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Sex allocation in animals

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Introduction

Sex allocation refers to the apportionment of effort between 'male' and 'female' functions, whether it takes the form of deciding the ratio of male to female offspring, the age at which to change from one sex to the other, or the balance between male and female functions in a hermaphrodite. This subject represents one of the most fruitful blends of theory and observation in all of evolutionary biology[9]. The theory of sex allocation provides precise, distinctive, seemingly accurate predictions concerning a wide variety of measurable phenomena. Moreover, sex allocation has assumed particular importance as the source of the best evidence for the efficacy of selection within populations[29].

This paper is not a review of the literature on sex allocation in animals: Charnov[9] has done that, and he needed a whole book for the job. Instead, we will treat the theory of sex allocation as an example of the study of adaptation. We will analyze the assumptions and display the structure of the theory of sex ratio, and consider some generalizations of this theory. We then ask when sexes should be separate and permanent, when animals should be one sex when young and the other when old, and when they should be simultaneous hermaphrodites. Finally, we consider the nature of the evidence for this body of theory, and the wider implications of this theory for evolutionary biology.

Prolegomena to the study of adaptation

One shows that a structure or behavior is adaptive by demonstrating its appropriateness for a function important to the life or wellbeing of the organism[51]. This involves two steps: enumerating the 'fitness set' of possible structures or behaviors that could conceivably serve that function, that is to say, the set of possible phenotypes; and providing a criterion for comparing their appropriateness. Levins[32,33] schematized the analysis by generating contours of equal value on the fitness set.

Finding the fitness set is often a rather ad hoc affair, at best an art, at worst a tissue of circular argument. The art lies in choosing a problem where the relevant aspects of the possible phenotypes can be specified by one or two coordinates, and where the optimum lies *inside* the fitness set, leaving no question that the 'best' phenotype is attainable.

To find the adaptive function, we must first discover how natural selection acts on the trait concerned: is the decisive process selection between genes, between individuals, or between whole populations? Thus we must know how to distinguish the various levels of selection.

Selection presupposes units which replicate, with some degree of heritable variation affecting the rates of survival and replication of these units[34]. Genes are units of selection[14], but so can species be[47,48]. Populations within a species are sometimes so distinct, exchanging almost no migrants, and so obviously replicate by 'fission', that one can then speak of selection between populations[34].

Genes, however, replicate primarily according to their contribution to the viability and fecundity of the individuals carrying them: to this extent, we may say that the fates of these genes are governed by selection between individuals. Genes occasionally spread by means harmful to their bearer organisms, but such genes violate the common interest of the genome as a whole: at other loci, individual selection favors modifiers which suppress these 'outlaw genes'[1,28,29]. Genes may also be selected according to their effects on interactions between relatives, leading to kin selection[21].

One also hears of 'group selection'. Unfortunately, this term has a double meaning. Group selection can mean selection between populations, a term that only applies if populations are units of selection. Group selection can also mean the influence on genic fitness of interactions between members of the same 'traitgroup'[65], an influence which reflects how an individual's fate is linked with that of its neighbors. This influence does not even depend on the subdivision of a species into discrete groups[6]. The latter definition is so much more inclusive – contrast, for example, the conclusions of Uyenoyama and Feldman[56] with those of Leigh[30] – that group selection is no longer a useful word. When populations are units of selection we will speak of selection between populations, and we will refer to all other processes where genic selection is influenced by interactions between groups as traitgroup or neighbor selection.

Analytical review of sex allocation theory

I) Introit

Sex allocation theory seems to be a textbook analysis of adaptation[28,39]. We begin by outlining the theory as it first developed, and then analyze its mathematical foundations and consider some of the relevant evidence. The theory originally asked how offspring of gonochores – organisms where sexes are separate – should be apportioned between the two sexes. The theory assumed that

1) alleles affecting sex ratio affect nothing else, neither the survival prospects of their bearers, nor their bearers' ability to find mates, nor other features of their reproductive capacity;

2) each allele affecting sex ratio prescribes a fixed sex

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ratio, which cannot be adjusted to suit fluctuations in the sex ratio of the rest of the population;
3) mating is random: that is to say, each female is equally likely to mate with any mature male in the population;
4) offspring inherit sex ratio genes equally from each parent: thus both sexes have the same ploidy, and the sex ratio locus is autosomal.

In contrast to most characteristics whose adaptation might be analyzed, the array of possible sex ratios is easy to specify, and the fitness value of a given sex ratio is fairly easy to calculate. Let the proportion of reproductive effort devoted to sons be k. Then k represents a possible sex ratio, and the array of possible sex ratios is represented by the set of k: $0 \leqslant k \leqslant 1$. To be specific, assume that a mating can produce S sons, W daughters, or kS sons and $(1 - k)$W daughters. Thus W daughters can be said to cost as much as S sons, and k represents the proportion of effort devoted to sons.

To compare the values of different sex ratios, remember that every individual has father and mother[18]. Thus each sex contributes equally to the genes of the next generation, and the frequency of an allele is best measured by the average of its frequency among males and its frequency among females[44]. It follows that selection on autosomal genes favors a rare allele A if the proportionate gain to one sex from its change in sex ratio exceeds the proportionate loss to the other. To see this, suppose that there are $n_{AM} = k_A S$ A sons and $n_{AF} = (1 - k_A)$W A daughters per parent bearer of a rare allele A, while there are $n_M = k'S$ sons and $n_F = (1 - k')$W daughters per parent in the population at large. Then A's selective advantage is

$$\tfrac{1}{2}(n_{AM}/n_M + n_{AF}/n_F) - 1 \approx \tfrac{1}{2}[k_A/k' + (1-k_A)/(1-k')] - 1$$
$$= \tfrac{1}{2}[(k_A - k')/k' + (k' - k_A)/1 - k')] = (k_A - k')V(k'),$$

where $V(k') = (\tfrac{1}{2} - k')/k'(1 - k')$. A's advantage depends on the sex ratio k' of the population as a whole: if $k' < \tfrac{1}{2}$ genic selection favors A if k_A exceeds k, and vice versa. We may measure the population's vulnerability to *genic* selection for a new sex ratio mutant by the absolute value of V(k'): the lower $|V(k')|$, the more nearly perfect is the population's sex ratio or, more concretely put, the less liable the population's sex ratio is to further change from new mutants. The population is 'perfect', and no new sex ratio mutant is favored, when $V(k') = 0$, that is to say, when $k' = \tfrac{1}{2}$ and the population is devoting equal effort to each sex. Is $|V(k')|$ the adaptive function for the fitness set of possible sex ratios k'?

k' is a *population* property. So long as the mean sex ratio k' for the population as a whole is ½, it makes no difference what sex ratio any given allele prescribes: half the population's females could bear only daughters, and half, only sons. Indeed, an infinite array of polymorphisms in sex ratio is consistent with the condition $k' = \tfrac{1}{2}$. $|V(k')|$ is thus an adaptive function for the *population's* sex ratio from the standpoint of selection on autosomal genes.

Moreover, an allele for the 'optimum' sex ratio $k = \tfrac{1}{2}$ always increases in frequency unless the population sex ratio k' is already ½. If no mutant adjusts its sex ratio to suit the circumstances by producing the rare sex when one sex is in short supply, the optimum ratio is unbeatable.

This optimum represents the common interest of the genome[18, 29, 38]: any autosomal gene benefits from having its replicates partitioned in the optimum ratio between sons and daughters[49]. Thus it makes no difference whether the sex ratio is controlled by one or both parents, by each offspring individually, or collectively by the offspring of a given brood or family. $k = \tfrac{1}{2}$ is optimum whether our animals are haploid or diploid, whether sex ratio is governed by the usual segregation of sex chromosomes, or determined by the environment as in certain turtles[4] where eggs incubated above a given temperature are female and vice versa.

This sex ratio theory also applies to randomly mating hermaphrodites. If we view the gametes, male and female, they produce as haploid individuals of separate sexes and treat the diploid hermaphrodite as an elaborate and wantonly protracted mating which eventually produces more gametes, then the mathematics of haploid sex ratio apply. Now, however, the decline in a hermaphrodite's own egg production imposed by fertilizing an additional egg from another individual depends on the fraction of its effort already devoted to making eggs. Thus each hermaphrodite should divide its effort so that the proportionate gain in egg production conferred by a slight increase in female function just balances the proportionate loss thereby imposed on number of eggs fertilized. This optimum does not allow for the variety of interchangeable polymorphisms possible among gonochores.

One might expect the mathematics of sexual haploids to cover all sex allocation theory: after all, we could treat diploid gonochores as matings each one of which produces either all male, or all female, gametes. Introducing unequal costs for male-producing and female-producing matings is, however, an artificial business, at least as yet. Shaw and Mohler's[44] criterion of fitness value can be generalized to cover different types of sex allocation. If we compare the number n_{AF} of eggs produced, and the number n_{AM} of eggs fertilized, per newborn of a rare genotype A with the corresponding numbers n_F and n_M per newborn in the population as a whole, then A spreads if $n_{AF}/n_F + n_{AM}/n_M$ exceeds 2.

II) Basic theory of sex allocation

How does selection affect autosomal alleles which influence sex ratio? Is Shaw and Mohler's[44] criterion of fitness correct? Is there a true common interest among different autosomal genes? To address these questions, we consider sexual haploids, and assume that the probability an individual is male is governed by which sex ratio allele it carries. The diploid version complicates the mathematics without changing essentials[27, 54]. The conditions under which selection on diploids favors a mutant so rare that it occurs only in heterozygous form are the same as for a rare mutant among sexual haploids. We also suppose that a sex ratio allele influences the probability that its bearer will abort before birth, as well as the probability that it will be male, in such a way that each mating produces kS sons and $(1 - k)$W daughters, where k differs for different sex ratios alleles. This assumption restricts our attention to alleles whose reproductive effort per mating is the same: it is not our desire to confound

the effects of changing sex ratio with those of changing reproductive effort. Our model of sex determination seems artificial, but again, it simplifies the mathematics. Moreover, were an allele prescribing a given sex ratio to act, not directly on the sex of the bearers themselves, but on the sex ratio of the offspring of female bearers, genic selection would affect it the same way: female bearers of that allele would pass it on to their offspring in the sex ratio that allele would have prescribed had it affected the offspring directly. However, the selection would be only half as strong, as copies of the allele carried by males exert no effect. The same remark applies if sex ratio is controlled by fathers, or by both parents conjointly.

We consider populations where successive generations are distinct, first assuming sex ratio is only influenced by one locus, then passing to a polygenic model to ask about the probable precision of adaptation in sex ratio. Then we show why the overlapping of generations does not affect the direction of selection on the sex ratio.

A) Separate sexes

1) Discrete generations, one locus

Consider a locus affecting sex ratio with two alleles, A and B, in a population of sexual haploids, and let $N_{BF}(t)$, $N_{BM}(t)$, $N_{AF}(t)$ and $N_{AM}(t)$ be the numbers of adult B females, B males, A females and A males at generation t. Suppose that each female mates only once, and let a mating of B with B produce Sk B males and W(1 − k) B females, while a mating of A with A produces S(k + d) A males and W(1 − k − d) A females, and a mating of A with B produces ½Sk B males, ½W(1 − k) B females, ½S(k + d) A males and ½W(1 − k − d) A females. If each generation mates at random, and if a fraction F of the young females and a fraction M of the young males mature, then $N_{BF}(t + 1)$ is

$$WF(1-k)[N_{BF}(t)N_{BM}(t)/N_M(t) + \tfrac{1}{2}N_{BF}(t)N_{AM}(t)/N_M(t) + \tfrac{1}{2}N_{AF}(t)N_{BM}(t)/N_M(t)]$$

$$= WF(1-k)\{\tfrac{1}{2}N_{BF}(t)[N_{BM}(t) + N_{AM}(t)]/N_M(t) + \tfrac{1}{2}N_{BM}(t)[N_{BF}(t) + N_{AF}(t)]/N_M(t)]\}.$$

$$N_{BF}(t+1) = WF(1-k)N_F(t)[\tfrac{1}{2}N_{BF}(t)/N_F(t) + \tfrac{1}{2}N_{BM}(t)/N_M(t)] = WF(1-k)N_F(t)Q_B(t),$$

where $N_F(t) = N_{BF}(t) + N_{AF}(t)$ is the total number of adult females at generation t, $N_M(t)$ is the number of adult males, and $Q_B(t)$ is the average of B's frequency $N_{BF}(t)/N_F(t)$ among the females, and its frequency $N_{BM}(t)/N_M(t)$ among the males, of generation t. Similarly,

$$N_{BM}(t) = MSkN_F(t)Q_B(t);$$
$$N_{AF}(t) = FW(1-k-d)N_F(t)Q_A(t);$$
$$N_{AM}(t) = MS(k+d)N_F(t)Q_B(t);$$

where $Q_A(t) = 1 - Q_B(t)$. Letting $k' = Q_Bk + Q_A(k + d) = k + Q_Ad$ be the average proportion of the population's reproductive effort spent on males, we find that

$$Q_B(t+1) = \tfrac{1}{2}\left(\frac{1-k}{1-k'} + \frac{k}{k'} + \frac{k}{k'}\right)Q_B(t) = \tfrac{1}{2}(n_{BF}/n_F + n_{BM}/n_M)Q_B(t);$$

$$Q_A(t+1) = \tfrac{1}{2}\left(\frac{1-k-d}{1-k'} + \frac{k+d}{k'}\right)Q_A(t) = \tfrac{1}{2}(n_{AF}/n_F + n_{AM}/n_M)Q_A(t);$$

where n_{BF} and n_{BM} are the numbers of B daughters and B sons per B parent, n_{AF} and n_{AM} are the numbers of A daughters and A sons per A parent, and n_F and n_M are the numbers of daughters and sons per parent in the population at large. As Shaw and Mohler[44] expected, genic selection increases A's frequency if $n_{AF}/n_F + n_{AM}/n_M$ exceeds 2. More specifically,

$$Q_A(t+1) - Q_A(t) = \tfrac{1}{2}\left(\frac{1-k-d}{1-k'} - \frac{1-k'}{1-k'} + \frac{k+d}{k'} - \frac{k'}{k'}\right)Q_A(t)$$
$$= \tfrac{1}{2}Q_A(t)[1 - Q_A(t)][d/k' - d/(1-k')]$$
$$= Q_A(t)Q_B(t)V(k')d,$$

where $V(k') = (\tfrac{1}{2} - k')/4k'k(1 - k')$. If the proportionate gain in male offspring from an increase d in sex ratio outweighs the proportionate loss to the females, genic selection favors the change: the sex ratio is optimum when $k = \tfrac{1}{2}$ and the loss just balances the gain.

The equations in Q apply whatever the values of F, M, S or W, no matter how they change from generation to generation, so long as they are the same for A's and B's. Moreover, so long as the number of matings per female is not affected by the sex ratio locus, it does not matter how often they mate, or how their frequency of mating changes from generation to generation.

2) Discrete generations, polygenic control of sex ratio

Suppose now that other loci besides the one with alleles A and B affect sex ratio. How does selection at all these loci change the mean sex ratio k'? How precisely can we expect k' to approach ½? If the different loci assort independently, so that A and B have the same spectrum of genetic backgrounds, our equation for change in Q_A applies. Since $k' = k + Q_Ad$, the change in k' from one generation's selection on A is

$$k'(t+1) - k'(t) = [Q_A(t+1) - Q_A(t)]d = Q_A(t)Q_B(t)V(k')d^2 = S_A^2V(k'),$$

where $S_A^2 = Q_A(k - k')^2 + Q_B(k + d - k')^2$ is the variance in k associated with the A locus[18, 19, 24]. Considering the joint effect of all sex ratio loci,

$$k'(t+1) = k'(t) + S_T^2(t)V(k') = k'(t) - S_T^2(T)(\tfrac{1}{2} - k')/4k'(1-k'),$$
$$k'(t+1) - \tfrac{1}{2} = [k'(t) - \tfrac{1}{2}][1 - S_T^2(t)/4k'(1-k')],$$

where $S_T^2(t)$ is the heritable part of the total variance in sex ratio at generation t. Polygenic control does not affect the direction of genic selection on sex ratio[5].

How does the variance $S_T^2(t)$ change over time? Since different loci assort independently, we expect the proportion of individuals at generation t programmed for sex ratios between k and k + dk to be

$$dk(1/S_T\sqrt{2\pi})\exp - (k - k')^2/2S_T^2$$

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(see Falconer[15]). This is reasonable only if S_T^2 is so small that individuals programmed for k near 0 or 1 are rare. Individuals programmed for sex ratios between k and k + dk are 1 + (k − k′)V(k′), or exp (k − k′)V(k′), times as abundant in generation t + 1 as in generation t: at t + 1, their proportion in the population is

$$Cdk(1/S_T\sqrt{2\pi}) \exp[(k - k')V(k') - (k - k')^2/2S_T^2]$$
$$= dk(1/S_T\sqrt{2\pi}) \exp - [k - k' - V(k')S_T^2]/2S_T^2,$$

where C is a constant which makes the proportions sum to 1. Selection increases k′ an amount $V(k')S_T^2$ per generation, as we expect, but it does not shrink the variance. Instead, variance is governed by the balance between mutation and the restricting effects of finite population size. In a finite population with N equally reproductive individuals, a fraction 1/N of the variance is lost each generation through the random sampling of N mature individuals from the gametes formed by their parents, while each generation's mutation adds an amount U to the variance[26]. Thus, at equilibrium, $S_T^2/N = U$, $S_T^2 = NU$, so long as U is small enough that S_T^2 is far less than 1.

Finally, how precisely should k′(t) approach ½? If sex ratio alleles act directly on their bearers,

$$k'(t + 1) - ½ = [k'(t) - ½][1 - S_T^2/k'(1 - k')].$$

Selection thus decreases the mean square of the difference between k′ and ½ by a factor $[1 - S_T^2/k'(1 - k')]^2$ each generation. In a finite population, however, sampling N mature individuals from an array where k has variance S_T^2 introduces a variance of S_T^2/N in k′[26]. If S_T^2 is much smaller than 1, then, when the mean sex ratio k′ is varying in steady state about its optimum,

$$[k'(t) - ½]^2[2S_T^2/k'(1 - k')] = S_T^2/N.$$

At steady state, when k′ has ceased to change in a directed manner, finite population size causes a mean square deviation of k′ from ½ of k′(1 − k′)/2N. Were sex ratio maternally controlled, the mean square deviation of k′ from ½ would be four times greater. It would require a huge sample of the population's young to verify such a deviation. If each newborn had probability k′ of being male, the variance in the estimate of k′ from a sample of n young would be k′(1 − k′)/n: even for n larger than N, this variance is of magnitude comparable to that in the deviation we would be trying to detect. Theoretically, adaptation in sex ratio should not differ significantly from utter perfection.

3) Separate sexes, overlapping generations

Is the optimum sex ratio the same where generations overlap? Consider a locus affecting sex ratio with two alleles, A and B, in a population of sexual haploids with overlapping generations, and assume that these alleles only influence sex ratio. How is A's frequency among the adults of year t + 1 related to that in year t? It is a compound of A's frequency among the survivors from year t, and of A's frequency among the newly matured 'recruits' of generation t + 1. As survival prospects are unaffected by the sex ratio locus, A's frequency only changes through the newly matured recruits. But the difference in A's frequency among these recruits from that among their parents is just what it would be were the recruits and their parents separate and distinct generations. Thus the optimum sex ratio is the same whether or not generations overlap.

Suppose, to be specific, that there are $N_{BF}(t)$ mature B females, $N_{BM}(t)$ mature B males, $N_{AF}(t)$ mature A females and $N_{AM}(t)$ mature A males at year t. Let proportions S_F and S_M of the mature females and males of year t survive to year t + 1; let mature females each mate once a year, and let matings of B with B each yield kS B sons and (1 − k)W B daughters, matings of A with A yield (k + d)S A sons and (1 − k − d)W A daughters, and matings of A with B yield ½kS B sons, ½(1 − k)W B daughters, ½(k + d)S A sons, and ½(1 − k − d)W A daughters. If a proportion F of the daughters mature, taking s + 1 years to do so, while a proportion M of the sons mature, taking r + 1 years to do so, then $N_{AF}(t + 1)$ is

$$S_F N_{AF}(t) + WF(1 - k - d)[N_{AF}(t - s)N_{AM}(t - s)/N_M(t - s) + ½N_{AF}(t - s)N_{BM}(t - s)/N_M(t - s) + ½N_{BF}(t - s)N_{AM}(t - s)/N_M(t - s)]$$
$$= S_F N_{AF}(t) + WF(1 - k - d)N_F(t - s)Q_A(t - s).$$

Similarly,

$$N_{AM}(t + 1) = S_M N_{AM}(t) + SM(k + d)N_F(t - r)Q_A(t - r);$$
$$N_F(t + 1) = S_F N_F(t) + WF(1 - k')N_F(t - s);$$
$$N_M(t + 1) = S_M N_M(t) + SMk'N_F(t - r).$$

$N_{AF}(t + 1) - S_F N_{AF}(t)$ is the number of adult A females recruited to the population between years t and t + 1; the numbers of recruits of other types are defined similarly. The sum of A's frequencies among the female, and the male, recruits to the population in year t + 1 is

$$\frac{N_{AF}(t + 1) - S_F N_{AF}(t)}{N_F(t + 1) - S_F N_F(t)} + \frac{N_{AM}(t + 1) - S_M N_{AM}(t)}{N_M(t + 1) - S_M N_M(t)}$$
$$= Q_A(t - s)\left(\frac{1 - k - d}{1 - k'}\right) + Q_A(t - r)\left(\frac{k + d}{k'}\right).$$

Since recruits are the only source of change in A's frequency, it appears that A's frequency increases if $(1 - k - d)/(1 - k') + (k + d)/k'$ exceeds 2, that is to say, if d(1 − 2k′) exceeds 0. This is obviously true if r = s: if r ≠ s, the argument is more delicate, but the conclusion still holds. The optimum sex ratio is not affected by the values of W, S, F, M, S_F and S_M or the yearly fluctuations therein, so long as the sex ratio locus does not affect these quantities. Indeed, regardless of the mode of sex determination, it makes no difference to the optimum sex ratio whether or not generations overlap, *if* sex ratio is influenced only by autosomal genes.

4. Evidence for basic sex ratio theory

Most often, as many males are born as females[9]. Among humans, and in several other well-studied vertebrate populations, genetic variation in sex ratio is undetectably small[64], in striking contrast with most characteristics,

Proportions $\bar{k}$ of males among offspring of fig wasps in traitgroups of different size m belonging to species with different harmonic means of traitgroup size (foundress number) n, as compared to the optimum k*, $(2n-1)(m-1)/(4n-1)m$

Wasps from *Ficus insipida* (mean foundress number n=3.57)						
Foundress number	m = 1	2	3	4	5	6
Optimum k*	0	0.2312	0.3082	0.3468	0.3699	0.3853
Observed $\bar{k}$	0.1441	0.2568	0.3180	0.3873	0.4105	0.4119
95% CL	0.124–0.165	0.216–0.300	0.274–0.364	0.316–0.461	0.341–0.481	0.357–0.468
Sample size N	34	17	17	8	7	7
Wasps from *Ficus popenoei* (mean foundress number n = 1.72)						
Foundress number	m = 1	2	3	4		
Optimum k*	0	0.2102	0.2803	0.3153		
Observed $\bar{k}$	0.1011	0.2008	0.2287	0.3199		
95% Cl	0.089–0.110	0.168–0.236	0.201–0.258	0.270–0.372		
Sample size N	8	15	10	9		
Wasps from *Ficus citrifolia* (mean foundress number n = 1.17)						
Foundress number	m = 1	2				
Optimum k*	0	0.1821				
Observed $\bar{k}$	0.0595	0.1568				
95% CL	0.050–0.070	0.123–0.195				
Sample size N	25	33				

Observed $\bar{k}$ and 95% confidence limits (CL) are calculated from the formula

$$\bar{k} \pm CL = \sin^2\left[\frac{1}{N}\sum_{i=1}^{N}\left(\arcsin\sqrt{k_i}\right) \pm \frac{2SD}{N}\right],$$

where k_i is the proportion of males in fig i out of the sample of N, and SD is the SD in $\arcsin\sqrt{k_i}$.

which exhibit detectable genetic variation[15]. This contrast is particularly remarkable because, as we have seen, genic selection on the sex ratio does not restrict genetic variation in sex ratio, while most characteristics are subject to stabilizing selection[18], which does restrict their variance[53]. The absence of genetic variation in sex ratio must therefore mean that mutation rarely affects sex ratio, and that sex ratio in the population is not responsive to selection. Instead, their equal sex ratios must reflect the symmetry of meiosis, the rigidity of which will be discussed below. If the costs of male and female births differ, and if the trade-off between sons and daughters does not affect the mother's survival, then selection on autosomal genes should lead the population to spend equal effort on offspring of each sex. How should we measure effort? The problems involved are suggested by a study of European sparrowhawks[40]. Of 2163 fledgelings, 1063 were female, even though females were twice as heavy. There was no sign of differential mortality in the nest. As it turns out, nestlings of each sex received equal amounts of food, so equal effort was spent on each, but one would never know it from their weights. To test sex ratio theory, one must assess the relative costs of each sex in a manner that avoids the temptation of choosing a measure so as to verify the theory, and this is likely to prove a tricky business.

On the other hand, where sex ratios vary significantly among the members of a population, it is reasonable to test sex ratio theory by asking whether the mean ratio k' for the population at large differs significantly from ½. Among redwing blackbirds in Indiana[2], young mothers hatched 36 males out of a total of 88 sexed hatchlings, middle-aged mothers hatched 54 males of 120, and old mothers hatched 55 males among 89 young. Although the heterogeneity in sex ratio was significant, in all 145 of 297 sexed hatchlings were male, not significantly different from $k' = ½$.

B) Hermaphrodites

1) Theory

When might gonochores be replaced by a genotype where one individual performs both sexual functions? Let us suppose that a hermaphrodite which devotes a fraction $1-k$ of its reproductive effort to producing eggs and the remainder to fertilizing the eggs of others, produces $n_F(k)$ eggs and fertilizes $n_M(k)$ eggs, while a female produces $n_F(0)$ eggs and a male fertilizes $n_M(1)$. If members of our population mate at random, and if we treat eggs and 'successful' sperm as haploid males and females, and their zygotes, whether gonochore or hermaphrodite, as matings between these haploids, then sex ratio theory tells us when selection favors hermaphrodites. In a population a fraction k″ of whose zygotes produce only male gametes and the rest, only female, selection favors a dominant mutant, all of whose zygotes devote a fraction k of their effort to producing sperm (or ensuring their success), if $n_F(k)/(1-k'')n_F(0) + n_M(k)/k''n_M(1)$ exceeds 2. In a population composed exclusively of such hermaphrodites, selection favors a dominant mutant for gonochorism a fraction k″ of whose zygotes are 'male', if $(1-k'')n_F(0)/n_F(k) + k''n_M(0)/n_M(k)$ exceeds 2.

If $n_F(k) = n_F(0)(1-k)$, $n_M(k) = kn_M(0)$, there is selective equipoise between such hermaphrodites and a gonochore genotype a fraction k of whose zygotes are male. Usually, producing eggs and fertilizing them are sufficiently different occupations – at least for 'higher' animals and plants – that, other things being equal, it pays to specialize. Thus sexes should be separate unless there is some special advantage to combining the sexual functions.

If the advantage to combining the sexual functions is great enough that, for some suitable value of k, a hermaphrodite produces more than half as many eggs as a female gonochore and fertilizes more than half as many eggs as a male, then selection favors hermaphroditism. In

a population of gonochores, a mutant for such hermaphroditism would spread, while selection eliminates gonochore mutants from a population of such hermaphrodites. In this case, selection favors the value of k for which the proportionate gain in number of eggs produced $[n_M(k + d) - n_M(k)]/n_M(k)$ from a slight increase d in effort fertilizing eggs balances the proportionate loss $[n_F(k + d) - n_F(k)]/n_F(k)$ thereby imposed on eggs fertilized[10, 31].

Finally, selection might favor a rare mutant for hermaphroditism in a population of gonochores, even though it would favor a mutant whose bearers were all gonochores of one sex in a population composed exclusively of such hermaphrodites. For example, an all-male mutant would be favored if $n_M(0)/n_M(k) > 2$. If so, selection would increase this mutant's frequency to the value X where this mutant's bearers fertilized just twice as many eggs per zygote as the average for the population at large – where $n_M(0) = 2Xn_M(0) + 2(1 - X)n_M(k)$. However, hermaphroditism cannot be maintained in stable polymorphism with both male and female gonochores[10].

2) Simultaneous hermaphroditism

There are four categories of animals where one would most expect individuals to benefit from carrying on both sexual functions at once[10, 16]:

1) Simple animals, where different sex roles require little specialization.

2) Those sessile animals with planktonic larvae which can only fertilize near neighbors. By sacrificing less than half of a female's egg production, a sessile hermaphrodite can fertilize more than half as many eggs as a male.

3) Animals which, like most higher plants, carry out their male functions (such as making pollen) before bearing their young. Here, the energy devoted to male function does not divert equivalent energy from female function, and a hermaphrodite can do more than half as well as a gonochore at either.

4) Animals whose simultaneous hermaphroditism permits a reciprocal altruism obviating most of the competitive aspects of maleness.

Many factors which favor simultaneous hermaphroditism could, under different circumstances, favor either gonochores with males fewer or smaller than females (as opposed to females which carry out male functions on the side), or monogamous gonochores whose males devote much of their effort to 'female functions' such as parental care. Thus animals where males can only mate with a very few females should raise more females than males[6]. Synchrony of reproduction, which favors simultaneous hermaphroditism in plants, favors male participation in parental care among gonochores, by depriving males of the prospect of mating with other females when they desert their offspring[25].

Particularly interesting in this respect is the role social convention sometimes plays in sex allocation. Just as social conventions promote male participation in parental care by closing off other avenues of increasing reproductive success, such conventions provide a striking advantage for simultaneous hermaphroditism among coral reef fish of the family Serranidae. Hamlets, *Hypoplectrus,* are simultaneous hermaphrodites which mate in pairs, switching sex roles after successive spawns: in effect, members of a pair trade eggs for each other to fertilize. In a remarkably human manner, mates only offer each other a few eggs at first, then more as confidence builds up. This 'hesitancy' penalizes fish that move from mate to mate fertilizing eggs without offering any in return[16], imposing a law of sharply diminishing returns on male function. Thus nearly fully functional females act as males 'on the side', with little sacrifice in egg production[17].

Although the incidence of simultaneous hermaphroditism appears to agree at least roughly with theory, no survey of its incidence is complete enough to judge the accuracy of theory in this respect. Nor can we predict the circumstances under which it is feasible to evolve an otherwise desirable hermaphroditic habit. Finally, it is even more difficult to measure, unambiguously, the effort hermaphrodites apportion to male versus female functions than to assess the relative costs of male versus female births among gonochores. In short, hermaphrodites do not offer much scope for precise tests of sex allocation theory.

3) Sequential hermaphroditism

The mathematics of hermaphroditism also apply to animals which are born into one sex and change sex later on. Such animals divide their effort between male and female functions by choosing an age T to change sex. Formally, we may imagine our hermaphrodite passing through successive ages, each more favorable, say, to producing male gametes rather than female, to be an animal in a heterogeneous environment moving through patches successively more favorable to producing males. In a constant environment, such an animal should produce only males in the patches most favorable for males, and vice versa, and a mixture of the two sexes in the one intermediate patch where increasing the proportion of females confers a proportionate gain in the total number of females produced in all patches, just balancing the proportionate loss in males[3]. Likewise, sex change should be 'all or nothing', the hermaphrodite producing only female gametes until it is more profitable to produce males, then switching entirely to the latter.

Such sequential hermaphrodites have offered some spectacular tests of sex allocation theory. The clear temporal distinction between male and female functions makes the apportionment of effort between them much easier to assess than in simultaneous hermaphrodites. Moreover, since the age of sex change is usually an individual decision, it is more responsive to environmental circumstance than sex ratio among gonochores, or the apportionment of effort between male and female functions by simultaneous hermaphrodites. The most conspicuous difficulty is that sex ratio theory assumes that all offspring of a given sex from one mating are produced simultaneously, and are equally valuable. The offspring of sequential hermaphrodites are produced over a long period: to apply the theory, we must assume that the numbers and age composition of a population of sequential hermaphrodites do not change with time.

When should sequential hermaphrodites replace gonochores? Ghiselin[20] predicted that if one sex gained fertility

more rapidly with age than the other, an animal should be born into the sex where youth hurts least, and change later to that sex where age benefits most. To explore the matter further, consider an animal which is born female and turns male at age T. Let us measure the proportion $1 - k(T)$ of its effort devoted to female function by the proportion of a newborn gonochore female's prospective egg output produced by age T. If $l(x)$ is the probability a female survives to age x, and if $b(x)$ is the egg output per unit time of a female aged x, then the average egg output n_{FT} per newborn fish programmed to change sex at age T is

$$n_{F\infty}[1 - k(T)] = \int_0^T b(x)l(x)dx,$$

where $n_{F\infty}$ is the egg output per female gonochore (female that never changes sex).

Let male fertility grow more rapidly with age than female. In particular, let a male aged x fertilize $b(x)f(x)$ eggs per unit time, let a proportion $L(y + x)/L(y)$ of the animals turning male at age y survive to age $y + x$, where $L(y)$ is the proportion of male gonochores surviving to age y, and let $G(x) = L(x)f(x)/l(x)$ be an increasing function of x, so that $dG/dx > 0$. Then the fraction $m(T)$ of a male gonochore's reproductive success realized by a fish which turns male at age T is

$$\frac{\int_T^\infty dx b(x)f(x)L(x)l(T)/L(T)}{\int_0^\infty dx b(x)f(x)L(x)} = \frac{l(T)\int_T^\infty dx b(x)l(x)G(x)}{L(T)\int_0^\infty dx b(x)l(x)G(x)} < \frac{l(T)k(T)}{L(T)}.$$

Let T″ be that age when a female exhausts half its prospective egg output. Then $1 - k(T'') = k(T'') = ½$. If the chance $l(T'')$ a female survives to age T″ is at least as great as the chance $L(T'')$ a male does so, then $m(T'') > ½ = k(T'')$, and there is an age of sex change $T'' + s$, for which $m(T'' + s) > ½$, $1 - k(T'' + s) > ½$. Selection favors animals changing sex at that age over gonochores. The more rapidly male fertility grows relative to female, the larger dG/dx, the greater $m(T'') - ½$, the greater the advantage of changing sex, and the older animals should be when they do so.

Although animals which do change sex provide good tests of sex allocation theory, many animals whose males only reproduce when old and large are not sequential hermaphrodites. Thus sex allocation theory provides a necessary, but not a sufficient, condition for sex change. It is easy to say that in higher vertebrates sex roles are so specialized, morphologically and behaviorally, that it is impractical to change sex[57], but we cannot disguise our inability to predict accurately those lineages for which sex change is an attainable alternative.

The best studied sequential hermaphrodite is the bluehead wrasse, *Thalassoma bifasciatum*, most of whose individuals are born female and turn male later. A female bluehead spawns once a day, while a large male spawns 40 times a day: there is a distinct advantage to being male when large[59]. Pandalid shrimp, on the other hand, are often born male, but all turn female when large: it is thought that males derive no advantage from size, while the egg output of female shrimp increases with the cube of their length[8].

Theory predicts that females should turn male at that age T′ when the proportionate gain in egg production from a slight delay in changing sex just balances the proportionate loss in male reproductive success, that is to say, when

$$\frac{b(T')l(T')dT}{\int_0^{T'} b(x)l(x)dx} = \frac{f(T')b(T')l(T')dT}{\int_{T'}^\infty f(x)b(x)L(x)dxl(T')/L(T')}.$$

If we assume all eggs are fertilized, and that as many eggs are fertilized per newborn as are produced, then the proper age to change sex is that age when $b(T') = f(T')b(T')$, that is to say, when a fish can fertilize just as many eggs as it produces. If sex change involves some delay, during which the probability of death is p, the proper age to change sex is that for which $b(T') = f(T')b(T')/p$. Female bluehead wrasses all spawn once a day. Crediting $1/n$ spawn to a male which fertilizes a female's eggs jointly with $n - 1$ other males, male blueheads that have only recently changed sex fertilize 1.5 spawns per day[59].

In many social systems where the largest males compete most successfully for females, very small males may be able to escape notice and fertilize females 'on the sly'. Some bluehead wrasses, for example, are born male, but with female colors: these fertilize females by various forms of 'stealth'. When a 'drab' male is large enough to compete overtly for mates, it assumes the gaudy colors of transformed males and joins the 'establishment' it previously 'subverted'. If the growth and death rates of females and similar-sized males are the same, then, at equilibrium, the average number of matings per day should be the same for drab males as for females[59]. On small reefs, the proportion of individuals born male is the same among 'drab' as among 'gaudy' fish, suggesting that females and drab males suffer the same mortality. Drab males fertilize a mean of 1.1 ± 0.2 spawns a day, indistinguishable from the females' one spawn a day[59]. If the same conditions apply on larger reefs, then, where gaudy males fertilize a fraction Y of the spawns, there should be $1 - Y$ drab males per female[9]. On one archipelago of Caribbean reefs, gaudy males fertilize a proportion $Y = 0.55 \pm 0.06$ (estimate ± SE thereof) of all spawns, while there is 0.52 ± 0.03 drab male per female. For an eastern Pacific congener of the bluehead, gaudy males fertilize far less than 1% of the spawns, and there are roughly as many drab males as females[58]. In a variety of populations, the relation between the proportion of bluehead wrasses born male and the proportion of matings secured by gaudy males agrees, more or less, with theory[9].

Pandalid shrimp reproduce once a year. In most populations, all individuals are female by their second reproductive season. The number $b(x)$ of eggs a female produces in her x^{th} reproductive season is roughly $n[1 - \exp - k(x - x_0)]^3$, where K, x_0 and n differ among populations[7]. If an individual born male fertilizes $b(1)$

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eggs, and if a proportion L of these shrimp survive from one reproductive season to the next, then, since the number of eggs fertilized per newborn equals the number produced, the proportion W of shrimp born male is such that

$$Wb(1) = (1 - W)b(1) + b(2)L + b(3)L^2 + b(4)L^3 + \ldots$$

The relation between W, death rate L, and growth rate as reflected by K and x_o agrees, roughly, with this prediction[7]: too many inaccurately estimated variates are involved to hope for better. Moreover, fishing increases mortality: theory predicts, and observation confirms, that when mortality increases, these shrimp change sex earlier, and at a smaller size[9].

III) Assumptions underlying sex allocation theory

In this section, we examine the results of abandoning various assumptions of the theory: the constancy of a genotype's sex ratio, the absence of any relation between a mother's survival prospects and the sex ratio of her offspring, randomness of mating, and symmetry of inheritance.

A) Adjustability of the sex ratio

If animals can adjust their sex ratio in response to fluctuating conditions, they will do so. Consider, for example, an insect population with two generations a year, and suppose that no adult can survive the winter, that no female can mate in more than one season, but that males destined to mature in the spring can mate again in the fall. Then selection favors an excess of males among young destined to mature in the spring, for they might be able to mate again in the fall, but an excess of females among young destined to mate in the fall, for females are in shorter supply then[60]. Again, if females born earlier in a breeding season invariably survive better, relative to males, than those born later, selection favors bearing only females early in the season and only males later on, just as if the early breeders were in patches favoring females, and vice versa[3]. If the remaining assumptions of sex ratio theory apply, selection on sex ratio adjustment is unaffected by whether one or both parents, or the offspring, control sex ratio. The optimum sex ratio thus reflects the common interest of the autosomes.

When sex ratio is adjustable, selection on bivoltine insects modifies it in the expected direction[43]. Among Michigan grackles, females born early in the season are more likely to survive, and more of the young born then are female[24]. Because the relative survival probabilities of male and female young vary differently with the progress of the breeding season in successive years[24], theory does not predict a sharp distinction between 'early' reproduction when all young are female, and late reproduction when only males are born[3]. Nevertheless, it is unclear whether grackles could modify their sex ratio in so extreme a manner even if the environment were constant. The uncertainties of what optima are actually attainable, coupled with the difficulties of measuring environmental variation and predicting how it shapes selection on the sex ratio, have so far prevented precise tests of predictions concerning sex ratio adjustment.

B) Sex ratio and maternal mortality

What happens if the mother's survival prospects depend on the sex ratio of her offspring? We do not know any animals for which this is true: our concern with the subject is to show how crucial to sex ratio theory is the assumption that sex ratio and parental mortality are unrelated.

Suppose females each produce one young a year, and that a female bearing a son has probability S'_F, and one bearing a daughter, probability $S''_F = S_F - E$, of surviving the year. A mother producing only males survives an average of $1/(1 - S'_F)$ years, bearing that many sons, while a mother producing only daughters bears an average of $1/(1 - S''_F)$ daughters. Thus a male birth costs as much as $(1 - S'_F)/(1 - S''_F)$ female births.

First, let males control sex ratio, and let B males father k sons per 1 − k daughters, while A males father k + d sons per 1 − k − d daughters. The average survival rates of A and B mothers are the same, for they have equal chance of mating with A males, and thus equal sex ratios. The assumptions of standard sex ratio theory apply here, and selection on the fathers favor k = ½ despite the different costs of the two sexes to the mother.

If mothers control the sex ratio, then the fraction of sons produced is greater by d, and the survival rate accordingly greater by $d(S'_F - S''_F) = dE$, for A than for B females. Let S_{BF} and $S_{AF} = S_{BF} + dE$ be the average survival rates of A and B females respectively. Now let A be very rare, and d very small, and let A's frequency be in equilibrium, so that $N_{AF}(t + 1)/N_{BF}(t + 1) = N_{AF}(t)/N_{BF}(t)$, and similarly for males. Then the average of A's frequency among male and female recruits of year t + 1 is

$$\frac{N_{AF}(t+1) - (S_{BF} + dE)N_{AF}(t)}{N_{BF}(t+1) - S_{BF}N_{BF}(t)} + \frac{N_{AM}(t+1) - S_M N_{AM}(t)}{(N_{BM}(t+1) - S_M N_{BM}(t)} = Q_A(t) - \frac{\frac{1}{2}Q_A dE}{1 - S_{BF}}.$$

However, A's frequency among these recruits must be $Q_A + Q_A d(\frac{1}{2} - k)/2k(1 - k)$, just as it would be if these recruits comprised the whole population. Thus

$$-\tfrac{1}{2}Q_A dE/(1 - S_{BF}) = Q_A d(\tfrac{1}{2} - k)/2k(1 - k) \approx \tfrac{1}{2}(\tfrac{1}{2} - k)dQ_A;\ k \approx \tfrac{1}{2} + \tfrac{1}{4}E/(1 - S_{BF}).$$

If male births cause less risk of death to the mother, selection on mothers favors an excess of sons, creating a conflict of interest with the fathers, for whom selection favors an equal sex ratio[9]. Correlation between sex ratio and maternal survival thus destroys the community of interest of autosomal genes in the sex ratio.

C) Randomness of mating

What happens in populations whose members do not mate at random? Mating is never truly random. In most populations, individuals are more likely to mate with their neighbors, giving rise to 'traitgroup' or 'neighbor' selection. We accordingly ask how such selection can be expected to affect sex ratio, even though our knowledge of mating patterns is rarely precise enough to test exact

predictions of the relation of mating pattern to sex ratio. Consider a population of sexual haploids, where successive generations are distinct, mated pairs of which are distributed over a rectangular lattice, one pair at each point (m, n). Following Bulmer and Taylor[6], let the sex ratio locus be occupied by an allele B, whose mated pairs produce Sk sons and W(1 − k) daughters apiece, except for one member of the pair at (0, 0), which is A. Let a son have probability p(b) of dispersing a total of b moves over the lattice – some combination of a moves in the x direction and b − a moves in the y – while a daughter has probability q(b) of dispersing b moves. For b > 0, there are 4b lattice points b moves away from a given mated pair, so the proportions of their sons, and of their daughters, landing at a *particular* point b moves away are p(b)/4b and q(b)/4b respectively. Finally, let one of the males, and one of the females, landing at a lattice point be chosen at random for that point's next mated pair.

Suppose first that individuals control their own sex ratio, and that the mated pair at (0, 0) produces ½S(k + d) A sons, ½W(1 − k − d) A daughters, ½Sk B sons, and ½W(1 − k) B daughters. Then the lattice point (a, b − a) receives ½S(k + d)p(b)/4b A sons and ½Skp(b)/4b B sons from (0, 0) and Sk[1 − p(b)/4b] B sons from elsewhere. Similarly, it receives ½W(1 − k − d)q(b)/4b A daughters and W(1 − k)[1 − q(b)/8b] B daughters in all. The expected number of A genes in the next mated pair at (a, b − a) is the sum of A's frequencies among the sons, and among the daughters, which dispersed there. This is

$$\frac{S(k+d)p(b)/8b}{Sk+Sdp(b)/8b}+\frac{W(1-k-d)q(b)/8b}{W(1-k)-Wdq(b)/8b}$$

$$=\frac{p(b)}{8b}\left[\frac{1+d/k}{1+dp(b)/8bk}\right]+\frac{q(b)}{8b}\left[\frac{1-d/(1-k)}{1-dq(b)/8b(1-k)}\right].$$

If d is small, this is roughly

$$\frac{p(b)}{8b}+\frac{q(b)}{8b}+\frac{p(b)d}{8bk}\left[1-\frac{p(b)}{8b}\right]$$

$$-\frac{q(b)d}{8b(1-k)}\left[1-\frac{q(b)}{8b}\right].$$

Since there are 4b points b moves away, and one point 0 moves away, from (0,0), the total number of A genes in all mated pairs is

$$\sum_{b=0}^{\infty}\left\{p(b)\left[\tfrac{1}{2}+\frac{d}{2k}\right]+q(b)\left[\tfrac{1}{2}-\frac{d}{2(1-k)}\right]\right\}$$

$$-\frac{½A(p)d}{2k}-\frac{½A(q)d}{2(1-k)}$$

$$=1+[1-½A(p)]d/2k-[1-½A(q)]d/2(1-k),$$

where $A(p)=p^2(0)+\sum_{b=1}^{\infty}p^2(b)/4b$ and A(q) is the same save that $q^2(b)$ replaces $p^2(b)$ for every b. Here, the optimum sex ratio is given by $k/(1-k)=[1-½A(p)]/[1-½A(q)]$. Sex ratio is skewed in favor of the better dispersed sex.

Suppose now that sex ratio is controlled by the mother, and that the A-bearer at (0, 0) is female. Now the point (a, b − a) receives ½S(k + d)p(b)/4b B sons as well as that number of A sons from (0, 0), and thus Sk + Sdp(b)/4b sons in all, as well as W(1 − k) − Wdp(b)/4b daughters. Here, the next generation's mated pairs include an average of $1+[1-A(p)]d/2k-[1-A(q)]d/2(1-k)$ A-bearers, and the optimum sex ratio is given by $k/(1-k)=[1-A(p)]/[1-A(q)]$, which is twice as skew as before. There is a slight conflict of interest between genes which control the sex ratio of their bearers and those which control the sex ratio of their offspring: nonrandom mating thus blurs the community of interest of autosomal genes in the sex ratio.

If males are equally likely to disperse to any of m lattice points (so that their 'mating radius' is $\sqrt{m}$), and females to any of n, the optimum sex ratio[6] $k/(1-k)=(1-1/m)/(1-1/n)$.

A different mating model assumes that fertilized females aggregate into 'traitgroups' of n individuals apiece to bear their young, which mate at random within their trait-groups: afterwards, the fertilized females join a common pool from which the next generation's trait-groups are formed at random. Here, the optimum sex ratio $k/(1-k)$ is $(1-1/n)/(1+1/n)=(n-1)/(n+1)$ if the mother controls sex ratio[22, 50], and $(n-1)/n$ if the offspring do so individually. The effects of trait-group selection on haplodiploid sex ratios are well known, as we shall see, but for diploids we have no empirical evidence at all.

D) Haplodiploids

Haplodiploids, whose unfertilized offspring are haploid males and whose fertilized offspring are diploid females, provide striking opportunities for testing sex allocation theory. Their asymmetric inheritance causes a striking conflict of interest between mothers and daughters over sex ratio. As daughters can only influence sex ratio in those social species where they provide essential help in raising siblings, the species where daughters win the conflict are predictable. Moreover, in many asocial hymenoptera, mothers adjust their sex ratio to suit their circumstances by 'deciding' how many of their eggs to fertilize. Such animals offer a variety of tests of theory rivalling those in sequential hermaphrodites.

1) The conflict of interest

Consider a population where individuals mate at random, where successive generations are distinct, and where sex ratio is influenced by a single locus with a common allele B and an allele A so rare that it occurs among females only in heterozygous form.

First suppose the mother controls the sex ratio: let BB mothers bear Sk sons and W(1 − k) daughters, while AB mothers bear S(k + d) sons and W(1 − k − d) daughters. If $N_{ABF}(t)$ and $N_{BBF}(t)$ are the numbers of AB and BB females at generation t, then, retaining other notation from earlier sections,

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$$N_{ABF}(t+1) = \tfrac{1}{2}WF(1-k-d)N_{ABF}(t) + WF(1-k)N_{BBF}(t)N_{AM}(t)/N_M(t);$$

$$N_F(t+1) \approx N_{BBF}(t+1) = WF(1-k)N_{BBF}(t);$$

$$N_{AM}(t+1) = \tfrac{1}{2}S(k+d)N_{ABF}(t);\ N_M(t+1) = SkN_F(t).$$

Setting $Q_{AF}(t) = N_{ABF}/2N_F(t)$, $Q_{AM}(t) = N_{AM}(t)/N_M(t)$, we find

$$2Q_{AF}(t+1) = Q_{AM}(t) + Q_{AF}(t)\left[1 - \frac{d}{1-k}\right];$$

$$Q_{AM}(t+1) = Q_{AF}(t)\left[1 + \frac{d}{k}\right].$$

The correct way to solve these equations[55] is to find s and v such that

$$Q_{AM}(t+1) + vQ_{AF}(t+1) = (1+s)[Q_{AM}(t) + vQ_{AF}(t)].$$

The quick and dirty solution, which yields the same answer, is to say that females count twice as much because they carry twice as many genes, and write

$$2Q_{AF}(t+1) + Q_{AM}(t+1) = Q_{AM}(t) + 2Q_{AF}(t) + Q_{AF}(t)\left[\frac{d}{k} - \frac{d}{1-k}\right].$$

Mothers favor spending equal effort on each sex.

Now suppose that daughters control sex ratio: let matings yielding only AA daughters produce $W(1-k)$ daughters and Sk sons, while matings yielding only AB daughters produce $W(1-k-d)$ daughters and $S(k+d)$ sons, and matings yielding both produce $W(1-k-\tfrac{1}{2}d)$ daughters and $S(k+\tfrac{1}{2}d)$ sons. Then

$$N_{ABF}(t+1) = WF[\tfrac{1}{2}N_{ABF}(t)(1-k-\tfrac{1}{2}d) + N_{BBF}(t)(1-k-d)Q_{AM}(t)];$$

$$N_{AM}(t+1) = \tfrac{1}{2}S(k+\tfrac{1}{2}d)N_{ABF}(t);$$

$$2Q_{AF}(t+1) + Q_{AM}(t+1) = 2Q_{AF}(t) + Q_{AM}(t) + \tfrac{1}{2}Q_{AF}(t)\left[\frac{d}{k} - \frac{d}{1-k}\right] - Q_{AM}(t)\left(\frac{d}{1-k}\right).$$

If $Q_{AM} = Q_{AF}$, as is nearly true when d is small, then the sex ratio is optimum when $d/k - 3d/(1-k) = 0$ and $(1-k)/k = 3$. Daughters favor spending three times as much effort on sisters as brothers[7, 52, 55].

In general, solitary, outbreeding hymenoptera spend equal effort on each sex[9, 22]. The parasitic wasp, *Pachysomoides stupidus*, puts mostly males or mostly females into any single host, apparently to prevent the faster-growing female larvae from suppressing the growth of their brothers, but in all, half its young are male[41]. By contrast, it appears that at least some outbreeding ants whose workers (female offspring) raise their siblings, spend roughly three times as much effort raising daughters as sons[52], although further work is needed on this topic.

2) Sex ratio adjustment

Fig wasps provide perhaps the most striking example of sex ratio adjustment, in response to traitgroup selection. A certain number of fertilized females enter a fig fruit: their young grow up and mate at random within that fruit, and the fertilized females sally forth to colonize fruit on other fig trees[12]. Each species of fig tree has just one species of fig wasp[12]: these species differ in average number of foundresses per fruit. In a wasp where the harmonic mean of the number of foundresses per fruit is n, Herre[23] has calculated that the optimum proportion k* of sons among the offspring of a female colonizing a fruit with $m-1$ similar foundresses is $(2n-1)(m-1)/(4n-1)m$. k* is smaller, the smaller m, and for fixed m, k* is smaller, the smaller n. Herre[23] finds that three species of Panamanian fig wasp with adjustable sex ratio obey the theory except when there is only one foundress, who must provide enough sons to ensure the fertilization of her daughters (table). Charnov[9] has reviewed examples of sex ratio adjustment to other factors, again finding agreement with theory.

Some find it ironic to invoke sex ratios reflecting 'voluntary decision' as evidence of adaptation, but if the decisions are demonstrably those which best propagate the mother's genes within the population, they testify to the decisive importance of selection within populations.

E) Sex chromosomes and sex ratio distortion

Asymmetry of inheritance is particularly marked in the sex chromosomes. In diploids, the 'odd chromosome' of the heterogametic sex (for heterogametic males, the Y chromosome) shares no genes with its homogametic counterpart. Theory predicts that a Y chromosome gene can spread through its population by biassing segregation at meiosis in favor of its bearer chromosomes[22, 38], even if the population dies out as a result. The homogametic sex chromosome can also spread through an appropriate meiotic bias against Y chromosomes, but a given bias spreads the X only a third as fast, as only a third of these chromosomes occur in the heterogametic sex[22].

As we might expect, selection on sex chromosome loci for the sex ratio bears no relation to the common interest of autosomal genes. Lyttle[35] translocated a segregation-distorter locus onto the Y chromosome of *Drosophila melanogaster*, and observed instances where the distorter spread through entire populations, causing their extinction. Extrachromosomal factors also spread without regard to the common interest of the genome. In experimental populations of a parasitic wasp, a factor, inherited through the father, which causes his offspring to be nearly all male without reducing brood size, increases to high frequency when introduced[61]. In this same wasp, a bacterium which is passed on from mother to daughter, kills males before they hatch, perhaps because this enhances female survival[46]. Finally, a maternally inherited extrachromosomal factor in this wasp causes her offspring to be entirely female[45]. The spectrum of asymmetrically inherited sex ratio factors in this wasp begins to remind one of the 'war of all against all'.

How is this anarchy resolved? In populations infected by sex ratio distorters, whether genetic or extrachromosomal, any autosomal modifier that suppresses the distortion effect spreads, because it assures *its* offspring an advantageous sex ratio[38], just as selection favors modifier alleles suppressing the effects on meiotic segre-

gation-ratio of a distorter which is thereby spreading a phenotypic defect through the population[42]. Moreover, selection must favor those *species* where autosomal loci have enforced the common interest of the genome with respect to sex ratio[29]. At least among those species living today, this common interest appears to have prevailed. Genes carried by sperm are inactive, as if to prevent 'sperm competition' and the like from distorting the results of meiosis[13,22]. Y chromosomes, which would otherwise be most susceptible to selection for sex ratio distortion, use usually inert[22], even in recently evolved systems of sex determination[63]. The absence, in many populations, of genetic variation in sex ratio[64] may itself reflect how effectively selection for the 'common interest of the genome' has eliminated ways of tampering with the sex ratio.

Indeed, there is experimental evidence for the efficacy of the common interest of the autosomes. When Lyttle[35] translocated a distorter locus onto the Y chromosome of *Drosophila melanogaster* and inoculated these mutants into cages of normal flies, he found that populations which survived the introduction of these distorter Y responded in two ways. Most populations accumulated polygenic suppressors of the Y chromosome distorter, each suppressor slightly decreasing the strength of the sex ratio distortion[36]. After 350 days sex ratios, though still very skew, were significantly closer to 1:1 than when the distorter had first spread through the population. In one population, aneuploid genotypes, XXY and XYY, appeared, and attained frequencies such that two females were now being born for every three males, even though the distorter Y had completely replaced the normal one[37].

Conclusions

For most outbreeding organisms, theory predicts that selection at all autosomal loci favors the same allocation of reproductive effort between male and female offspring or function. Even though selection on sex chromosomes favors different, conflicting schemes of sex allocation, it is the common interest of autosomal genes to suppress such 'outlaw' schemes. Mating with near neighbors, or effect of sex ratio on maternal mortality, however, blurs the common interest of autosomal genes with respect to sex ratio, while asymmetric inheritance completely destroys it, as in haplodiploids.

This theory is reasonably successful. There are problems. In populations for which the theory's predictions are simplest, there is no genetic variation in sex ratio, so the theory seems irrelevant. Factors which skew the sex ratio quite markedly are sometimes quite common. In many populations with obvious genetic or behavioral variation in sex allocation, predictions of the theory are affected by that multiplicity of relevant variables which makes so many biologists despair of bringing their work within the realm of the exact sciences. Yet most animal populations with separate sexes bear roughly equal numbers of sons and daughters. In a few well-studied populations, such as bluehead wrasses and fig wasps, patterns of sex allocation match theoretical prediction so precisely that the theory 'must be close to the truth'. Lyttle's experimental test of sex ratio theory[35-37] confirms the likelihood that, given time, the common interest of the genome will prevail over challenges from outlaw genes. Moreover, the theory correctly specifies the origin of conflicts of interest concerning sex allocation such as outlaw genes represent. What does sex allocation reveal about which factors direct evolutionary process? It seems difficult to say without knowing what increases the fitness of species. Discussions of 'the good of the species' tend to emphasize the avoidance of extinction, neglecting speciation. Thus sexual selection is called 'bad for the species' because it can favor characteristics which hasten extinction[28], even though sexual selection enhances speciation, as Darwin knew[62]. Nevertheless, the good of traitgroups is clearly enhanced by sex allocation favoring females or female function[11], and this must also be true for most species.

Sex allocation theory, however, bases its predictions exclusively on selection within populations, even though it allows for the influence thereon of the aggregation of individuals into traitgroups. This theory's success shows that selection between populations rarely overrides selection within populations[30]. The rigidity of sex ratio in many populations suggests, however, that selection has favored those *species* least susceptible to 'infection' by outlaw genes: thus selection between species sometimes reconciles individual advantage with the good of the group.

It thus appears that a theory whose main triumphs concern the loves of coral reef fish and the curious sex ratios of fig wasps tells us rather a lot about which factors direct evolution.

Note added in proof: For an alternative approach the interested reader should also see: Frank, S. A., Fla. Ent. *66* (1983) 42–75; and Parker, E. D., and Orzack, S. H., Genetics *110* (1985) 93–105.

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