be subject to natural selection. In addition, it has been widely noted that (i) the relative fitness of a male versus a female ranges widely according to a variety of ecological factors (e.g., size of individual, local sex ratio, inbreeding), implying that the selection of sex-ratio traits should be strong; and (ii) relative to other traits studied by behavioral ecologists, sex ratios can often be measured with a high degree of precision. Hence, the subject of sex ratios is ideal for testing the efficacy of natural selection, and for testing our understanding of the selective process. Applied researchers also have a long history of interest in sex ratios. Efficiency of live-stock breeding would be greatly enhanced by control over the sex ratio; mass-rearing of parasites as biological control agents is often improved by a female-biased sex ratio; and causing a male-biased sex ratio in a population of pests would aid in reducing the amount of damage.

This paper is an introduction to a way of viewing sex-ratio puzzles that has had some success—both theoretically and empirically—and points out some of the difficulties of sex-ratio problems. After a methodology has been developed, the following questions will be addressed: What sex-ratio patterns are observed in nature? What are the causes and what are the ecological correlates of the patterns? What sort of logic is required to explain each pattern? What general themes underlie the patterns? Some assumptions and extensions of the theory are discussed in the appendix.¹

METHODOLOGY

Before introducing the categories of sex-ratio patterns, we must have (i) definitions to describe the possible relationships between genotypes and their expressed phenotypes, (ii) a common understanding of proximate and ultimate causes, (iii) an appreciation of the interactions among the biological levels of life (e.g., genes, individuals, populations), and (iv) a language to describe how organisms allocate their energy and resources with respect to sex ratios (the language of economics). These four topics are discussed in turn.

GENOTYPES AND PHENOTYPES

The relationship between an individual's genotype and the sex ratio it produces (phenotype) is an important descriptor of sex-ratio patterns. Three possible relationships exist. (i) The sex ratio produced by an individual is *genetically determined*; i.e., a particular genotype expresses a

fixed sex ratio and the sex ratio differs (except for random variation) for different genotypes. (ii) The sex ratio produced by an individual is independent of its genotype, and becomes fixed before the individual becomes an adult. The sex ratio may be determined by the environment during pre-adult development, or it may be independent of the environment and therefore constant for all individuals in the population. A fixed response independent of the genotype is referred to as a *phenotypically canalized* response. (iii) The sex ratio produced by an individual is independent of its genotype, and may be variable during the adult stage. This is the situation in which individuals respond to ecological correlates of fitness (see next section), and is referred to as a *phenotypically plastic* response.

PROXIMATE AND ULTIMATE CAUSE

Since organisms are, for the most part, products of evolution by natural selection, most biologists agree that the *ultimate cause* of many behavioral and physiological attributes of an organism is the maximization of fitness. For example, the ultimate cause (or adaptive significance) of food acquisition is obtaining energy in order to increase the number of progeny successfully reared. We also know from our everyday experience that the behavior and physiology of an organism is often a response to its environment. Ecological cues (such as daylength, population density) that trigger particular genetic, physiological, developmental, or psychological mechanisms are referred to as *proximate cues*, and the mechanisms are referred to as *proximate causes*. These mechanisms, and the response to particular cues, usually evolve by the process of natural selection, and therefore often have an adaptive significance. Thus, in an evolutionary sense, the particular cues that are used by an organism to trigger behavioral and physiological responses ought to be those that have historically been correlates of successful (in terms of fitness) responses. Therefore, I often refer to proximate cues as *ecological correlates* of fitness. This usage stresses the key postulate—the proximate cues that organisms respond to only have meaning (in an evolutionary sense) because they are correlates of fitness (but see Williams 1966 and Gould and Vrba 1982 for relevant caveats).²

The specific proximate cues which are correlates of the sex ratio (and fitness, since sex ratio affects fitness) in different species follow no discernible pattern. For example, different species of mass-reared insects yield different sex ratios under similar ecological conditions (e.g., Hoelscher and Vinson 1971, Bouletreau 1976).³ In any species the particular proximate cues (e.g., host size, daylength) used are likely to be those that have historically correlated with a successful (in terms of fitness) sex-ratio pattern, and which the organism can assess. In other words, the correlates must contain information about which sex-ratio response confers the greatest fitness *and* the individual organism must be able to assess and respond to this information. Since any particular phylogeny is the result both of chance events and of the process of natural selection, just which proximate cues an organism uses are not predictable with certainty. Phenotypic plasticity of the sex ratio often does not exist (e.g., Williams 1979). This may be because (i) no ecological correlates of fitness with respect to sex ratios exist, or (ii) the correlates exist, but the organism has not evolved a response to these correlates.⁴

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GENETIC HIERARCHY

A variety of sex-ratio patterns occurs in nature (see Charnov 1982 for a review). Presenting these patterns one at a time leaves the impression that they are only vaguely related and due to a potpourri of causes and effects. In order to capture all of the patterns within a single cast, we must weave a broader net. The approach I will use is a hierarchical one with respect to genetic organization. Collections of genes make up chromosomes, a particular set of chromosomes forms an individual genome, an isolated group of individuals forms a subpopulation, and the collection of all subpopulations is the population gene pool. The similarities and differences among several sex-ratio patterns can be understood within such a hierarchical framework. For example, a 1:1 population sex ratio may be observed, while the sex ratio of the progeny of particular individuals may differ from 1:1 in a predictable manner (e.g., Trivers and Willard 1973). At what levels are the causes of these individual- and population-level patterns to be found? The patterns will be classified according to the level(s) at which natural selection is analyzed, and the level at which the sex-ratio effect is observed.

A common view, which will be extended here, is that the effects of natural selection can be observed as changes in gene frequencies within the population (e.g., Crow and Kimura 1970). Alleles are the lowest unit, or atom, of the hierarchy that we are interested in, and the population the highest level. The fitness of an allele within the population describes whether the frequency of that allele is increasing, decreasing, or remaining stable. A means of quantifying the fitness of an allele within the population is the number of replicates an allele produces divided by the average number of replicates produced by all alleles within the population, over one or more generations. The number of replicates produced can be called reproductive success (=RS), thus fitness of an allele within the population is

$$\text{fitness}(\text{allele/pop}) = \text{RS}(\text{allele/pop}) / \text{E}[\text{RS}(\text{allele/pop})].$$

This phrase is read as 'the fitness of a particular allele within the population is equal to the RS of that allele within the population divided by the expected (or average) RS of all alleles within the population gene pool.' This notation is used throughout the paper.⁵

The key to the hierarchical approach is realizing that *the fitness of an allele within the population can be partitioned into components*. For example, analyzing the success of an allele within the population can be carried out by first analyzing the success of that allele within a subpopulation, and then analyzing the success of the subpopulation within the entire population (Price 1970, 1972, Hamilton 1975; see fig. 1). In general, the fitness of a unit at a given level (e.g., chromosome) within a higher level (e.g., individual) is the number of alleles the unit contributes (RS) within a higher level divided by the average number of alleles contributed by all similar units [E(RS)] within the higher level. Fitness is measured over some time period, usually one or more generations. The interpretation of fitness values is simple. A fitness(allele/subpop) less than one occurs when a particular allele is decreasing in frequency within that subpopulation. A

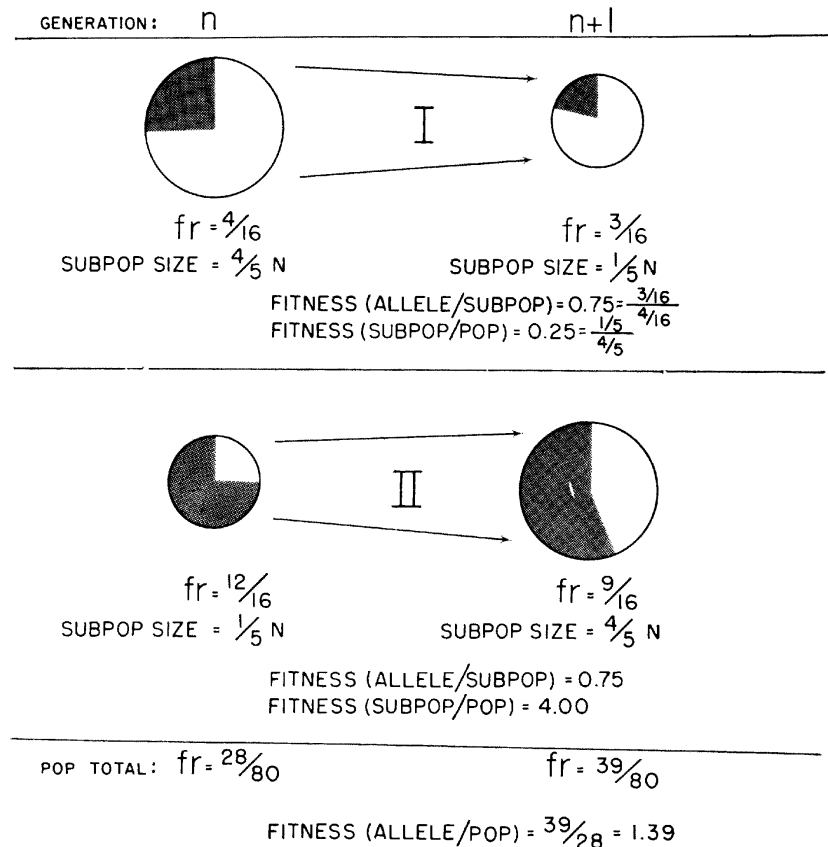


Fig. 1. A simplified example analyzing the components fitness(allele/subpop) and fitness(subpop/pop) for a population made up of two subpopulations (I and II). fr is the frequency of the allele, and N is total population size. The frequency of the allele decreases in both subpopulations, however for this example it is assumed that the subpopulation decreases in size when the frequency is less than 0.5, and the subpopulation increases in size when the frequency is greater than 0.5. Therefore, although the frequency of the allele decreases in both subpopulations, in this example the allele increases in frequency within the population. Although this example is artificial, the analysis of fitness components is a useful tool for understanding certain types of sex-ratio patterns discussed in this paper.

fitness greater than one means an increase in frequency over time, and a fitness equal to one occurs when the frequency is remaining stable. Further properties of the hierarchy will be developed while discussing particular patterns. The utility of the approach will be apparent when the patterns are viewed as a whole.

THE ECONOMICS OF LIFE

Over long periods of time natural selection favors behavioral and physio-

logical patterns of individual organisms (and entities at other levels of the hierarchy) that are consistent with the maximization of the individual's (or entity's) fitness within the population. The sort of behavior or physiology one is often interested in concerns the allocation of resources. For example, producing a particular ratio of sons and daughters, or a particular ratio of "male" and "female" flowers, is an allocation of resources into two alternatives—males and females. A useful language for discussing the allocation (=investment) of resources already exists—the language of economics. A particular investment pattern is said to have costs and benefits in terms of fitness. When resources are limiting, natural selection favors investment patterns that maximize the benefit/cost ratio (see Staddon 1980 for a review of this approach).

SEX-RATIO PATTERNS

Observed sex ratios often form patterns at several biological levels (e.g., individuals, populations), and these patterns are the result of both selection acting at various genetic levels and the relationship between genotypes and phenotypes. Each pattern is named according to an interesting phenomenon underlying the pattern. Table 1 lists the patterns in the order they will be presented, and shows how the patterns are classified according to the level(s) at which fitness components are analyzed, the level of the observed sex-ratio effect, and the genotype-phenotype relationship. These five patterns are chosen to illustrate the recurrent themes underlying most sex-ratio patterns. These themes are discussed in the conclusion. Sex ratios are given as 'males:females' or 'males/total'.³

(1) EQUAL INVESTMENT IN MALES AND FEMALES

The first question is why are the population sex ratios of many species about 1:1. To analyze this question the expected fitness (ind/pop) through a single son is compared to expected fitness(ind/pop) through a single daughter as the population sex ratio changes.^{6,7} Fitness of an individual in the population is $RS/E(RS)$. I will analyze fitness over 2 generations, where RS is the number of grandprogeny through a single male or female offspring divided by 4 (in diploid organisms each parent contributes 1/2 of the genome to an offspring, and thus a grandprogeny represents 1/4 of each grandparent). Consider the situation depicted in table 2. The expected fitness of an individual through a daughter added to this population is 2/3, through a son 4/3. In general, when males are rarer than females, expected fitness to an individual through a son is greater than through a daughter, and thus selection at the level of the individual within the population favors the production of males. Similarly, when females are rarer than males, expected fitness is greater through daughters, and the production of females is favored. Thus, the population sex ratio tends to stabilize at 1:1. However, this conclusion is only approximately correct, and a closer look reveals a more subtle pattern.

The situation where either one male or one female offspring was added to the population was just considered. However, producing a male or a female may require different amounts of resources. Perhaps females are provisioned with more food than males, as in many solitary bees and wasps

TABLE 1. THE FIVE SEX-RATIO PATTERNS DISCUSSED IN THIS PAPER. The patterns can be described by (i) an interesting biological phenomenon that underlies the observed pattern; (ii) the genetic levels of organization at which fitness components are analyzed for each pattern (the phrase 'individuals/pop' is read as 'individuals within the population', see text); (iii) the level at which the sex-ratio pattern is observed in nature; and (iv) the type of relationship between genotypes and phenotypes which is responsible for the observed phenotypic variability of the sex ratio produced by individual organisms. "Plastic", "canalized", and "genetically determined phenotypes" are defined in the text. The numbers in each row represent my opinion, based on the empirical evidence and theory summarized in the text, of the percent of each type of genotype-phenotype interaction for each pattern.

Phenomenon underlying pattern	Level(s) at which fitness is analyzed	Level of sex-ratio effect (= observed pattern)		Phenotypic variability		
		individuals/pop ind/pop	population individual	Plastic	Canalized	Genetic
1) Equal investment in the sexes	individuals/pop ind/pop		population individual	10%	65	25
2) Fitness of sexes varies with environment	ind/pop		deme	70	25	5
3) Population breeding structure	ind/deme deme/pop elements/ind ind/pop		individual	33	33	33
4) Extranuclear genetic elements	chromosomes/ind ind/pop		individual	0	0	99
5) Sex chromosome drive			individual	0	0	99

TABLE 2. EXPECTED FITNESS (IND/POP) THROUGH A SON VERSUS THROUGH A DAUGHTER. Sex ratio of the generation being added to is 1 male per 2 females, the population size is assumed to be large, mating random, and the average clutch per female is assumed to be 10. Thus, a (diploid) parent expects 10 grandchildren through a daughter for a RS of $10/4 = 2.5$. Since the sex ratio is 1:2, each male, on average, sires the progeny of 2 females, and thus is expected to sire 20 progeny. Expected RS through a son is therefore $20/4 = 5$. Expected fitness is average RS by a certain strategy (male v. female) divided by $E(RS)$ over all strategies, $(5+2.5)/2 = 3.75$. Thus, under these conditions, expected fitness through a son is greater than through a daughter.

Population sex ratio of generation being added to: 1:2 (males:females) Average clutch size/female = 10			
		expected RS of parent per individual offspring by sex	fitness = RS/E(RS)
if parent produces	1 male	$20/4 = 5$	$5/3.75 = 4/3$
	1 female	$10/4 = 2.5$	$2.5/3.75 = 2/3$

(Krombein 1967). In this case different amounts of resources are invested in male and female progeny.⁸ As an example suppose that the cost in terms of resources to produce one female is equal to the cost of producing 2 males (see table 3). The situation can be analyzed as before, but asking instead what are the expected fitnesses through sons and daughters *per unit investment* (rather than per offspring, as above). In this case, what is the fitness through 1 daughter versus 2 sons? The example in table 3 shows expected fitnesses when the population sex ratio is 1:1, males being

TABLE 3. EXPECTED FITNESS (IND/POP) PER UNIT INVESTMENT. The cost of producing 2 males and the cost of producing 1 female each represents one unit of investment. Since for a single unit of investment a parent may produce 2 males or 1 female, the fitness through 2 males must be compared to the fitness through 1 female. The remainder of the table is obtained as in table 2. With the population sex ratio 1:1, production of the cheaper sex, males, is favored.

Population sex ratio of generation being added to: 1:1 Cost of 2 males=1 unit of investment=cost of 1 female Average clutch size/female=10			
		expected RS of parent per unit investment	fitness = RS/E(RS)
parent produces	2 males	$20/4 = 5$	$5/3.75 = 4/3$
	1 female	$10/4 = 2.5$	$2.5/3.75 = 2/3$

favored over females. In general, the sex that is cheaper to produce will be more abundant than the more costly sex [this is Fisher's (1958) theory, reviewed by Charnov (1982)].

At what point does the sex ratio stabilize in this situation? Or, at what population sex ratio are the expected fitnesses through the 2 alternatives, 1 daughter or 2 sons, equal? Table 4 demonstrates that the fitnesses are equal when the population sex ratio equals the ratio of the number of males to females per unit investment. This is generally true, and since producing 1 female represents 1 unit of investment, and producing 2 males also represents 1 unit of investment, the population sex ratio stabilizes such that the investment ratio in male and female progeny is 1:1—i.e., where the sex ratio is 2:1 (fig 2).

Equal investment is a population-level attribute, therefore the problem of genotype-phenotype interaction is usually irrelevant. Only when individuals respond to a fluctuating population sex ratio is phenotypic plasticity important. An example of such a response is given in pattern (2) below.

An Example

Data consistent with the equal investment hypothesis have been collected for a few species of Hymenoptera (see Charnov 1982 for other examples). Solitary bees and wasps often seem to have sex ratios skewed toward the sex that is cheaper to produce (Trivers and Hare 1976, but see Alexander and Sherman 1977 for problems in interpretation). Two studies have been conducted that were designed to test the equal investment hypothesis on natural populations of the social wasp *Polistes*. Noonan (1978) studied *P. fuscatus*, and Metcalf (1980) examined *P. metricus* and *P. variatus* (table 5). These data represent several nests from each population. Wet weight of individuals was measured at eclosion, and was used to quantify invest-

TABLE 4. EXPECTED FITNESS (IND/POP) PER UNIT INVESTMENT; AS IN TABLE 3, EXCEPT THE POPULATION SEX RATIO IS 2:1. Expected number of grandchildren through one daughter is the average clutch, or 10. Since the population sex ratio is 2:1, each male, on average, sires one-half of a female's total progeny. Thus expected number of grandchildren through each male is 5, and through 2 males 10. Fitnesses per unit investment for the 2 alternatives, 2 males versus 1 female, are equal. Therefore the sex ratio stabilizes at 2 males per female, which is the ratio of the number of males to females produced per unit investment (see fig 2).

Population sex ratio of generation being added to: 2:1 Cost of 2 males=1 unit of investment=cost of 1 female Average clutch size/female=10			
		expected RS of parent per unit investment	fitness = RS/E(RS)
parent produces	2 males	$10/4 = 2.5$	$2.5/2.5 = 1$
	1 female	$10/4 = 2.5$	$2.5/2.5 = 1$

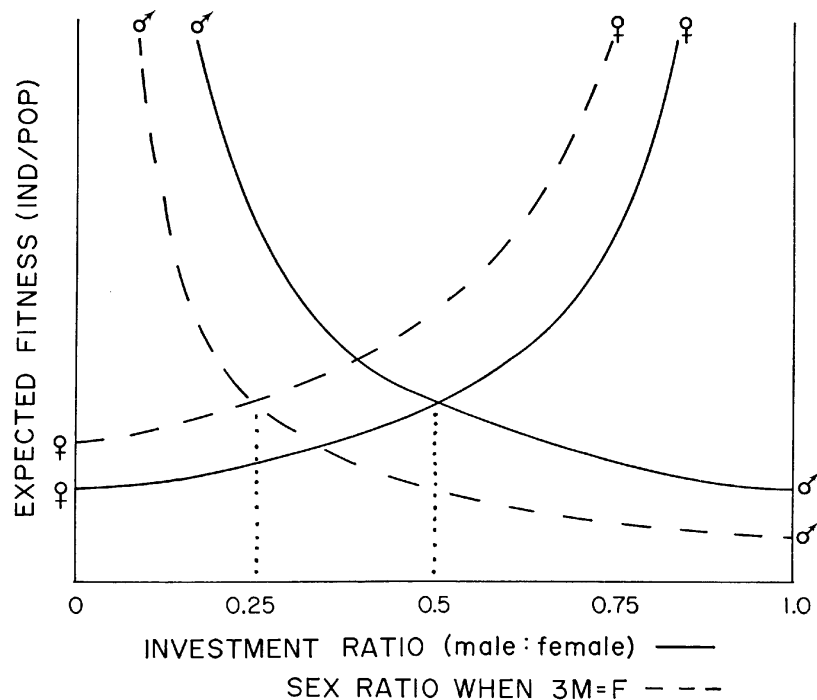


Fig. 2. Graphic representation of equal investment in the sexes theory. The solid curves represent the expected fitness of a single male and a single female added to a population as a function of the overall population investment ratio, where the population investment ratio is the amount of resources allocated to male progeny (or male function in hermaphrodites) to the amount allocated to female progeny (or function). For example, when the overall investment ratio is 0.25, males have a higher expected fitness than females, thus the investment ratio will increase. The expected fitnesses of males and females are equal only when the population investment ratio is 0.5. General proofs of this result can be found in Charnov (1982) and papers cited by him. The stable sex ratio will equal the number of males produced per unit investment to the number of females produced per unit investment. An intuitive argument can be constructed as follows. Let the number of males produced per unit investment be M , and the number of females per unit investment be F . Define the proportion of males (sex ratio) as x , the proportion of females as $1-x$, and the population size as N . Since the sex ratio is stable when the total investment in males equals the total investment in females, $Nx^*/M = N(1-x^*)/F$, where x^* is the stable sex ratio. Hence, $x^* = M/(M+F) = M:F$. So, when $3M = F$, $x^* = 0.25$ (represented by the dashed curves).

ment in males and females (Noonan 1978). In *P. fuscatus* the observed sex ratio is male biased and the investment ratio per individual is female biased, yielding a population investment ratio very close to 1:1. In *P. metricus* the opposite is observed, a female-biased sex ratio and a male-biased investment ratio per individual, also yielding a population invest-

TABLE 5. DATA SUGGESTING EQUAL INVESTMENT IN THE SEXES IN THREE SPECIES OF *Polistes*. *P. fuscatus* DATA FROM NOONAN (1978), *P. metricus* AND *P. variatus* FROM METCALF (1980). ALL RATIOS ARE MALE:FEMALE.

	<i>P. fuscatus</i>	<i>P. metricus</i>	<i>P. variatus</i>
Population sex ratio at eclosion	1.07:1.00	0.82:1.00	0.94:1.00
Sample size	819	17,701	3630
Wet-weight ratio at eclosion	0.94:1.00	1.23:1.00	1.01:1.00
Population investment ratio (weight ratio)•(sex ratio)	1.01:1.00	1.01:1.00	0.95:1.00

ment very nearly 1:1. See Noonan (1978) and Metcalf (1980) for statistical analyses and discussion of these data.

(2) RELATIVE FITNESS OF SEXES VARIES WITH ENVIRONMENT

Equal investment is a population-level pattern; however, empirical studies suggest that the investment ratio of individuals sometimes differs from 1:1 (see Charnov 1982). Why might such individual variation exist? If expected fitness (ind/pop) per unit investment through one sex is greater than through the other sex in a particular ecological setting, then the investment ratio in that setting is predicted to be biased toward the more successful sex (Trivers and Willard 1973, Charnov et. al. 1981). The hypothesis can be stated as follows: the ecological correlates of relative fitness of the sexes per unit investment are also correlates of individual-level investment ratios in the sexes (fig. 3). In other words, individuals can adjust their investment ratio according to local conditions in a phenotypically canalized or plastic manner. Notice that pattern (1) can be a special case of this hypothesis, where the population sex ratio is an ecological correlate of relative fitness of the sexes (Werren and Charnov 1978). As an example, in the mite *Marcrocheles* offspring sex ratio varies inversely with adult population sex ratio (Filipponi et. al. 1972, Filipponi and Petrelli 1975).

Example 1

Lariophagus distinguendus (Pteromalidae) is a hymenopteran parasite that lays one egg per host (Charnov et. al. 1981). In Hymenoptera unfertilized eggs give rise to males and fertilized eggs to females (haplodiploidy). Females are known to have some control over the sex of each egg laid, so the necessary mechanism for variation of the sex ratio exists at the level of individual phenotypes (i.e., plasticity) (Flanders 1956). Host sizes that a parasite encounters in nature often vary, and a positive correlation between host size and offspring size has been demonstrated for *Lariophagus* (Charnov et. al. 1981). The sex ratio is male biased on small hosts and female biased on large hosts (fig. 4, see also Clausen 1939). If it were known that small males have a greater fitness than small females, and large females have a greater fitness than large males (e.g., that size affects

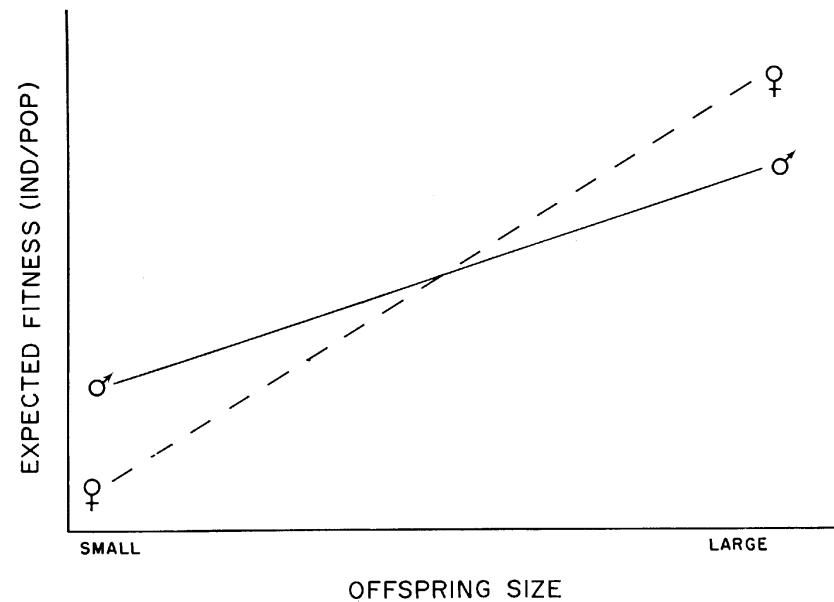


Fig. 3. Hypothetical relationship between expected fitness (ind/pop) for the alternatives male and female as a function of offspring size. In this situation, if an individual is small it has a higher expected fitness as a male, and if large a higher expected fitness as a female. Offspring size often has correlates; in example 1 (see text) host size is a positive correlate of offspring size; in example 2 parasite density is a negative correlate of offspring size.

number of eggs laid by females more drastically than number of successful matings by males), then the hypothesis that correlates of relative fitness of the sexes (e.g., host size) are also correlates of the sex ratio would be supported by this study. Empirical evidence of relative fitness shifts of the sexes with size has not been obtained for *Lariophagus* (measuring fitness is difficult), thus direct support of the hypothesis cannot be claimed. However, likely correlates of individual fitness (fecundity for females, age at death for males) as related to size were measured for *Lariophagus* and *Heterospilis prosopoidis* (Braconidae), and the data suggest that changes in fitness with size are greater for females than for males (Charnov et. al. 1981). This is consistent with the observed sex-ratio response.

What ecological cues might *Lariophagus* use to determine whether a host is large or small? A logical hypothesis is that the insect assesses the size of the host (or a correlate of size, such as tunnel diameter of a burrowing host) relative to other hosts encountered, and adjusts the sex ratio of its offspring according to the above prediction. Charnov et. al. (1981) conducted an experiment with *Lariophagus* to test this hypothesis (fig. 4). A particular host size was presented alone, with smaller hosts, and with larger hosts. The sex-ratio shifts observed support the hypothesis that relative host size is assessed by these wasps (see fig. 4 for details).

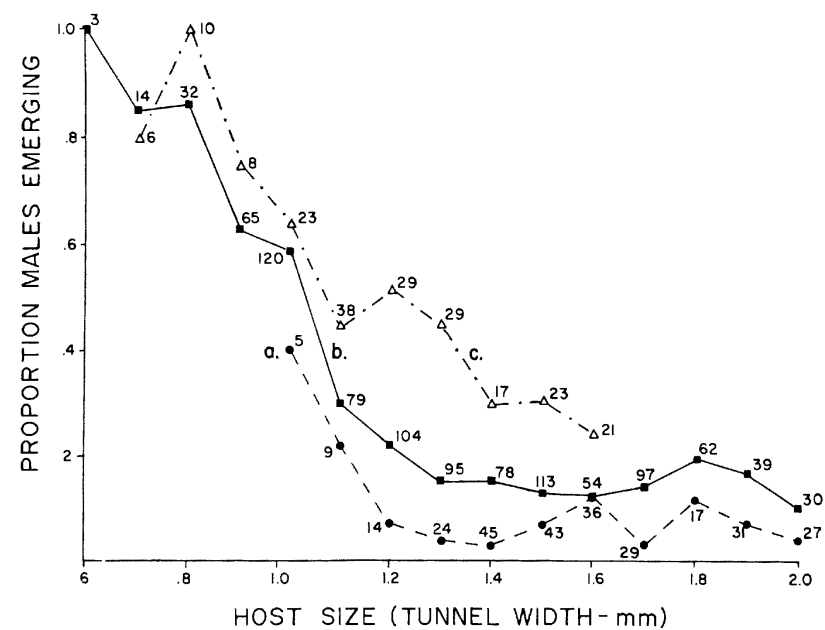


Fig. 4. Correlation of relative host size and sex ratio in *Lariophagus*. Curve *b* represents the sex ratio produced by female wasps presented sequentially with 20 hosts of a single size. Curves *a* and *c* represent the sex ratio where the wasps were presented with an alternating sequence of two host sizes, the hosts differing by 0.4 mm. Curve *c* represents the sex ratio when the host was the smaller of two alternating sizes, and *a* where the host was the larger of two alternating sizes. For example, hosts of 1.4 mm were offered alone and gave a sex ratio 15% males (curve *b*). When 1.4 mm hosts were offered alternately with 1.8 mm hosts, the 1.4 mm hosts yielded a sex ratio of 30% males (curve *c*). When 1.4 mm hosts were offered alternately with 1.0 mm hosts, the 1.4 mm hosts yielded a sex ratio of 2% males (curve *a*). Host size was determined by measuring tunnel diameter of the burrowing host. The number of emerging wasps is given for each datum point. From Charnov et. al. (1981).

Example 2

Some parasites lay several eggs on a single host. Often as the number of developing parasites on a host increases, the size of each emerging parasite decreases due to competition for food among the larvae. In other words, initial parasite density is a negative correlate of eventual parasite size [e.g., *Hydromeris contorta* (Mermithidae), Charnov (1982) citing Johnson (1955, not seen)]. The sex that has the greater increase in expected fitness with increasing size should be favored in hosts with few parasites (large individual parasites emerging), the other sex in crowded hosts (small individuals) (see fig. 3). The nematode *Hydromeris* provides a good example. The sex of mermithid nematodes is determined after birth, while the nematode is still immature, and environmental conditions have been shown to be correlates of sex (i.e., sex is a phenotypically canalized trait) (Christie

1929, Anya 1976). If a greater change in expected fitness with size for females is assumed, as in example 1, the prediction is that females will be favored in hosts with few parasites, males in hosts with many parasites. Figure 5 shows the number of nematodes on a single host and the sex ratio. The data are consistent with the prediction that density is a correlate of sex. Given the *assumption* that relative fitness of the sexes differs as a function of density (or size) as in figure 3, then correlates of relative fitness of the sexes are correlates of the sex ratio. Figure 6 shows a hypothetical relationship between an ecological correlate of fitness and the expression of a sex-ratio pattern.

(3) POPULATION BREEDING STRUCTURE—GROUP-LEVEL EFFECTS

When the population is divided into small, isolated groups (=demes), the expected fitness of an individual within a deme (ind/deme) is greatest according to equal investment in the sexes (due to selection within demes, or intrademic selection), and the fitness of the deme within the population (deme/pop) is greatest when the investment ratio of the deme is extremely female biased (due to selection between demes, or interademic selection) (paternal care assumed unimportant). The fitness of the individual within the population as a function of its investment ratio in the sexes is a compromise of the conflict between the two fitness components, (ind/deme) and (deme/pop). The resulting sex-ratio pattern due to conflicts among

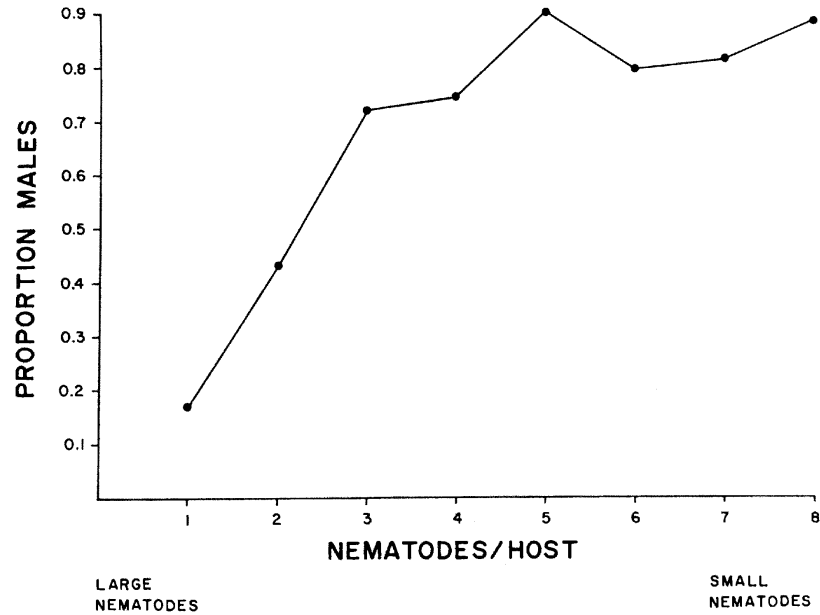


Fig. 5. Correlation of number of nematodes per host and sex ratio. Few nematodes per host corresponded to relatively large emerging individuals, and many nematodes per host corresponded to relatively small individuals. Data of Johnson (1955, not seen) for the mermithid nematode *Hydromeris*, redrawn from Charnov (1982).

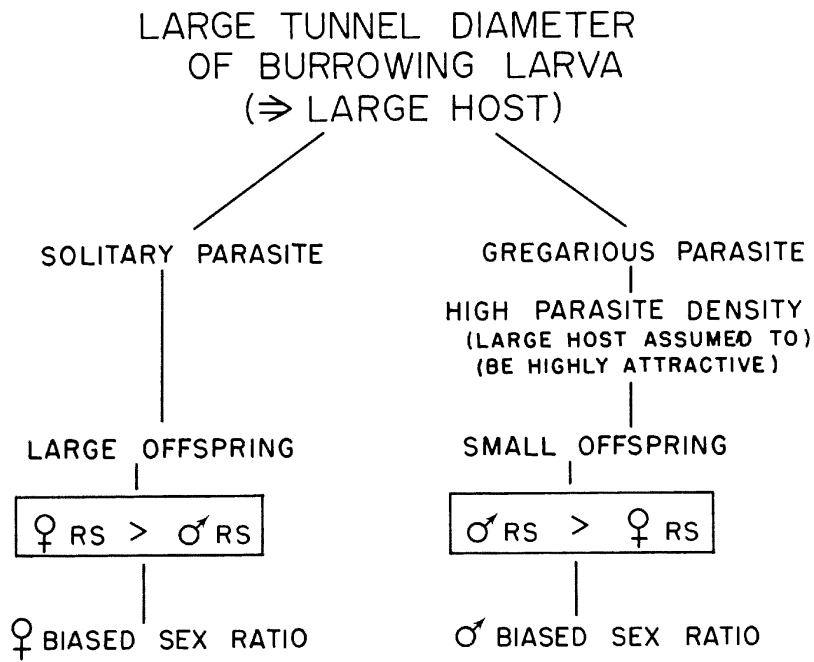


Fig. 6. Link between proximate cues and observed sex-ratio patterns. In order to determine the size of a host, parasites often use ecological correlates of host size such as daylength, ambient temperature, chemical properties of the host, and as in this example, tunnel diameter of a burrowing larva. The same proximate cue, large tunnel diameter which implies a large host, may signal high fitness of a male-biased or female-biased individual-level sex ratio to different parasite species according to their particular natural histories. The two pathways are similar to examples 1 and 2 of pattern (2) in the text, which assume the fitness-size relationship shown in figure 3.

levels of the hierarchy is surprising, and this pattern allows us a view of a type of hierarchical organization rarely observed.

It is generally thought that the conditions under which interademic selection will occur are very rare, and this has influenced many biologists' perceptions of nature. Indeed, in the vast majority of cases interademic selection is likely to be trivial (Williams 1966, but see Wilson 1980). But the situations where such selection may be relevant do exist and include extraordinary life styles (Hamilton 1967, 1978, 1979). In order to illustrate this difficult problem I will discuss a particular population breeding structure that is common among many groups of parasitic Hymenoptera and mites (although probably rare among organisms in general). Consider an isolated resource patch that is ephemeral in time and space, such as a larval host. One to a few mated females arrive (=foundresses) and lay eggs. The eggs develop in this patch, and eventually the adults emerge. All matings occur just after eclosion within the isolated patch. In addition, for this example we assume that the resource patch is depleted after only one

generation. Therefore the already-mated females must disperse to find and colonize new patches, groups of these dispersing females settling on a new patch in a random fashion.

Numerical Example

In this example individual fitness within the population is partitioned into individual fitness within a deme and deme fitness within the population.⁶ Individual fitness within the deme is analyzed as in pattern (1), taking into account the very small size of the deme. [An assumption of pattern (1) is that mating occurs randomly within the population. Here mating occurs randomly within the deme, and no mating occurs between members of different demes, so fitness(ind/deme) is analyzed instead of fitness(ind/pop)]. As in pattern (1), RS is analyzed over 2 generations. The method of determining fitnesses of individuals within a deme is demonstrated in table 6, for 2 foundresses forming a deme. From table 6 note that foundress I has the same sex ratio in 2 cases, 1:1, but her fitness changes according to the sex ratio adopted by foundress II. Since the fitness of each foundress depends on the sex ratio of the other foundress, the situation has a game-like quality (Hamilton 1967). Such an approach can be expanded to include all combinations of sex ratios by foundresses I and II. As Hamilton (1967) showed for 2 foundresses per deme, and Colwell (1981) demonstrated for arbitrary number of foundresses per deme when analyzing only the fitness component (ind/deme), the overall investment ratio within a deme stabilizes at 1:1. Generally, the overall investment ratio within the next higher stage of the hierarchy in which the mating occurs at random stabilizes at 1:1. [In pattern (1), the population is the next stage above individuals in which mating is random.] As we proceed to the next component, demes within a population, a conflict with the individual level emerges (Hamilton 1967, 1979, Colwell 1981, Wilson and Colwell 1981).

For the particular form of breeding structure being discussed, the fitness of a deme within the population is positively correlated with number of colonizing females produced. This conclusion follows from the fact that a deme's contribution to the next generation depends on the number of colonizers it produces. Therefore, selection among demes within the population favors demes with the most female-biased sex ratio possible such that there are just enough males to mate with all of the females. The result of conflicting selection pressures between individuals within a deme and demes within the population is a stable sex-ratio pattern intermediate between the extremes.

Hamilton (1967, 1979), and later Colwell (1981) and Wilson and Colwell (1981) arrived at a general solution for the stable sex ratio in diploid organisms, where clutch sizes of the foundresses are equal. The solution is

$$r^* = 1/2[(n-1)/n] \quad n=2,3,4,\dots$$

where r^* is the sex ratio (males/total) of a deme with n foundresses.⁹ The effect is a predicted sex-ratio pattern at the level of the deme. A similar solution for the haplodiploid case has also been found (Hamilton 1979, see fig. 7).

TABLE 6. THE FITNESS (IND/DEME) OF 2 FOUNDRASSES FORMING A DEME AS A FUNCTION OF THE SEX RATIO OF THEIR CLUTCHES. The technique of calculating fitness (ind/deme) is shown in (i) for foundress I using the methods developed previously. Notice that in (i) and (ii) the sex ratio of foundress I is 4:4, but her fitness changes as the sex ratio of foundress II changes. Also notice that the number of colonizing females produced by the deme decreases in (ii), and thus the fitness (deme/pop) has decreased. Mating within the deme is assumed to be random, and the organism is diploid.

Foundress I			Foundress II			Total RS (ind/deme)	Average fitness (ind/deme)	Sex ratio of clutch	Average RS (ind/deme)	Total RS (ind/deme)	Ave. fitness (ind/deme)
Sex ratio of clutch	Average RS (ind/deme)	Average RS (ind/deme)	Average RS (ind/deme)	Sex ratio of clutch	Average RS (ind/deme)						
(i) 4 males 4 females	$4(80/6)/4 = 80/6$ $4(8)/4 = 8$	21 1/3	$\frac{21 \ 1/3}{(21 \ 1/3 + 18 \ 2/3)/2} = 1.07$	2 males 6 females	40/6 12	21 1/3	18 2/3	80/6 progeny 4 males 4 females	1.07	18 2/3	.93
(ii) 4 males 4 females	8 8	16	1	4 males 4 females	8 8	16	1	8 8	1	16	1

deme sex ratio = 6 males:10 females
Each male sires (on average) 10/6 of a clutch of 8 = 80/6 progeny
deme sex ratio = 8 males:8 females

Empirical Support

Females of the hymenopteran parasite *Nasonia vitripennis* (Pteromalidae) lay up to 50 eggs on each dipteran host puparium encountered, and 2 or more females often lay eggs on a single host (natural history reviewed by Whiting 1967). The adult males usually emerge first, and stay on or by the host waiting for the females to emerge. The males lack the ability to fly, and all mating occurs near the host. The mated females then fly off to find a fresh host to parasitize. Since these are hymenopteran parasites, they are haplodiploid, and the females have the potential to control the sex ratio of their clutches (phenotypically plastic sex ratio). These parasites have the sort of breeding structure outlined above, and Werren (1980a, 1980b, in press) examined the sex ratio to see if it conformed to Hamilton's (1979) prediction for haplodiploid organisms. The data he obtained seem to agree with the predicted sex ratio (fig. 7).

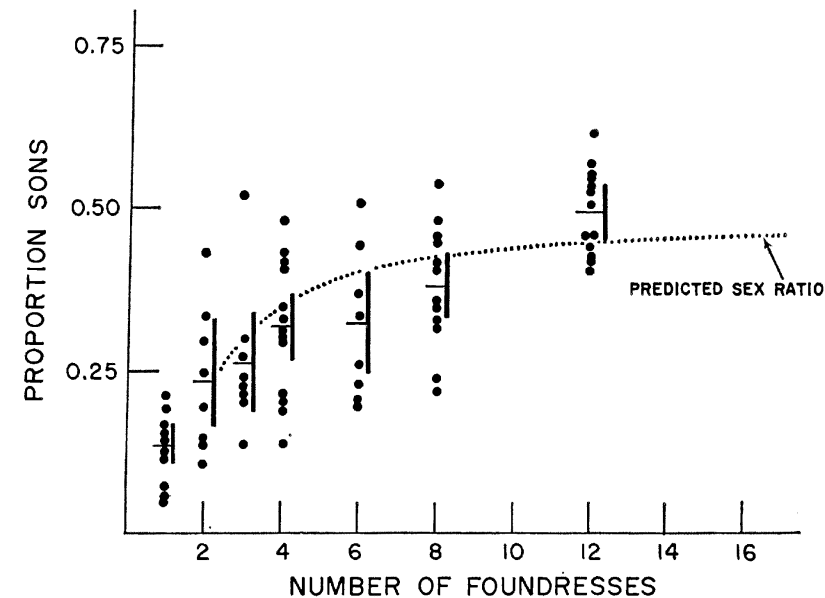


Fig. 7. Correlation of sex ratio and number of foundresses per host for *Nasonia* reared on *Sarcophaga bullata* in the laboratory. Mean \pm 1.96 s. e. are shown for each level. The dotted line represents the predicted sex ratio for haplodiploid organisms with the breeding structure as described in the text, where $r^* = 1/2[(n-1)/n][(4n-2)/(4n-1)]$ (Hamilton 1979, Werren in press). Notice that this formula only differs from the one in the text for diploid organisms by the factor $[(4n-2)/(4n-1)]$. Haplodiploidy can be treated as a case of sex-linked sex-determination control, where females are XX and males are XO. This sex-linked sex determination creates an additional female-biasing selection pressure as discussed in pattern (5). Data and experimental details in Werren (1980b).

More Levels in the Hierarchy?

Although the *Nasonia* data fit the prediction fairly well, other parasitic wasps have a sex ratio distinctly more female biased than predicted [e.g., *Melittobia* (Eulophidae), fig wasps (Agaonidae), see Hamilton 1967, 1979 for a review]. The highly skewed sex ratios among these species remain a mystery. I suggest that adding further levels to a hierarchical analysis between the level of demes and the population may provide a clue.

Consider the natural history of fig wasps as an example (see Wiebes 1979, Janzen 1979, and Hamilton 1979 for reviews of fig-wasp biology). Fig trees bear inflorescences (=figs) that are hollow receptacles with hundreds of staminate and pistillate flowers opening into a sealed, central cavity. A few female wasps (=foundresses) enter the fig through a narrow passage, and once inside can never leave. They pollinate some of the pistillate flowers with pollen carried from the fig from which they emerged, lay an egg in each ovary of the remaining pistillate flowers, and then die. Each larva develops by eating the tissue of a single flower. About one month later the next generation of wingless males emerges and mates with the not-yet-closed females within the still-sealed fig. Therefore, each individual fig constitutes a deme (as described in the previous discussion) that forms from a pool of colonizing females and disintegrates after one generation. Hamilton's (1979) prediction about the sex ratio is relevant (given in fig 7), since the fig wasps appear to have the sort of breeding structure assumed in that model. Data indicate that the sex ratio of some fig-wasp species does become less female biased with increasing number of foundresses, however at a rate much slower than predicted (Frank in prep., fig. 8).

Let us return to the natural history of fig wasps for a moment. The fig-wasp species for which data are reported in figure 8 occupy fig trees that occur in the Caribbean, throughout the Florida Keys archipelago, and in the extreme southern portion of the Florida peninsula. The host-tree's distribution on the peninsula is quite patchy, and occasional frosts cause local extinctions followed by recolonization (pers. obs.). Each fig tree bears thousands of figs per crop, and several hundred wasps are reared from each fig. Thus a single tree produces between 10 thousand and 10 million fig wasps per crop. The emerging wasps fly and/or are blown to a new tree bearing figs at a stage receptive to wasps. Since the trees occur in patches isolated by long distances over land or water, the emerging wasps will probably most often enter receptive figs within their own patch. Occasionally a few wasps will be blown from one patch to a neighboring patch. Thus each patch of trees represents a distinct subpopulation of wasps. Fitness(ind/pop) can be partitioned into individuals within a deme (= a fig), demes within a subpopulation (= an isolated patch of trees), and subpopulations within the population. The sex ratio that maximizes fitness(ind/subpop) is now represented by the formula stated in figure 7, since the subpopulation now satisfies the assumptions of that model [i.e., a two-level analysis of fitness(ind/deme) and fitness(deme/subpop)]. The contribution of a subpopulation to the whole population is dependent on the number of colonizing females produced by that subpopulation, creating an additional female-biasing selection pressure. This population structure may cause the stable sex ratio at the level of the deme to be more female biased

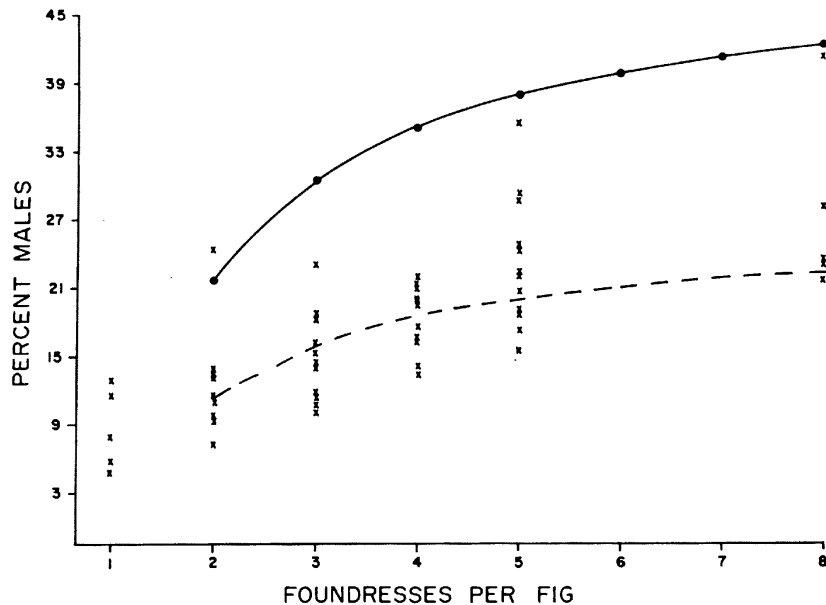


Fig. 8. Correlation of sex ratio and number of foundresses per fig for the fig wasp *Pegoscapus* sp. reared from figs of *Ficus citrifolia*. Each datum point represents the sex ratio of the wasps from a single fig; the average number of wasps per fig is 207. The solid curve is the predicted sex ratio for haplodiploid organisms as in figure 7, which assumes random mating within a deme (=fig) and no differentiation among subpopulations. The dashed curve is the predicted sex ratio assuming some assortative mating and/or differentiation among subpopulations [$P_{\text{ind/fig}} \cdot P_{\text{subpop/pop}} = 0.52$; see Wright 1969, Frank in prep for notation]. Data, statistical analyses, and experimental details in Frank (in prep).

than in a population structure such as *Nasonia*'s, where a significant partition of demes/pop may not exist.¹⁰

This explanation of fig-wasp sex ratios is hypothetical, and will require further testing on several fig-wasp species over a variety of host-tree distributions (i.e., continuous distributions with poorly defined subpopulations to isolated distributions with well-defined subpopulations). An advantage of this approach is that it is amenable to simple modelling, which will reveal exactly which measurements of isolation are important. The hypothesis is that as the distribution of a species becomes relatively (among all species of fig wasps) more isolated, the sex ratio at a given number of foundresses per fig will be relatively low for that species. Also, within any species the sex ratio will increase with increasing number of foundresses per fig (i.e., phenotypic plasticity is expected, see next section).

Ecological Correlates of Breeding Structure and Sex Ratio

As discussed in pattern (2), the best sex ratio for an individual mother may change with ecological parameters—in this case it changes with the

number of foundresses in a patch. Therefore, a phenotypically plastic response to ecological correlates of breeding structure is possible. Consider the natural history of *Nasonia* outlined above. The females search for puparia and lay up to 50 eggs per host. Each female probably parasitizes several hosts. When a female lays eggs on a host she may be the first parasite, or the host may already have been parasitized by one or more foundresses. The sex ratio that maximizes her fitness (ind/pop) depends on how many other foundresses lay eggs on that host (Werren in press). What are the ecological correlates of foundress number that a female can assess? It has been demonstrated that a female can differentiate between parasitized and unparasitized hosts (Wylie 1965), and adjust her sex ratio accordingly (Holmes 1972, Werren 1980a). Such discrimination may be a chemical cue (Holmes 1972). Because two females parasitizing the same host may never see each other, chemical cues are likely candidates for being assessed as ecological correlates of fitness. Females may also respond to high densities of hosts or conspecifics assessed visually. What about fig wasps? The foundresses must enter and oviposit within a tiny, sealed cavity from which they never leave, and it seems likely that the foundresses adjust their sex ratio according to an assessment of number of other foundresses. This may be accomplished either tactilely, or by reacting to chemical cues that are correlated with number of foundresses.¹¹

(4) EXTRANUCLEAR GENETIC ELEMENTS AFFECTING SEX RATIOS

In the patterns examined so far, fitness of levels below the individual (=subgenomic) within the population have been assumed to be equivalent to fitness of individuals (=genomes) within the population. Such an assumption is legitimate when a phenotypic attribute benefits all subgenomic elements equally.^{6,10} For example, changes in expected fitness (chromosome/ind) as a function of sex ratio are the same for all autosomal chromosomes of a diploid individual. When, depending on some phenotypic attribute, the fitness of a subgenomic element within the population differs from the fitness of individuals within the population that carry these elements, a conflict arises (see Birky 1978, Cosmides and Tooby 1981 for reviews). These subgenomic elements may include bacterial and viral infections, cellular organelles such as mitochondria that contain genetic material, and a variety of other extranuclear cellular symbionts (reviewed by Margulis 1981). The fitness (element/pop) often differs from the fitness (ind/pop) in which the element resides as a function of the sex ratio of the individual's progeny (i.e., the sex ratio is a phenotypic attribute of the individual). Figure 9a represents a case where such a conflict is likely to exist. In this case a bacterial infection is passed from generation to generation through the eggs of a female. The infection is not passed through sperm. Therefore, the mode of inheritance of the infection is from mother to daughter, or matrilineal. The expected fitness of the infection within an individual host's progeny clearly increases as the sex ratio produced by its host becomes more female biased. The fitness of the individual host within the population with respect to sex ratio is often greatest according to patterns (1), (2), and (3). A conflict exists between the infection and much of the genome (= intragenomic conflict).¹² Several cases of extranuclear genetic elements affecting the sex ratio have been documented (i.e., the sex ratio of the host is genetically determined by the element; for example, Sakaguchi and

Poulson 1963, Johnson 1977, Werren et. al. 1981, Skinner 1982, Bryant et. al. 1982, reviewed by Uyenoyama and Feldman 1978). Male sterility in maize is probably controlled by mitochondrial genes, and therefore is another case of this pattern (Rhoades 1933, Levings and Pring 1976).

Several questions about sex-ratio biases controlled by subgenomic elements need to be addressed. When they exist, do they spread through the entire population? If not, what is their frequency, and why is this frequency observed? Since the interests (in terms of fitness) of the sex-ratio biasing elements conflict with the interests of other parts of the genome, do forms of *suppression* exist (i.e. are there alleles that can reduce or remove the effect of the sex-ratio bias even when the element is present)? The effect of the sex-ratio bias due to these elements is expressed at the level of the individual that has such an element, but what about the population sex ratio? To determine how common a subgenomic element affecting the sex ratio will be within a population, fitness (element/pop) must be analyzed. Applying the hierarchical method of analyzing fitness, fitness (element/pop) is a function of both fitness (element/ind) and fitness (ind/pop) harboring these elements. This partitioning of fitness into components highlights biologically important phenomena such as suppression (intragenomic conflict)

and leads to predictions of observed sex-ratio patterns at the individual and population level, as we shall see below.

Empirical Evidence

The existence of extranuclear particles causing a sex-ratio bias is well documented (see above references); however, clear answers to the above questions do not exist for any single population. Here I briefly summarize some observations on the terrestrial isopod *Armadillidium vulgare* (Howard 1942, 1958), and a few other species of isopods. (i) Individual females consistently produce either female-biased broods (thelygeny), 1:1 broods (amphogeny), or male-biased broods (arrhenogeny). (ii) The brood sizes are the same for all sex ratios, suggesting there is no reduction in fecundity associated with sex-ratio traits, and ruling out sex-biased mortality as an explanation. (iii) A male parent has no effect on the brood's sex ratio. (iv) Other loci (e. g., eye color) segregate in a Mendelian fashion, ruling out parthenogenesis. (v) Thelygenic females have a particle in the ovarian tissue absent from amphogenic females. Ovarial tissue grafted from thelygenic females to amphogenic females converted the amphogenic females to thelygeny. The nature of arrhenogeny is not entirely understood. Arrhenogeny may be a different response to the same particle by genetically different females, different particles, or some other mechanism (Juchault and Legrand 1970, Legrand and Juchault 1972). (vi) The mode of sex determination is unknown, although suggestions have been made (Bacci 1965). (vii) The inheritance of sex-ratio traits is somewhat confusing. Howard (1958) reviewed data of his own and other workers, and concluded that (a) in *Trichoniscus provisorius* and *A. vulgare* the typical pattern is amphogenic females giving rise to amphogenic daughters, and thelygenic females giving rise to both thelygenic and arrhenogenic daughters. The effect of a male parent's history (i.e., from an amphogenic or arrhenogenic brood) has not been studied, and (b) in *Cylisticus convexus* it has been demonstrated that the male parent's history has an effect on the sex-ratio characteristics of his daughters. Both Howard (1958) and Johnson (1977) have suggested that the inheritance pattern may be due to an interaction between extranuclear elements and nuclear genes. This hypothesis seems reasonable with respect to both the observations mentioned above and the theory of intragenomic conflict over control of the sex ratio as outlined above. (viii) The sex-ratio theory outlined in this paper predicts equal investment in the sexes over the entire population, in spite of individual-level biases [since when a sex is rare, it has a higher expected fitness than the more abundant sex, see pattern (1)]. Nuclear genes interacting with extranuclear elements could provide a genetic mechanism for variability of population-investment ratios, allowing equal investment in spite of sex-ratio distortion in infected individuals (Johnson 1977). The observed population sex ratios are difficult to interpret due to small sample size, method of sampling, and the fact that the particle may spread in an infectious manner under laboratory rearing conditions. Due to the nature of the sex-ratio trait, the accuracy of an estimate probably depends more on the number of broods reared than on total number of offspring counted, and the variance of the population sex ratio is likely to be high. Johnson (1977), studying *Venezillo evergladensis*, reports the largest random sample that I could find in the literature. For the life-time broods of 87 females, the

MATRILINEAL ELEMENT PEDIGREE

Y CHROMOSOME PEDIGREE

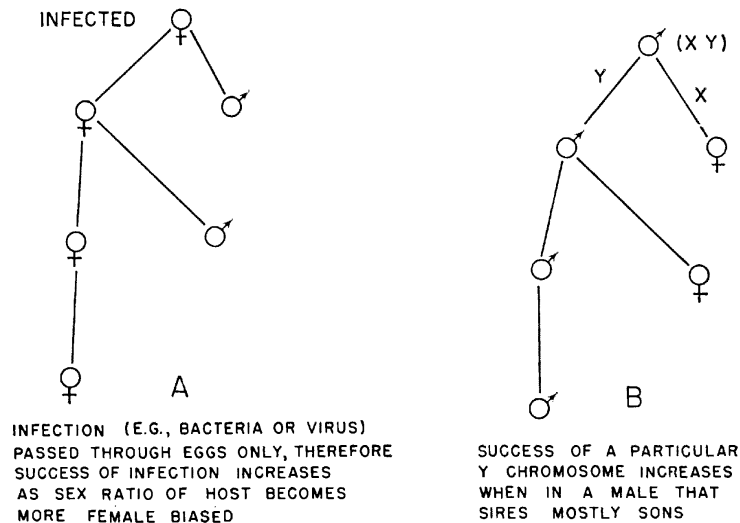


Fig. 9. Uniparental inheritance pedigrees. (a) Matrilineal inheritance of an extranuclear (cytoplasmic) genetic element. The element is passed from mother daughter through the egg—the element may be passed to a son through the egg, but is not passed through sperm, so males are a dead end in the pedigree. Cytoplasmic elements, including mitochondria, chloroplasts, and bacterial and viral symbionts are almost always inherited matrilineally. (b) Patrilineal inheritance of a Y chromosome. Males contribute a Y chromosome to sons and an X chromosome to daughters. Since females are XX, daughters represent a dead end in the pedigree of a Y chromosome.

overall sex ratio was 1311:1310 (males:females), with what appear to be 6 thelygenic and 6 arrhenogenic females. Johnson cites Vandel as having found 42 arrhenogenics and 32 thelygenics in a sample of 88 *T. provisorius*, and 4 of each type in *A. vulgare* in a sample of 15. Johnson (unpub. data) found 4 thelygenics and 5 arrhenogenics in a random sample of 30 gravid *A. vulgare* females collected from the wild, with an overall sex ratio of 1085:1089 (males:females). Thus, it seems likely that population sex ratios at birth do not differ greatly from 1:1 (but see Howard 1942).¹⁶

(5) MEIOTIC DRIVE AND SEX RATIOS

When considering the effects of selection, the level of the individual seems the most natural to contemplate; it is individuals that we can see actively reproducing and competing. It is more difficult to imagine subgenomic elements competing for their own reproductive success, as in the case of the matrilineal infections discussed in pattern (4). Consider the case of a diploid organism. Each chromosome is paired with a homologue, and only one of the two paired chromosomes is found in any single gamete. It is often assumed that each chromosome of the pair has an equal chance of appearing in any gamete due to random assortment. But, a chromosome that can bias assortment such that it is in more than half of the successful gametes (referred to "as a driving chromosome", or as "a case of meiotic drive", reviewed by Zimmering et. al. 1970, White 1973, Crow 1979, see also Bryant et. al. 1982) will increase in relative frequency within the individual's progeny that it resides in, since the expected RS of a chromosome/ind is one-half the number of progeny by that individual. Therefore whether a driving chromosome will spread in the population depends on (i) the fitness of (ind/pop) with such a chromosome, and (ii) the ability of other elements in the genome to suppress a driving chromosome.¹³

The sex chromosomes of *Drosophila* are designated XY for males and XX for females. The male's contribution of an X or Y chromosome determines the sex of his progeny (see fig. 9b).¹⁴ Thus, a driving X chromosome in a male causes a female-biased sex ratio of his progeny; similarly, a driving Y chromosome causes a male bias. What about the fitness of an individual within the population bearing a driving sex chromosome? For example, consider a male with a driving X chromosome. Most of his progeny will be females. The sex ratio of his progeny that would maximize his fitness within the population often differs from the female-biased sex ratio caused by the driving X chromosome [according to patterns (1), (2), and (3)]. Thus, although a driving chromosome may increase its own fitness (chrom/ind) within an individual above one, it may decrease the fitness of the entire genome within the population (including itself), and may or may not have a fitness (chrom/pop) greater than one.¹² If a driving chromosome decreases fitness (ind/pop), selection favors other elements of the genome that can suppress the driving chromosome. Thus the apparently random assortment often observed in meiosis may actually be a competitive stalemate of subgenomic elements (Alexander and Borgia 1978); for example, a Y chromosome with the ability to suppress the driving behavior of an X may yield a 1:1 sex ratio. Driving X chromosomes are widespread in wild populations of some *Drosophila* species (reviewed by Zimmering et. al. 1970, White 1973); an interesting sex-ratio-distortion condition in *D.*

paramelancia was studied by Stalker (1961). There are two types of driving X's, called 'Northern' and 'Southern', corresponding to locations of the populations where they are most commonly found, although some overlap exists. Northern X's have their sex-ratio-distortion effect suppressed by Southern Y's (i.e., males with Northern X, Southern Y do not produce excessively female-biased broods). Southern X's are not suppressed by any type of Y's. In this case, no reduction in fecundity was reported for males with a driving X chromosome and a female-biased progeny sex ratio. Other cases of drive with reduced fitness (ind/pop), or suppression due to other chromosomes, have been documented (White 1973, Crow 1979).

Hartl (1977) reviews potential applications of meiotic drive for breeding and for population control of insect pests. Hamilton (1967) pointed out that the introduction of a driving Y chromosome might be an effective form of control in species with XY (heterogametic) males, but also noted a possible complication due to suppression of driving Y's by other elements of the genome, and suggested methods for overcoming this difficulty. Thus meiotic drive and extranuclear elements may be of practical use, and will certainly help explain some of the variability of sex ratios observed in nature.

CONCLUSION

Five sex-ratio patterns were discussed in this paper (table 1).¹⁵ Two general themes of sex-ratio patterns emerge—(i) ecological correlates of fitness and (ii) conflicts within and among the levels of selection. These two themes are not mutually exclusive, and both affect and are affected by the mode of sex determination. (i) When relative fitness of the sexes per unit investment varies according to some ecological correlate, then local sex ratios are expected to vary according to that ecological correlate. There are at least two complications. First, not only must ecological correlates of relative fitness exist, but whatever controls the sex ratio must also, in some sense, be able to respond to the levels of that correlate (by a proximate mechanism causing phenotypic plasticity or canalization which responds to proximate cues such as temperature, daylength, etc.). This leads to the second complication. There are many types of sex-determination systems, and the sex-determination system may constrain the nature of the genotype-phenotype relationship. The sex-determination system may also constrain which genetic entities can control the sex ratio. (ii) The second general theme is conflict within and among the levels of selection. A particular subset of the genome (e.g., a chromosome, or an extranuclear element) may maximize its own fitness according to a sex ratio different from the sex ratio that maximizes the fitness of other subsets of the genome [and hence different from fitness (ind/pop)]. The phenotype of an individual depends on the resolution of this inherent conflict (which may be within or among levels of the hierarchy), and this resolution will depend on whatever element(s) can control the expressed sex ratio. Thus, there is much overlap among genotype-phenotype interactions, ecological correlates of fitness, the mode of sex determination, and the levels of selection.

A hierarchical approach to sex ratios has at least two advantages. First, it provides insight into the fitness components which must be analyzed

when considering particular problems, and this highlights biologically interesting phenomena such as intragenomic conflict and population breeding structure. Second, a hierarchical approach provides a framework for specifying the level at which a sex-ratio pattern will be observed, such as individuals infected with a sex-ratio distorting bacteria, or the population as a whole when considering overall investment in males and females. A hierarchical approach also provides insight into a much broader class of problems than sex ratios. Current research has revealed that what we usually refer to as an individual's genome is actually a rich mosaic of genetic elements of diverse taxonomic origins (Margulis 1981). These genetic elements within an individual have both common and conflicting interests with respect to their own fitnesses, leading naturally to the expression of both cooperative and antagonistic traits. Such cooperative-antagonistic symbioses are perhaps more common than is immediately apparent (Axelrod and Hamilton 1981); hints that knowledge of such symbioses may be of great practical value have recently appeared in the literature (e.g. Stoltz and Vinson 1979, Edson et. al. 1981, Clark 1982). For example, a termite hindgut is known to be a veritable ecosystem unto itself (reviewed by Margulis 1981). The success of any microbe within a termite depends mostly on the success of the termite, thus the association is mainly a cooperative one. However, a microbe's success also depends on its success within the subpopulation of similar microbes in the gut, and a microbe may be transmitted horizontally (i. e., infectiously among termites) as well as vertically (i.e., inherited from generation to generation). Thus, it is likely that on some occasions the interests of the microbe and the termite will conflict, and that the microbe may alter the phenotype of its host accordingly [e.g., a prediction is that a microbe is more likely to become virulent as its host becomes old or sick, thus increasing its ability for horizontal (infectious) transmission when the probability for vertical (inheritable) transmission is low; for an elegant example of this, see Lewin (1977) and Ptashne et. al. (1982) on the biology of the temperate bacteriophage lambda, see also Axelrod and Hamilton (1981)]. With specific knowledge about a termite gut, or any genetic mosaic, testable predictions about subtle patterns of conflict flow naturally from a hierarchical view. Recognizing these subtle patterns will occasionally prove critical to both the identification and unraveling of elusive puzzles.

SUMMARY

Sex ratios are complex phenomena. In order to formulate a general theory that spans this complex issue, four concepts were first established: (1) the possible relationships between genotypes and their expressed phenotypes were stated; (2) the significance in terms of fitness (ultimate cause) of behavior was contrasted with environmental (proximate) cues associated with behavior; (3) the levels of a genetic hierarchy (genes, chromosomes, individuals, population) at which natural selection acts effectively, and the level at which sex-ratio patterns are observed were described; and (4) the language used to describe investment of resources into males and females by examining benefit/cost ratios was defined. After these four concepts were discussed, five general sex-ratio patterns were described. In each case the theory was presented briefly, followed by a supporting em-

pirical study. Two general themes emerged; (i) an individual organism's expressed sex ratio may be consistent with ecological correlates of the relative fitness of the sexes, if the organism can assess these correlates, and (ii) conflicts in the direction of selection on the sex ratio at different biological levels (e.g., chromosomes, individuals) may result in a complex sex-ratio pattern.

APPENDIX

¹There is a large body of theory on sex ratios, mostly in the mathematical style of theoretical population genetics (Charnov 1982 and refs). Such a rigorous approach is helpful in specifying assumptions and testable predictions. However, in a broad description of sex-ratio patterns a rigorous population-genetics approach may obscure interesting biological phenomena (e.g., phenotypically plastic responses to the environment versus genetically determined responses). Therefore, I have adopted a heuristic approach that highlights biologically important concepts and often parallels the mathematical theory.

²The mechanisms involved in the expression of traits can usually be thought of as having a genetic component (in organisms such as insects). However, the phenotypic trait actually expressed may be a response to an environmental (proximate) cue mediated by that mechanism such that the response is independent of the genotype (=phenotypic plasticity). For example, if a parasitic insect has the ability (genetically determined mechanism) to vary its sex ratio according to the size of its host, the expressed, or observed sex ratio (phenotypic trait) may not have a genetic component (i.e., may be phenotypically plastic). In other words, the expressed phenotypic trait may have a direct genetic component (genetically determined), or may be independent of (or uncoupled from) the genotype by being mediated through a mechanism that yields a variable response. This observation will be important for deciding how to analyze particular sex-ratio patterns.

³It must be stressed that the patterns I discuss do not refer to the sex ratio among adults, and do not account for differential mortality and different behavioral patterns between the sexes that may lead to observed sex-ratio biases. In general, the patterns refer to the total allocation ratio of resources into males and females (Charnov 1982), e.g., the sex ratio of a clutch of eggs by an insect that does not give parental care to its offspring. These concepts are discussed later in this paper.

⁴If no correlates of fitness exist, then a genetically determined or phenotypically canalized response is likely, since nothing is gained by phenotypic plasticity. A difficult but very important problem is why, when correlates do exist, a mechanism that responds to these correlates sometimes evolves and sometimes does not. Part of the answer must be whether the organism can evolve the ability to assess the level of the correlate and a mechanism to mediate the response (a phylogenetic constraint). Another part of the answer lies in an implicit assumption about the levels of selection; the individual is considered the unit of selection in the problem stated above. For sex ratios, this assumption is not always met, and thus the problem as stated is misleading. See notes 6 and 9 for further discussion.

⁵Usually one refers to a change in the frequency of an allele (fitness) only with respect to alternative alleles found at the same locus. The accumulation of evidence in the past few years suggesting that gene conversion, gene duplication, mobile genetic elements, and viruses are not rare phenomena (Cold Spring Harbor Symposium on Quantitative Biology, v. 45 1980) makes the concepts represented by 'locus', 'alternative allele', and

'population gene pool' somewhat fuzzy. Thus, a broader view is taken here that does not restrict alleles to a particular location within the genome and allows the analysis of fitness to be conducted within a class broader than the population gene pool (e.g., the population in the usual sense plus symbiotic viruses).

⁶I am analyzing fitness(ind/pop), thus ignoring the component (allele/ind) necessary to complete the analysis of (allele/pop). A difficult and critical problem of evolutionary theory is to determine the level(s) at which selection can be meaningfully analyzed (Williams 1966, Lewontin 1970, Hamilton 1975, Alexander and Borgia 1978). Theoretical models of behavior often posit alternative alleles that have associated phenotypic characters expressed in their host, and then analyze fitness(allele/pop) (see Dawkins 1980 for discussion of models). However, in many situations (e.g., dominant autosomal allele, no allele-genotype interaction, Mendelian segregation, these being common assumptions of many population-genetic models) the benefits of a phenotypic character associated with a particular allele, or set of alleles, are shared equally by all alleles residing in that individual. For the majority of population-genetic models the phenotypic trait associated with the allele that obtains the greatest fitness is the same phenotypic trait that maximizes the number of offspring of the individual possessing that allele. Thus, in many cases, the component fitness(allele/ind) adds nothing to the analysis in a *heuristic* sense, and requires the strong assumption of an allele with a direct phenotypic effect. Often, a phenotypically plastic response (i.e., variability of the sex ratio at the level of individual phenotypes) yields the greatest individual RS within the population.

I am not suggesting that the individual is the unit of selection, and the gene is not, or vice versa. Rather, as a heuristic device, I am considering whether the underlying mechanism of sex-ratio variability is phenotypic or genetic (or a combination of the two). If sex-ratio control is genetic, then whether or not there are conflicts of interest among subgenomic elements over the sex ratio needs to be considered in order to determine the lowest level analyzed for the arguments presented here. The reasons for this will be clearer after patterns (4) and (5) are discussed.

⁷A second assumption here is that parents control the sex-ratio of their progeny, based either on the parents' genotype and/or phenotype. This certainly is not true of many plants and other organisms which are hermaphrodites, and for other sex-determination systems (Bacci 1965, Bergard 1972, Charnov and Bull 1977). Charnov (1982) handles this problem with a general approach: He asks what allocation ratio of resources, into males versus females, maximizes the benefit/cost ratio with respect to whatever entity controls this allocation ratio (e.g., a parent controls its offspring's sex, a hermaphrodite controls the allocation ratio into its sexes, etc.). Using this approach Charnov shows that equal investment holds for a much wider class of situations than parental control of progeny sex ratio.

⁸The concept of "investment of resources" is widely used, but rarely explicit. Organisms invest time, calories, rare nutrients, etc., and determining which of these is critical to measure empirically is difficult. The theory of fitness sets may be applied (see Levins 1968). In practice it is often useful to ask the question, which resource is most limiting (e.g., calories), and thus places the greatest constraint on investment patterns.

⁹The contrast in approach between Hamilton (1967, 1979) and Colwell (1981) highlights a major theme of this paper. Hamilton assumed the breeding structure outlined in the text, and additionally (i) that deme sizes are constant (=n) throughout the population, and (ii) the sex ratio is

genetically determined. When sex-ratio control is autosomal, the optimum is given by the formula in the text, and when the genetic control of the sex ratio is sex-linked or extranuclear, the optimum is reported in Hamilton (1979, fig 6). The optimum sex ratio varies widely according to the type of genetic control [see also patterns (4) and (5) in this paper], highlighting the influence of intragenomic conflict on the sex ratio. A disadvantage of this approach is that deme sizes are likely to vary in nature, and thus the optimum sex ratio will fluctuate according to ecological conditions (i.e. deme size). Therefore, phenotypically plastic sex ratios may be favored; but intragenomic conflict cannot be ignored, since a given phenotype favors different subsets of the genome unequally. Hamilton (1967) showed that for $n=2$, the sex ratio that yields the greatest number of grandprogeny to a foundress is the same as predicted by the formula in the text, $1/4$. This agreement between an autosomal model and a phenotypic model is not surprising (see note 6). Colwell (1981) demonstrated the formula in the text is the unbeatable sex ratio in a phenotypic sense (number of grandprogeny), and it also seems to be the phenotypic solution for variable deme sizes (I know of no explicit statement of this in the literature). This suggests that when analyzing what I call fitness (ind/pop), the best sex ratio for an individual foundress is given by the formula in the text, and hence phenotypic plasticity is advantageous when deme sizes vary. Colwell (1981) suggested that this phenotypic result is independent of the underlying genetic basis of sex-ratio variability. He implicitly assumed, however, that fitness (ind/pop) is equivalent with autosomal fitness within the population gene pool; and in light of Hamilton's (1979) demonstration of the importance of intragenomic conflict, this suggestion seems erroneous.

Returning to Hamilton's (1979) population-genetic approach, some biologically interesting insights emerge. First, recall that when one takes a random sample from a population, the expected sampling variance is less than the population variance— $E(s^2) = [(n-1)/n]\sigma^2 < \sigma^2$. This implies that when a few individuals randomly settle in a patch (=deme), they are genetically more homogeneous than the population as a whole. This leads to a biologically meaningful interpretation of the formula in the text. $r^* = 1/2[V(wg)/V(t)]$, where $V(wg)$ is the within-group genetic variance and $V(t)$ is the population variance (Hamilton 1979). With random assortment of colonizing females between generations, $E[V(wg)] = E(s^2) = [(n-1)/n]\sigma^2$, and $V(t) = \sigma^2$, hence $r^* = 1/2[(n-1)/n]$. Notice that the lower the within-group variance at the loci controlling the sex ratio, the more "related" ind/group are, and the more female biased the sex ratio becomes.

¹⁰This is a logical extension of note 9. The driving force behind female-biased sex ratios is the ratio $V(wg)/V(t)$. Each fig represents a random selection of wasps mostly from the local subpopulation (Frank, in prep.), and therefore genetic variance within a fig(=wg) is a sampling variance of the total variance in the subpopulation. In addition, it is likely that the subpopulations differ in genetic composition due to sampling error, selection, and drift (see Wright 1969), and possible that assortative mating occurs within a fig (Frank, in prep.), which effectively reduces $V(wg)/V(t)$. Note that $V(wg)/V(t) = [V(wg)/V(subpop)][V(subpop)/V(t)]$, which identifies the contribution of differentiation among subpopulations (Wright 1969) to female-biased sex ratios (Hamilton 1979). Figure 8 suggests that the sex ratio is phenotypically plastic, foundresses responding to the number of other foundresses (or some correlate of foundress number). It is not clear what the quantitative relationship is between the autosomal genetic variance model and a phenotypic (number of grandprogeny) model, although the predicted trends are very likely the same.

¹¹If the sex ratio is controlled by a subset of the genome, and these alleles can produce a phenotypically plastic response, then it is not number of foundresses, but genetic variance within a fig at the loci controlling the sex ratio that is important (see note 10). Hence, if these loci have a mechanism that can assess cues correlated to genetic relatedness and produce a sex-ratio response, such a response is expected. Genetic recognition would be implicated if, for a given number of foundresses, relatedness is a correlate of sex ratio.

¹²Fitness(element/pop) is a function of both fitness(element/ind) and fitness(ind/pop). Individuals harboring these elements (or a driving chromosome) may have a reduced fitness(ind/pop), possibly explaining why such elements do not dominate many populations, or when they are widespread, why they are often associated with complex suppressor systems (this will be discussed in the text). Note that fitness(element/pop) can be less than one, even though fitness(element/ind) is greater than one, due to the component (ind/pop).

¹³Similar to note 6, benefits due to a particular allele on a chromosome are usually shared equally by all alleles on that chromosome, assuming no recombination. As a caveat, it is important to note that with recombination chromosomes are not stable entities, and thus analysis usually focuses on alleles (Dawkins 1976). In the text I refer to chromosomes because it is easy to observe the effect of a driving sex chromosome—a sex-ratio bias. Identifying alleles underlying sex-ratio distortion may be quite difficult (but see Crow 1979).

¹⁴Notice that a Y chromosome which is inherited patrilineally is not different from an extranuclear genetic element that is inherited patrilineally, except that a chromosome is located in the nucleus and extranuclear elements are not. Thus patterns (4) and (5) can be grouped into a more general class, where the defining characteristic is a conflict between fitness(subgenomic element/ind) and fitness(ind/pop), when examined with respect to the investment ratio in the sexes.

¹⁵There are many other patterns discussed in the literature that were not mentioned in this paper. Most are included in Charnov (1982). For example, much has recently been written about plant sex ratios (e.g., Barrett and Helenurm 1981 and refs), the ability of adults to change sex in some fish (sequential hermaphroditism, e.g., Warner and Hoffman 1980, Warner 1982 and refs), parent-offspring conflict and the sex ratio of eusocial colonies (e.g., Trivers and Hare 1976, Alexander and Sherman 1977), competition among siblings of the same sex for resources which may lead to a population investment ratio different from 1:1 (local resource competition, Clark 1978; this pattern may be relevant to vertebrates, e.g., Clutton-Brock et. al. 1981, Clutton-Brock 1982), competition among siblings between the sexes (Pickering 1980), and bivoltinism in insects with partial overlap of generations, a pattern which may yield an investment ratio that changes from generation to generation within a single season (Seeger in press, see also Longair 1981). In general, these patterns are extensions of the basic notions reviewed in this paper.

¹⁶Sam Skinner has pointed out to me some work that is more recent than that cited in the text. Legrand et. al (1980), studying *Porcellio dilatatus*, present evidence for three types of sex chromosomes (X, Y, and Z), of which any individual has two; and they discuss the occurrence of Zelygeny, arrhenogeny, and amphogeny, in light of these sex chromosomes and the inheritable extranuclear particles that affect the expression of gender.

Juchault and Legrand (1981 and refs) report that *Armadillidium vulgare* males are homogametic (ZZ) and females heterogametic (ZW), and that two feminizing factors act to make genetic males functionally a re-

productively successful female; and one masculinizing factor that, when present simultaneously with a feminizing factor, causes genetic males to be intersexual. The population dynamics and interaction of these factors with sex chromosomes to yield a global sex ratio of approximately 1:1 in nature are discussed in these papers.

Bulnheim (1978), synthesizing his own work on the amphipod *Gammarus duebeni*, reports that the sex of an individual has environmental, genetic (nuclear), and parasitic (extranuclear elements) components. The sex of an uninfected individual depends on an interaction between a polygenic sex-determination system and photoperiod during ontogeny (the photosensitive phenocritical period of sex differentiation is delimited by the second to fourth molts). In addition to environmental and genetic components, this amphipod harbors two transovarially transmitted (matrilinal) microsporidians that have a feminizing influence on a developing individual, but these parasites interact with environmental and probably nuclear genetic factors in determining gender.

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