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Sex determining mechanisms: An evolutionary perspective

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Summary. Theories on the evolution of sex determining mechanisms are reviewed for male and female heterogamety, environmental sex determination, and briefly, haplo-diploidy and hermaphroditism. Because of their discrete and well-defined nature, sex determining mechanisms lend themselves to three types of evolutionary questions: *what* variety occurs and might be expected but does not occur, *how* do changes occur from one mechanism to another, and *why* do certain changes occur? All three approaches were illustrated for these different sex determining mechanisms. A generality emerging from these studies is that, at the level of selection on the sex ratio, there are no intrinsic problems in evolving from one sex determining mechanism to another: straightforward transitions between different mechanisms exist under various conditions.

Key words. Sex; sex determination; sex ratio; evolution; animals; genetics.

1) Introduction

The beginning of this century marked the onset of studies on the inherited and environmental basis of sex, and a variety of sex determining mechanisms are now known in plants and animals*. The two major categories of sex determining mechanisms are dioecy (separate sexes) and hermaphroditism (both sexes within the same individual), but there is further variety within both of these classes. Considering dioecious species, the most widespread mechanism is *heterogamety,* in which one sex is labeled XX and the other XY. Systems slightly more complicated that this, involving three or more factors, are also known. In addition, some species are haplo-diploid, with females arising from fertilized eggs, males from unfertilized eggs. In contrast to these systems, some dioecious species have environmental sex determination, whereby sex is determined in response to environmental effects experienced early in life. With hermaphroditic species, the distinction between male and female is not inherited as such, because both sex types occur in each individual (whether sequentially or simultaneously). However, the extent of male/female expression may be subject to genetic and environmental influences[20].

The diversity of sex determining mechanisms poses an interesting set of questions to study from an evolutionary perspective[3, 13, 28, 48, 53]. As with any set of biological problems, three types of evolutionary questions may be addressed: what variety occurs, how the variety comes about, and why changes occur. All three levels of investigation are especially appropriate for sex determining mechanisms. For example, at the simplest level, we may consider *what* diversity of mechanisms is actually present. Understanding the existing diversity is chiefly a matter of observation, but more generally, to understand what mechanisms occur, it is often useful to first develop a conceptual framework for the larger set of theoretically possible mechanisms. Observations may then be made to reveal which mechanisms actually occur in nature, and which do not. Sex determining mechanisms are amenable to this analysis because they fall into discrete categories (heterogamety, haplodiploidy, and so forth), so the realm of possibilities is easily calculated.

The observed variety of sex determining mechanisms may then be studied to consider *how* the variety comes about. For example, how might male and female heterogamety arise in closely related species? Are the transitions straightforward or do they require fortuitous combinations of population structure and random effects? Along with investigating how different mechanisms evolve, it is rewarding to consider *why* they evolve: are some mechanisms selectively favored over others? The three evolutionary questions – what, how, and why – may be applied separately to the study of sex determination, but a perspective combining all three levels can lead to a particularly enriched paradigm of study.

This paper briefly reviews some recent work on the evolution of sex determining mechanisms along the lines of the above framework. This review aims to illustrate the different approaches outlined above, and is confined chiefly to two mechanisms, heterogamety and environmental sex determination. Some evolutionary problems concerning haplodiploidy and hermaphroditism will be considered briefly as well. Readers interested in further review and development of the ideas presented here are referred to my recent book on this subject[13]. In addition, the question of *why* different sex determining mechanisms may evolve has been considered extensively for the different forms of hermaphroditism versus dioecy in Charnov[20].

2) Sex ratio evolution

A fundamental property of sex determining mechanisms, especially as regards how and why different mechanisms evolve, regards the sex ratio. As first proposed by Fisher[32], there are a wide variety of conditions selecting a primary sex ratio of ½ in the population (primary sex ratio is the proportion male at conception). Consequently, sex determining mechanisms causing the population sex ratio to deviate from ½ will usually be selected against. This principle is fundamental to the following presentation and so will be elaborated here.

Fisher's argument may be explained as follows. Assume that a population mates randomly, and consider the number of offspring expected from a male zygote versus a female zygote. Suppose, for example, that a male zygote has a greater expected number of children than a female zygote: then selection will favor those genotypes producing an excess of males. Genetic variation permitting, the sex ratio will evolve to the point that a male and a female zygote each have the same expected number of children. The relative reproductive success of a male versus a female depends on the population sex ratio. If males and females have identical survival rates and ages of maturity, then equivalence of male and female individual reproductive success is clearly achieved with equal numbers of male and female zygotes (a primary sex ratio of ½). This equivalence is true even with polygamy or any other factor that merely contributes variance to individual reproductive success, since the *average* number of children per male and per female will remain the same. (For example, if half the males acquire two mates and the other half acquire none, the average number of mates per male is ½ by 2, which equals unity, just as if each male acquired one mate.)

Fisher's result holds even with differential mortality of the sexes. Consider a male zygote in a population with a primary sex ratio of ½. If the probability of a male surviving to maturity relative to females is s, an *adult* male has an average of $1/s$ offspring times that of an adult female. Since the factors s and $1/s$ cancel, a male and a female zygote have equal average reproductive success despite the differential mortality. Fisher's result does not apply in some special circumstances[20], but his result appears to be general enough that sex determining mechanisms should often be selected accordingly.

3) Evolution of heterogametic mechanisms

Heterogamety is the simplest sex determining mechanism with a consistent genetic difference between males and females: one sex is designated XX (homogametic) and the other XY (heterogametic). The X and Y influence the inheritance of sex in that any zygote inheriting Y develops as one sex, while a zygote without Y develops as the other sex:

$$\begin{tikzcd} & \text{Sex} & & \\ \underline{1} & & \underline{2} & \\ \text{XX} \arrow[d] \arrow[drr] & & \text{XY} \arrow[d] \arrow[dll] & \text{Parents} \\ \text{XX} & & \text{XY} & \text{Offspring} \end{tikzcd} \tag{3.1}$$

The X and Y are referred to as sex *factors*. (A sex factor is generally any gene or gene combination that influences the inheritance of sex.) Heterogamety is therefore more generally referred to as a two-factor mechanism. The notation ZZ/ZW is often used for female heterogamety, but in this review, XX/XY will apply to male and female heterogamety unless indicated otherwise. The symbol Y therefore also represents the W of female heterogamety, and X represents the Z. Species in which Y (or X) is absent are also consistent with this notation, since the labels X and Y serve only to represent the inheritance of sex.

Heterogamety is commonly identified by any of three means[1, 3, 28]: a) The sex factors may be inherited as part of *heteromorphic* sex chromosomes that are detectable cytologically (the sex chromosomes themselves also being designated as X and Y). b) Allelic variation at sex-linked

loci segregates in the fashion depicted in (3.1), so if this variation is observed, its segregation infers heterogamety.
c) Various means, artificial or natural, may be used to cause XY to develop into the sex that is commonly XX; XX may also be induced to develop into the sex that is commonly XY. The sex ratios from crosses of these 'sex-transformed' individuals with normal individuals distinguishes male heterogamety from female heterogamety and may be used in part to infer heterogamety. For example, XX males may be produced in a species with male heterogamety. The mating of these atypical XX males with normal, XX females produces all daughters and is consistent with male, but not female, heterogamety.

Heterogametic sex determination is widespread, occurring in some of the few dioecious flowering plants, in invertebrates such as insects, nematodes, and arachnids, and occurring in the vertebrates classes of fish, amphibians, reptiles, birds and mammals. The vast majority of cases have been identified only through cytological studies of sex chromosomes. Both male and female heterogamety occur in some groups (Diptera, lizards, amphibians; table 1), only male heterogamety occurs in others (e.g. mammals, arachnids, and nematodes), and only female heterogamety occurs in yet others (birds, snakes, Trichoptera) (reviewed in Bull[13]).

Variety and possible variety of mechanisms

If XX is strictly one sex and XY is strictly the other sex, only two heterogametic mechanisms are possible as regards these genotypes, male and female heterogamety. As noted above, both systems are known in animals, although some major taxonomic groups contain only male or only female heterogamety. Heterogamety has also been studied in other respects: the sex of YY individuals and the sex of aneuploid XO and XXY individuals, and these studies have revealed a wider variety of mechanisms than is evident from merely characterizing the heterogametic sex.

The sex of YY

The sex of YY is property of heterogametic sex determination that usually remains hidden because YY is not produced in the population: Y occurs only in the heterogametic sex, hence no zygote can inherit Y from both parents. However, various environmental effects such as temperature, hormones, or tissue transplants have been used to transform the phenotypic sex of XY, and YY can then be produced from matings of XY males with XY females. This analysis is limited to species in which the X and Y are not highly differentiated, since YY individuals are generally inviable in species with degenerate Y chromosomes.

There are two possible outcomes in this analysis: YY and XY are the same sex, or YY and XX are the same sex. Of the heterogametic systems studied in this respect (table 2), YY and XY are invariably the same sex, as first discovered by Aida for the medaka fish[1]. This conformity between different species is expected whether the X is a recessive female-determiner or the Y is a dominant male-determiner, or any intermediate scheme of this sort. However, there is no a priori basis for supposing that YY is never the same sex as XX, and there is no explanation of why nature consistently follows this one pattern. (A mechanism that operates on the principle that XX and YY are the same sex is in fact known in Hymenoptera; this mechanism operates with many sex factors under haplodiploidy, hence was not considered along with these heterogametic mechanisms.)

The sex of XO and XXY

Two possible interpretations of heterogametic sex determination are a) that the Y is a dominant inducer of the heterogametic sex, or b) that the X is a recessive inducer of the homogametic sex[6]. Many other models are possible for the mode of action of X and Y, but these two models have had the appeal that they draw on genetic principles taken across a variety of characters from many species (recessivity, dominance, additivity). Both interpretations

Table 1. Some groups exhibiting variety in the heterogametic sex

Taxon	Heterogametic sex Male	Female
1) Order Diptera (flies)		
F. Anthomyidae	+	−
F. Calliphoridae	+	−
F. Chironomidae	+	+
F. Culicidae	+	−
F. Drosophilidae	+	−
F. Muscidae	+	+
F. Phoridae	+	−
F. Simulidae	+	−
F. Tephritidae	+	+
F. Tipulidae	+	−
2) Class Amphibia		
Order Anura (frogs)		
F. Bufonidae	−	+
F. Discoglossidae	+	−
F. Hylidae	+	−
F. Leptodactylidae	+	−
F. Pipidae	−	+
F. Ranidae	+	+
Order Urodele (salamanders)		
F. Ambystomatidae	−	+
F. Plethodontidae	+	+
F. Proteidae	+	−
F. Salamandridae	+	+
3) Suborder Lacertilia (lizards)		
Infraorder Gekkota		
F. Gekkonidae	−	+
F. Pygopodidae	+	−
Infraorder Iguania		
F. Iguanidae	+	−
Infraorder Scincomorpha		
F. Lacertidae	−	+
F. Sincidae	+	−
F. Teidae	+	−
Infraorder Platynota		
F. Varanidae	−	+

Sources: Bull[13], King[42], Schmid[68], White[77]. F=family. + indicates that the indicated mechanism of heterogamety is know from at least one species in the taxon; − indicates that the indicated mechanism has not been observed.

are further consistent with the observation that XY and YY are the same sex. To distinguish these alternative models, individuals with the aneuploid genotypes XO and XXY have been studied (the individuals possessing a normal diploid complement of autosomes). Whereas the study of YY individuals was feasible only in species lacking heteromorphic sex chromosomes, the study of XO and XXY individuals is practical only with heteromorphic sex chromosomes, because the aneuploids are otherwise difficult to generate and to identify. The first such study was undertaken by Bridges on *Drosophila*[8,9]. Considering the sexes of XO and XXY, there are actually four possibilities (assume male heterogamety):

		XO	
		♂	♀
XXY	♂	a	b
	♀	c	d

The observations, ranging from mammals to plants, are again confined to only half the possibilities (table 3): a) recessive-X systems (c above): individuals with a single X are one sex, those with two XX's are the other sex, regardless of the Y. b) Dominant-Y systems (b above): individuals without a Y are the same sex, those with a Y are the other sex, regardless of the number of X's. Thus, even though not all possibilities have been observed, some variety among heterogametic mechanisms exists that is hidden from studies limited to those genotypes occurring in the population (XX, XY).

In summary, there are three characteristics of heterogametic systems that have been studied: the heterogametic sex, the sex of YY, and the sexes of aneuploids, XO and XXY. Only the heterogametic sex is evident in the context of the natural population; the other characteristics are observed only with special studies. These special studies have revealed a further variety of mechanisms than previously known, but some types remain to be discovered.

Table 2. The sex of YY individuals

Taxon	Sex of		
	XX	XY	YY
Fishes			
Oryzias	f	m	m
Xiphophorus	f	m	m
Poecilia	f	m	m
Carassius	f	m	m
Amphibians			
Xenopus	m	f	f
Pleurodeles	m	f	f
Ambystoma	m	f	f
Hyla	f	m	m
Dipterans			
Musca	f	m	m
Crustaceans			
Orchestia	f	m	m
Armadillidium	m	f	f
Plants			
Thalictrum	f	m	m
Asparagus	f	m	m
Mercurialis	f	m	m

Source: Bull[13], original references and additional details are reported there. m=male, f=female.

How and why the variety evolves

The study of variety, amid the larger set of possibilities, is interesting in itself, but it further inspires research into the processes influencing the observed variety. Because of their nature, sex determining mechanisms lend themselves to two questions regarding the evolution of variety: how does the change occur from one mechanism to another, and also, why does the change occur? In addressing the first question (how), there are obviously many possibilities that should be considered: a new heterogametic mechanism might evolve from a dioecious ancestor or might evolve from a hermaphroditic one[18,76]. Both of these alternatives in turn offer many possible ancestral mechanisms. For example the dioecious ancestor of a novel heterogametic mechanism may have been characterized by environmental sex determination, polyfactorial sex determination, haplodiploidy, or even a different form of heterogamety.

All of these transitions are in fact theoretically possible, but the most common transition in many groups is probably from one heterogametic mechanism to another heterogametic mechanism. The transition, which in principle could involve a variety of intermediates, probably occurs most often through a simple intermediate involving few sex factors – a multiple-factor system. Furthermore, the transition is intrinsically easy[14], as elaborated below.

A simple example is the multiple-factor system (3.2) first described by Beermann[6] in the midge *Chironomus tentans*,

Female	Male
bb XX	bb XY
	Mb XX .

(3.2)

This system represents the segregation of sex factors at two regions in the genome (e.g. separate loci). The factors *b* and *M* segregate opposite each other, as do X and Y. If we imagine that the ancestral heterogametic mechanism was *bb XX*/*bb XY*, this multiple-factor system could have arisen in one step, by a mutation changing *b* into *M*.

Table 3. The sexes of XO and XXY aneuploids

Taxon	Sex of			Interpretation
	XY	XO	XXY	
Mammals				
Homo (man)	m	f	m	Dom-Y
Mus (mouse)	m	f	m	Dom-Y
Diptera				
Drosophila	m	m	f	Rec-X
Musca	m	f	m	Dom-Y
Phormia	m	f	m	Dom-Y
Lucilia	m	f	–	Dom-Y
Pales	m	–	m	Dom-Y
Glossina	m	m	f	Rec-X
Lepidoptera				
Bombyx	f	m	f	Dom-Y
Plants				
Melandrium	m	–	m	Dom-Y
Rumex	m	–	f	Rec-X

Source: Bull[13], original references and additional details therein. m=male, f=female, – = not studied.

The population genetics of this multiple-factor system is straightforward. Only two matings are possible. The mating of *bb XX* with *bb XY* behaves like male heterogamety XX/XY; the mating of *bb XX* with *Mb XX* behaves like male heterogamety bb/bM. If the two male genotypes have equal fitnesses (i.e. equal viabilities, fertilities, and attractiveness to females), both may coexist at any intermediate frequency. For example, if the frequency of *bb XY* males is designated as p (and the frequency of *Mb XX* males 1-p), the frequency of *bb XY* in the next generation is symply

$$p' = \frac{p/2}{p/2+(1-p)/2)} = p. \tag{3.3}$$

Hence any value of p between zero and unity is an equilibrium, and this multiple-factor system consequently has a continuous path of equilibria connecting a system of pure male heterogamety for XX/XY to a system of pure male heterogamety for bb/bM[14].

The change from one heterogametic mechanism to another may occur for either of two reasons, chance and selection. Random changes in gene frequency may cause loss of Y or loss of *M*, for example. Alternatively, an advantage to one male genotype over the other – even a very slight advantage – will cause an increase in its frequency, and if the advantage persists, the favored male genotype will eliminate the other (subject to random effects). When fitness differences are introduced, there is no longer a path of equilibria (as occurred in the equal fitness case), and evolution proceeds toward the genotype of higher fitness. Thus the change from one heterogametic mechanism to another is not difficult in this system. It is easily noted that the change from *bb XY* males to *Mb XX* males involves a change in the locus of heterogamety and could also involve a change from a dominant-Y to a recessive-X system, or the reverse.

Other multiple factor systems are possible. An interesting one was discovered by Kallman[39,40] in the platyfish,

Female	Male
XX	XY
FX	
FY	YY

(3.4)

In this system, all three factors (F , X, and Y) assort in opposition; they are apparently undifferentiated sex chromosomes, because YY platys are viable and fertile. Again, starting with an ancestral system of XX/XY (or FY/YY), this multiple factor system could arise with a single mutation.

The population genetics of the platyfish system (3.4) is only slightly more complicated than in the midge multiple-factor system (3.2). With three female genotypes and two male genotypes, there are six possible matings. One of these matings constitutes male heterogamety, XX · YY, and another constitutes female heterogamety, FY · YY. Assuming equal fitnesses of all genotypes within a sex, there is again a continuous path of equilibria between the system of male heterogamety and that of female heterogamety. The sex ratio along this path is ½, even though one mating (FX · XY) produces an excess of daughters and another mating (XX · YY) produces all sons[14]. It seems remarkable that such a system can abide by the Fisherian sex ratio constraint along an entire path from male to female heterogamety.

There is no intrinsic problem in evolving from male heterogamety to female heterogamety or the reverse in the platy system. Random fluctuations in gene frequency may cause the loss of F or X and thereby establish heterogamety. In addition, certain combinations of fitness differences among the different genotypes also select pure heterogamety. The direction of change in these cases is not always from the ancestral mechanism to a new one; instead the multiple-factor system may degenerate to the ancestral mechanism, but many combinations do lead to the new system. However, the platy system has one property that was absent from the midge system: the possibility of an internal equilibrium in which F, X, and Y are all maintained by selection[14,56]. Thus selection does not invariably lead to the degeneration of a multiple-factor system[14,56,71].

The platy and midge systems illustrate how one heterogametic mechanism may evolve to another. Starting with a single mutation, a multiple-factor system is generated in which a second heterogametic mechanism occurs. Evolution of this new mechanism depends on viability and fertility differences among genotypes and on random fluctuations in gene frequencies, but only slight fitness effects of the right combinations are sufficient to enable the new heterogametic mechanism to evolve. Both of the multiple-factor systems studied above have the property that, with equal fitnesses and no random effects, a path of equilibria connects the two heterogametic mechanisms. The points on this path are no longer equilibria when fitness differences or random effects are introduced, but the region of the gene-frequency space near this path serves as a 'corridor' along which evolution proceeds toward equilibrium. Thus, the existence of a path of equilibria connecting two heterogametic mechanisms in the equal fitness case seems to reflect a simple transition between them when fitness differences and random effects are introduced.

The question remains as to whether the platy and midge multiple-factor systems are typical of other possible multiple-factor systems. This problem is investigated in the following way. First, the set of multiple-factor systems that are theoretically possible is enumerated; this enumeration necessarily depends on the number of sex factors and 'loci' allowed. Second, each of these theoretically possible systems is studied with the methods of population genetics to determine whether a feasible transition exists from one heterogametic mechanism to another. A study of this sort has been conducted for some simple classes of multiple-factor systems, and the properties described for the platy and midge systems are common to many, but not all, of the possibilities[13,70,71]. Cotterman[26] offered the first enumeration of multiple-factor systems relevant to these problems, and Scudo[70,71] provided some of the first theoretical investigations of the evolutionary maintenance of multiple-factor systems; all three papers offer many important insights to the study of sex determining mechanisms. (Cotterman's enumerations were not cast specifically within the narrow framework of sex determination.)

Evidence

The foregoing text offered a framework in which to address the evolution of heterogametic sex determination: the transition from one mechanism to another proceeds through a multiple-factor intermediate. In principle, the transition could occur through other intermediates, such as polyfactorial sex determination, environmental sex determination, and haplodiploidy. Any theory for the evolution of heterogamety must therefore consider whether these other intermediates are important.

The present evidence suggests that changes in the heterogametic mechanism usually occur through multiple-factor intermediates, but the evidence is weak, due chiefly to a lack of knowledge about the transitions. Two types of evidence provide the most powerful tests of the theory: a) observed changes from one mechanism to another, and b) hybridization of closely related populations differing in the heterogametic mechanism (the hybrids may reveal the mechanism that was present during the transition[2]). The actual transition from one form of male heterogamety to another was demonstrated in a laboratory population of houseflies by Kerr[41]; the transition occurred via the midge multiple-factor system (3.2). This study incorporated selection of DDT resistance, and the linkage of a resistance factor with *M* rendered the *Mb XX* males more fit than the ancestral, *bb XY* males. No other observations of a transition from one heterogametic mechanism to another have been reported.

Populations with different heterogametic mechanism have been hybridized to study sex determination in houseflies and midges[45–47, 67]. The observations are usually consistent with multiple-factor inheritance (i.c. only a few sex factors), but the multiple-factor systems have rarely been characterized in detail.

The present theory also predicts that multiple-factor systems should sometimes be observed in taxonomic groups exhibiting a variety of heterogametic mechanisms; alternative possible intermediates should be rare. Of the many theoretically possible multiple-factor systems, only two systems have been characterized in detail from natural populations, the midge system (3.2) and the platy system (3.4). The midge system, and simple extensions of it, have been reported in midges, mosquitoes, phorids, and houseflies[6, 37, 45, 47] (reviewed in Bull[13]). The platy system is known in platyfish and in lemmings, except that YY is inviable in lemmings[33, 34, 39, 40]. Some other probable multiple-factor systems have been partially characterized from dipterans, but the systems seem to be somewhat more complicated than (3.2) and (3.4), and full characterizations have been difficult[6, 37, 45, 47, 72] (reviewed in Bull[13]). The observations are therefore again consistent with the theory at the level of rigor permitted by the data, since changes in the heterogametic mechanism have occurred moderately often in *Xiphophorus* and Diptera[40] (table 1, above).

Studies on the nature of sex transformer mutations in nematodes *(Caenorhabditis)*, *Drosophila*, and mammals (man, mouse) add further credibility to the multiple-factor theory[4, 23, 31, 38, 53]. In all taxa, presumed single-locus mutations are known with major effect on the sex phenotype, although the transformations are sterile in many cases. In *Drosophila* and the nematode, the mutations seem to comprise a hierarchy of major genes regulating the sex phenotype. The existence of such genes in these species strengthens the plausibility that mutations of major effect on sex phenotype occur in many other species. Furthermore, the failure to detect other forms of inherited effects on sex (i.e. polygenic influence or environmental effects, although a few sex-transforming mutations are temperature-sensitive) casts doubt on the plausibility that other intermediate systems commonly lead to new heterogametic mechanisms.

Implications

Several implications of the above theory are apparent. First, there do not appear to be predictable advantages of one heterogametic mechanism over another. For example, male heterogamety does not appear to be intrinsically superior or inferior to female heterogamety. The change from one heterogametic mechanism to another may be fortuitous on several levels: random changes in gene frequency may influence the outcome, as may selection on alleles closely linked to the sex factor – a sex factor may be favored because it happened to arise near a beneficial gene (as proposed by Kallman[39, 40] for platyfish).

One general prediciton about the evolution of heterogamety is that systems with heteromorphic sex chromosomes (a degenerate Y) should be less likely to change than systems in which the X and Y chromosomes are similar. For example, the loss of an ancestral mechanism of male heterogamety requires creating XX males and/or YY males and females. YY will be inviable if Y is degenerate, and XX males may be sterile, as in mammals and *Drosophila*. As noted by Ohno, the X chromosome is apparently conserved throughout placental and marsupial mammals; the Z of snakes (female heterogamety) is also apparently conserved[52]. These conserved sex chromosomes are highly differentiated in both groups and are thus consistent with the above hypothesis, although alternative theories are also plausible[13].

An unsolved problem is the ubiquity of heterogamety in contrast to the rarity of multiple-factor systems and other systems with many sex factors (polyfactorial sex determination). When new sex factors arise repeatedly, heterogamety might be expected to yield to systems with more and more sex factors, yet this accumulation does not seem to occur. Some explanations have been proposed[13], but none are at present compelling, and there has been no careful study of this problem with respect to any of the hypotheses.

The present framework is useful in addressing correlations between the heterogametic sex and various other species' characteristics (e.g. the dominant gonad, viviparty, H-Y antigen[49, 51, 75]). When such a correlation can be demonstrated to be meaningful in an evolutionary sense (i.e. when the pattern has evolved independently too often to be ascribed by chance), two general explanations of the association may be considered. On the one hand, the association could be an invariant consequence of heterogamety (of heterozygosity per se); in this case the association with heterogamety should be preserved during the actual transition from one heterogametic system to another. In the platy system, for example, XY males should display the same characteristics as FY and FX females. An alternative explanation is that the association is not necessarily preserved during the evolution to a new mechanism, but that it arises after any new heterogametic mechanism evolves. These two hypotheses were considered in a nice paper on the association between H-Y antigen and the heterogametic sex, with special reference to platyfish: Nakamura, Wachtel, and Kallman[51] found that H-Y expression was confined to males rather than to heterogamety; in this species the male was H-Y positive under both male heterogamety (XX/XY) as well as under female heterogamety (FY/YY).

4) Environmental sex determination

At the other extreme from heterogametic sex determination is environmental sex determination: a zygote develops as male or female depending on an environmental effect experienced early in life. Whereas in heterogamety, a zygote's sex is determined by genotype virtually regardless of environmental conditions, here sex is determined by environmental conditions regardless of genotype. A necessary consequence of environmental sex determination (ESD henceforth) is that there are no consistent genetic differences between males and females. Thus, the term 'heterogametic sex' does not apply to ESD.

Variety

Mechanisms of ESD may be classified according to the environmental factors influencing sex and according to the relationship between those factors and sex ratio, although such categories necessarily lack sharp boundaries. A brief description of some known ESD mechanisms illustrates a considerable variety, however. In virtually all of these cases, the environmental effect on sex has been shown to be due to sex determination rather than to differential mortality of the sexes.

Reptiles

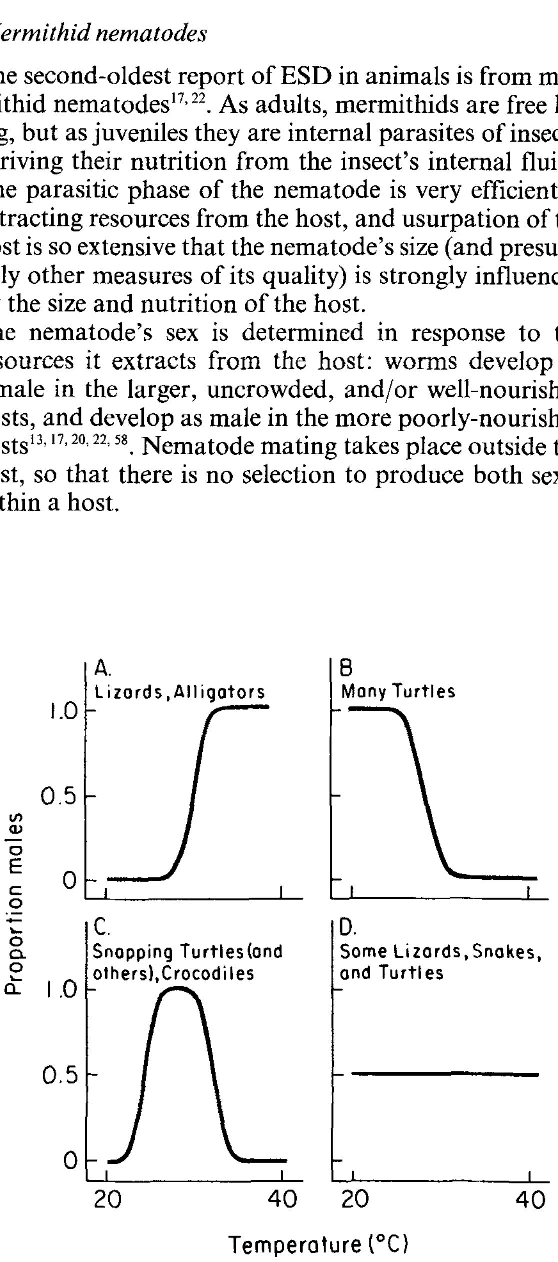
Some of the most recent discoveries of ESD have been in reptiles. The incubation temperature of the egg determines sex in various species of turtles, lizards, and crocodilians (reviewed in Bull[11], Bull[13] and Mrosovsky[50]). The first discovery of this phenomenon was in a lizard by Charnier[19], but the phenomenon was brought general recognition by the work of Pieau on two European turtles[59–62,64], and later by Yntema[79] on another turtle. The temperature influences on sex occur under natural as well as laboratory conditions[64,74]. More recently, the moisture available during development was also shown to influence sex determination in a species with temperature-dependent sex determination, so there may be a wide range of environmental effects on sex, with temperature the predominant one[35].

The magnitude and form of the temperature effect on sex varies between species (figure). In many species a wide range of temperatures produces only one sex, another range produces only the other sex, and a narrow range produces both sexes. However, in some turtles, low temperatures produce males and high temperatures produce females, whereas the reverse pattern occurs in alligators and some lizards. Furthermore, in some other turtles and a crocodile, another pattern occurs in which females are produced at low and high temperatures, males in between. Finally, some species show no temperature effects on sex (which presumably applies to the many reptiles with sex chromosomes, although no direct study of temperature effects has been undertaken in most of these species). Further details of these systems are reviewed elsewhere[13].

Mermithid nematodes

The second-oldest report of ESD in animals is from mermithid nematodes[17,22]. As adults, mermithids are free living, but as juveniles they are internal parasites of insects, deriving their nutrition from the insect's internal fluids. The parasitic phase of the nematode is very efficient at extracting resources from the host, and usurpation of the host is so extensive that the nematode's size (and presumably other measures of its quality) is strongly influenced by the size and nutrition of the host.

The nematode's sex is determined in response to the resources it extracts from the host: worms develop as female in the larger, uncrowded, and/or well-nourished hosts, and develop as male in the more poorly-nourished hosts[13,17,20,22,58]. Nematode mating takes place outside the host, so that there is no selection to produce both sexes within a host.



The relationship between incubation temperature and hatchling sex ratios for various reptiles. Four patterns are observed: *A* Females develop at low temperatures, males at high ones, as in two lizards and alligators. *B* Males develop at low temparatures, females at high ones, as in many turtles. *C* Females develop at both high and low temperatures, males only at intermediate ones; this pattern is known in several turtles and a crocodile. *D* Incubation temperature does not significantly influence hatchling sex ratios in some turtles, snakes, and lizards. Redrawn from Bull[13].

Bonellia

Baltzer's report of ESD in the marine echiurid worm *Bonellia viridis* constituted the first valid case of ESD in animals[5]. The larvae of *Bonellia* are initially planktonic but then settle on the marine substrate to become adults. Adult females are approximately the size of a walnut when contracted but have a long, extensible proboscis (1 m) used to filter-feed; males are tiny, living a parasitic existence in the reproductive tracts of females. Sex is determined when the larva settles from the water column: male development is induced by exposure to females (and also by exposure to female extracts and to various chemical effects); larvae settling in isolation develop as females[5,43].

Silverside fish

The Atlantic silverside, *Menidia menidia,* has temperature-dependent sex determination, although not to the extent of reptiles[24,25]. Eggs are spawned externally, and sex is determined by water temperature late in the larval phase: cool temperatures overproduce females, whereas warm temperatures produce males (as in lizards and alligators). The difference in sex ratio between the temperature extremes is small, only 0.2 to 0.4, and there appear to be large genetic effects on sex determination as well as the environmental ones.

Others

Environmental effects on sex determination are also known in other species, although the resulting sex ratio variations are either small or their importance in natural populations has not been clarified. a) Bulnheim[16] reported that the sex of the marine amphipod *Gammarus* was sensitive to photoperiod; major genetic effects on sex ratio were also evident. b) Laboratory-induced temperature effects on sex determination have been demonstrated in various amphibians that normally have heterogamety[30,63,65]. c) The pH of the water was reported to influence sex determination in various fish[66].

In summary, ESD as a natural mechanism or as a minor component of gentoypic sex determining mechanisms seems to be a widespread phenomenon, but the environmental factors influencing sex vary between species. The following sections will consider why these various cues may be used for sex determination.

How ESD may evolve from GSD

The evolution of ESD from GSD or the reverse poses no intrinsic problem. To illustrate, imagine that a species with male heterogamety experiences an environmental effect on sex such that some XX zygotes become male:

$$\begin{array}{cc} \underline{\text{Female}} & \underline{\text{Male}} \\ XX & XY \\ & XX \end{array} \tag{4.1}$$

The presence of XX males selects a particular frequency of XX and XY zygotes, the actual value depending on the level of environmental influence. XY is selected to lower levels as the influence increases, and XY is selected out of the population if half or more the XX zygotes become male. The sex ratio is ½ at the equilibria, and once again, there is a continuous path of equilibria from one sex determining mechanism to another *if fitnesses are equal* for all genotypes within a sex. Here the path extends from strict GSD (no XX males occur) to strict ESD (all males are XX).

The path of equilibria is somewhat different here than in the multiple-factor systems above. The path of equilibria from heterogamety to ESD is a function of changes in the level of environmental influence, and it might seem that the level of environmental influence is imposed by external conditions and cannot evolve. However, there is in fact a direct parallel with the previous cases: the level of environmental influence on sex determination may evolve through changes in the frequencies of genes modifying an individual's susceptibility to environmental effects on sex. For example, in *Drosophila,* in the nematode *Caenorhabditis,* and in a mosquito, temperature-sensitive sex transformer genes are known that convert XX from male to female[13,27,38]. Increasing the frequencies of these genes increases the frequency of individuals that become XX male in response to environmental conditions. The response to environmental factors can be influenced genetically, and this model for the evolution of ESD from GSD is therefore similar to those of section 3 in that a path of equilibria exists from one mechanism to another through changes in the frequencies of appropriate genes.

Why ESD?

Whereas one type of heterogametic mechanism did not appear to have a systematic advantage over any other (from section 3), there do appear to be relative advantages of ESD and GSD. A model in which ESD is favored over GSD was proposed by Charnov and Bull[21] along the lines of a sex ratio model proposed by Trivers and Willard[73]. The model involves three parts. First, a species inhabits a 'patchy' environment, so that offspring reared in some patches have a different lifetime fitness than individuals reared in other patches. This patchiness further influences male fitness differently than female fitness, some patches being more beneficial to females than males, and other patches doing the opposite. Second, offspring cannot choose patch type, and parents cannot preferentially put offspring of one genotype in patches of one type. Third, mating takes place among individuals reared in different patches.

These three conditions select ESD over GSD[13,15,21]. An individual developing in a patch relatively beneficial to females leaves more offspring if it develops as female, whereas the converse applies to patches beneficial to males. Therefore, if an individual cannot choose patch type, it leaves more offspring by delaying sex determination until it has entered a patch, so that it may become the appropriate sex. Genotypic sex determination is selected against because it causes some individuals to develop as female in patches beneficial to males and vice versa.

Consider now whether GSD might be favored over ESD. The Charnov-Bull model does not offer any advantage for GSD, since the model was based on the premise that

ESD allowed the evolution of sex ratios that GSD could not achieve. However, two possible advantages of GSD can be suggested on other grounds (described here as disadvantages of ESD). First, because sex is determined late in development and in response to environmental conditions, ESD might have two undesirable effects: intersexes might result, and the developmental onset of sexual dimorphisms must be delayed until sex has been determined. A second disadvantage of ESD results from its susceptibility to sex ratio variation. Systematic environmental fluctuations from year to year may cause at least mild fluctuations in sex ratio under ESD. In the absence of the effects described in the Charnov-Bull model above, this sex ratio fluctuation selects GSD[12, 15].

Evidence

Are the observations on ESD consistent with the Charnov-Bull model of a patchy environment? The model requires a) that the environmental determinant of sex correlate with some differential effect on male/female fitness, and b) that males (females) develop under conditions relatively beneficial to themselves.

In some cases the observations appear to be consistent with the model. In *Bonellia*, for example, one patch type consists of open substrate, and the other consists of adult females. A larva developing as male in open substrate would have negligable fitness (unless, perhaps, it was not dwarf); a larva developing as female inside of another female would also have low fitness (although a larva destined to be female might be able to avoid settling in another female). Thus, the patch types have differential benefits to males and females, and sex is determined accordingly. In mermithids, the observations are again potentially consistent with the model. Females develop under conditions of good nutrition. The model is consistent only if fitness as female is enhanced more by large size than is fitness as a male. These conditions seem to be met, although the evidence is circumstantial. (Despite the inadequacy of the data at present, it would be difficult to argue that males benefitted *more* from large size than females[13].)

Conover[24] specifically investigated silverside ESD in the context of the Charnov-Bull model; the model was tentatively supported. Conover observed that the waters inhabited by silversides were cooler early in spring than late in spring, and fish born early grew to a larger size before winter. Thus water temperature during development correlated with adult size. Conover further showed that fitness as a female was enhanced by large size; various components of male fitness did not seem to be so enhanced by large size, although male fitness could not be estimated as directly as female fitness could. At the level of these observations, the model is supported, because females are overproduced at cool temperatures – the temperatures that enable them to reach the largest sizes as adults.

Reptiles offer the greatest enigma from this perspective. There is no a priori reason to suggest that incubation temperature has any effect that would persist into adult life, much less one that differentially affected fitness as male versus female. (Temperature affects embryo survival, but the effect probably does not differ between males and females.) Some recent work has shown that various environmental conditions during incubation affect the size and yolk reserves of surviving hatchlings[57], but again it remains to be demonstrated whether these effects translate into ultimate fitness differences and whether they have different consequences for males and females. At present, the null hypothesis that the Charnov-Bull model does not apply to reptiles stands unchallenged.

5) Haplodiploidy

An interesting and common genetic system in arthropods is haplodiploidy: males develop from unfertilized eggs, females from fertilized eggs (also known as arrhenotoky). Haplodiploidy is known in all sexual Hymenoptera (ants, bees, wasps), in mites and ticks, whiteflies, thrips, scales, and some beetles[7, 13, 36, 54, 55, 69, 77, 78]. This section briefly outlines some major points about the possible evolution of haplodiploidy.

Consider heterogametic sex determination as a possible immediate ancestor of haplodiploidy. The origin of haplodiploidy requires that unfertilized eggs develop as male, and the heterogametic mechanisms in which all unfertilized eggs develop as male are as follows[13]:

5.1		5.2		5.3	
♀	♂	♀	♂	♀	♂
XX	XY	ZW	ZZ	ZW	ZZ
	X		Z		Z
			W	WW	W
			WW		

Here, uniparental offspring are represented as haploid; XX/XY represents an ancestral mechanism of male heterogamety, ZZ/ZW female heterogamety.

All three systems violate any notion of recessive-X and dominant-Y principles, and they have the property that the sex factors causing males come exclusively from females of the previous generation. From studies on the sexes of YY individuals and of XO and XXY aneuploids, as described in section 3, these three systems, 5.1, 5.2 and 5.3, are presumably rare among heterogametic species. Thus the very origin of haplodiploidy may be precluded from a lack of appropriate sex determination. (Several other requirements – viability and fertility of the haploid – may also prevent evolution of haplodiploidy[36, 78]).

Assume that a species has one of the three systems conducive to haplodiploidy, 5.1, 5.2, or 5.3, and consider the transition from heterogamety to haplodiploidy. If some eggs fail to be fertilized, the population contains haploid and diploid males (referred to as uniparental and biparental males, respectively). Hartl and Brown[36] showed for system 5.1 that the equilibrium frequency of these two types of males is governed by two parameters, the fitness of uniparental males relative to biparental males, and the fraction of eggs developing without fertilization. In system 5.1, XY males are lost if enough of the eggs go unfertilized and if uniparental sons are sufficiently fit. With the loss of Y, all males are X (all of them then arise from unfertilized eggs), and haplodiploidy is established[36]. An equivalent scenario applies to system 5.3, in which the loss of Z establishes haplodiploidy. (Obviously, systems 5.2 and 5.3 require that W and WW individuals be viable and fertile.)

Surprisingly, system 5.2 is only slightly different. Since all haploids and diploid homozygotes are male, diploid males cannot be lost (homozygous offspring are unavoidable). However, suppose that additional factors with this type of sex determination can arise, so that this mechanism is more generally

♀	♂
A_iA_j	A_iA_i
	A_jA_j
	A_i
	A_j

for $i \neq j$ $(i,j = 1, \ldots, n)$.

This system is in fact the one known from several hymenopteran species[13,29]. If enough of these factors are present, the evolution of uniparental males obeys the same principles that Hartl and Brown discovered in system 5.1, and haplodiploidy can evolve (homozygous males become vanishingly rare when many factors occur in the population[13].

In conclusion, there are simple transitions from heterogamety to haplodiploidy, just as transitions were found between other sex determining mechanisms. In this case the transition depends on two parameters, the fraction of unfertilized eggs and the relative fitness of uniparental males. As in the model for the change from GSD to ESD, therefore, the evolution of haplodiploidy appears to depend on a parameter that is largely environmental, in this case the fraction of unfertilized eggs. Once again, however, this parameter is subject to evolution. *A female may be selected to avoid fertilizing her eggs to produce uniparental sons.*

This last point may be the most important principle in the evolution of haplodiploidy, given that all the prerequisites have been met (sex determination, viability and fertility of uniparental males)[10]. Consider a mother producing a uniparental son versus a biparental son. When the uniparental son produces gametes, every gene locus in all of his gametes will carry her alleles. However, when the biparental son produces gametes, only half of the loci in his gametes will, on average carry her alleles; the other half will carry her mate's alleles.

Will selection therefore favor mothers who produce uniparental sons? The answer depends on the fitness of uniparental versus biparental sons. Denote w as the fitness of a uniparental son relative to that of a biparental son. The uniparental son transmits $2w$ maternal alleles for every one transmitted by a biparental son. Thus *if* $2w > 1$, *or* $w > \frac{1}{2}$, selection favors mothers who avoid fertilizing eggs and produce uniparental sons. Given this condition, the evolution of haplodiploidy from diploidy automatically follows in all the above systems 5.1, 5.2, and 5.3[13].

Hermaphroditism

The last case to be discussed here concerns hermaphroditism. The evolution of hermaphroditism from dioecy, or the reverse, has been addressed mostly from the perspective of *why* one system is favored over the other[20]. Briefly, the theory of sex allocation predicts that pure hermaphroditism is favored over dioecy when two conditions are met: a) the reproductive success realized by the hermaphrodite through male function is more than half that of a pure male, *and* b) the reproductive success realized by the hermaphrodite through female function is more than half that of a pure female. For example, if different resources are used to make male gametes than to make female gametes, a hermaphrodite may be able to make almost as many male gametes as a pure male and almost as many female gametes as a pure female; hermaphroditism would be favored in this case if reproductive success was limited chiefly by the number of gametes produced. Further details of these arguments are to be found in Charnov[20].

The question of how (and why) the transition occurs between hermaphroditism and dioecy has been addressed in one elegant case that warrants description here. The transition from hermaphroditism to dioecy has occurred in several flowering plants, and the genetic basis has been worked out in some cases[18,44,76]. According to Charlesworth and Charlesworth[18], the most plausible selective force for the transition is inbreeding depression: the hermaphrodite may not be able to avoid selfing, and the selfed offspring may have depressed fitness from the high incidence of homozygosity for otherwise rare, deleterious genes (inbreeding depression). If the rate of selfing is high enough and the inbreeding depression severe enough, a pure-female mutant can invade – a plant that suppressed pollen production. As this female-type increases, the conditions relax for invasion of a pure male (a plant suppressing ovule production). However, to avoid generating plants that suppress both pollen and ovule production (which would be sterile), both factors must sort in opposition; heterogamety results. Charlesworth and Charlesworth's model further explained the preponderance of male heterogamety over female heterogamety in these plants.

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* As used in this paper, a sex determining mechanism is simply a description of the inherited and environmental effects on whether an individual develops as a functional male and/or a functional female.

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Quantitative genetic models of sexual selection

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Summary. Quantitative genetic models of sexual selection have disproven some of the central tenets of both the handicap mechanism and the 'sexy son' hypothesis. These results suggest that the 'good genes' approach to sexual selection may generally lead to erroneous results.

Runaway sexual selection seems possible under a wide variety of circumstances. Quantitative genetic models have revealed runaway processes for sexually selected attributes expressed in both sexes and for attributes of parental care. Furthermore, the runaway could occur simultaneously in a series of populations that straddle an environmental gradient. While the models support the feasibility of runaway processes, empirical studies are needed to evaluate whether runaways actually happen. Estimates of critical genetic parameters are particularly needed, as well as measures of natural and sexual selection acting on the same population.

The models also show that sexual selection has tremendous potential to produce population differentiation, particularly in epigamic traits. Differentiation is promoted by indeterminancy of evolutionary outcome, transient differences among populations during the final slow approach to equilibrium, sampling drift among equilibrium populations, and the tendency of sexual selection to amplify geographic variation arising from spatial differences in natural selection.

Recent work with two- and three-locus models of sexual selection has produced results that parallel the results of the polygenic models[36–38, 58]. Thus the feature of indeterminate equilibria (outcome dependent on initial conditions) is common to both types of model.

Key words. Sexual selection; quantitative genetics; handicap principal; sexy son; runaway models; 'good genes'.

1. Introduction

The aim of this paper is to survey recent quantitative genetic models for evolution by sexual selection. These models treat the evolution of continuously distributed traits such as tail length in the males of a lek-breeding bird and female mating preference based on such an attribute. The quantitative genetic models have some advantages over two- and three-locus models of sexual selection, recently discussed by O'Donald[54], Kirkpatrick[36] and Seger[58]. Most traits of interest to students of sexual selection are continuously distributed with polygenic inheritance rather than composed of two or three discrete classes with single factor inheritance. Secondly, the parameters of inheritance and selection used in the quantitative genetic models can be measured in natural populations, whereas the crucial gene frequencies and selection coefficients in the oligolocus models are virtually always inaccessible to measurement. The polygenic models also permit sustained evolution, but the oligogenic models may quickly reach limits imposed by gene fixation, which may obscure some evolutionary phenomena. In defense of the two- and three-locus models, we may note that their logic is sometimes found to be more tractable. In any case, the principal conclusions are the same using both types of models.

A review of the quantitative genetic models is timely for two reasons. Models of this type have recently proliferated and it is easy to become overwhelmed by new results. Secondly, the model family is now large enough to provide some generalizations about equilibria and stability conditions that instruct our intuition about evolutionary phenomena in general, as well as our understanding of sexual selection. For both these reasons I will focus on the common denominators in the models and on a comparative analysis of their conclusions.

Several terms are used here in a formal sense that sometimes differs from popular usage. *Sexual selection* refers to selection acting via effects on the mating success of surviving males (or females) whereas *natural selection* refers to effects on other components of fitness (e.g. viability). This usage is common to all the theoretical models discussed here. The use of mutually exclusive terms rankles some, despite the fact that Darwin[14, 15] used 'natural' and 'sexual selection' in just this way[3, 28, 65]. Some prefer to see sexual selection as a subset of natural selection. The important point is that viability, or other forms of, selection may act in a different direction than sexual selection,