SEX DIFFERENCES IN RATES OF RECOMBINATION AND SEXUAL SELECTION*

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There is a very widespread sex difference in the underlying genetic system which has gone nearly completely unnoticed in our recent efforts to understand sexual selection and the evolution of sex (but see Bell, 1982, and Bull, 1983). That sex difference is a difference in the rates at which genes in the two sexes recombine during meiosis in the production of the two gametes. Although it has long been appreciated that the sex chromosomes themselves are associated with differences in recombination (e.g., XX chromosomes recombine freely while XY or XO do so little or not at all), much more striking is the widespread occurrence of a sex difference in rates of *crossing over* between paired autosomes. In almost every species studied, the sexes differ in rates of crossing over, and data from numerous species form a complex and interesting pattern which invites explanation. Even if the reader sees little merit in the theory I advance to explain these facts, I hope at least to show the potential value in the genetical facts reviewed here for our understanding of sexual selection and sex.

The facts concerning sex differences in recombination, to my mind, are most easily accommodated by imagining that sexual selection often acts adaptively from the female's standpoint; that is, it often causes superior genes to be passed in the male sex. This, in turn, selects for tighter linkage, in order to preserve the more highly favored combinations of genes being revealed by sexual selection. Twenty years ago the assumption that in species lacking male parental investment processes of sexual selection would raise to breeding status in males

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superior genes from a female's standpoint (as measured by an increase in the survival and fecundity of her resulting offspring) would have been controversial. Indeed, the common bias in this century has been the assumption that sexual selection usually acts maladaptively from the female's standpoint. That is, it gives her genes from the noninvesting males worse than those she would get from pairing at random with another female (were that possible). This assumption has been based on a keen appreciation of the pervasiveness and strength of male–male competition, especially aggressive, which, if unguided by female choice, would easily seem to generate extremes in male morphology and behavior useful only in a male–male competitive context, such that the other genes thereby elevated might actually be worse for a female's *daughters* than the genes of an average male.

In addition, female choice was given a male bias. When imagined to operate at all, female choice was usually assumed to favor traits in males which were useful predictors of their *sons'* future success. To put this in the vernacular: either females choose a "bully" as a mate in order to (produce) sons good at excluding other males, or they prefer a "pretty boy," a male preferred by many other females, in order to (produce) sons attractive to the daughters of these females. By contrast, a renewed appreciation of the importance of female choice, combined with the demonstration that systems of female choice are expected to evolve with a bias toward the interests of daughters, suggests that sexual selection may often act adaptively (Seger and Trivers, 1986; Trivers, 1985). In any case, our image of the possible adaptive value of sexual selection should affect our expectation regarding rates of recombination. If the logic presented in this chapter is accepted, then the pattern of evidence regarding sex differences in recombination provides additional reason to believe that sexual selection may often act adaptively. This is based on the assumption that a sex difference in recombination measures in part the degree to which, in one generation, selection has acted more adaptively (from the standpoint of resulting progeny) in one sex compared to the other. Put another way, if linkage is tighter in males, we are permitted the assumption that breeding males possess superior genes, on average, compared to breeding females.

Finally, I will emphasize that just as the genetic system may affect the operation of selection, so natural selection molds the genetic system. As Ernst Mayr (1963, 1982) has emphasized, Darwin's theory of evolution was a *two-factor* theory: two independent factors, genetic variation and natural selection, interact to produce evolutionary change over time. Since the rediscovery of Mendel's work, it has been common to emphasize the relevance of genetics for the operation of natural selection. Thus, details of the genetic system such as dominance, linkage, epistasis—and especially, mode of inheritance—may affect the direction and intensity of natural selection. The direction of causality in these arguments can be summarized as follows: genotype → phenotype → selection. That is, details of the genotype have some effect on the phenotype, which in turn affects selection. Or, as in the case of mode of inheritance, genes

affect selection directly by affecting the proportions with which alternate genotypes are inherited (e.g., sex chromosomes vs. autosomes). An example of the former causal chain is: heterogamety → higher mortality → sexual selection. That is, heterogamety (the XY or XO sex) causes higher mortality (due to the breakdown of diploidy and subsequent genetic defenselessness against the appearance of "lethal" recessives) which, in turn, alters the adult sex ratio, thereby affecting the degree of intrasexual competition for members of the opposite sex.

Much less emphasized has been the possibility that natural selection molds the genetic system, either via a phenotypic trait or directly. In the above example, sexual selection → higher mortality → heterogamety. That is, sexual selection causes higher mortality (Trivers, 1972, 1985) and this more severe selection—if oriented in the right direction—favors tighter linkage which, in turn, may predispose that sex to the evolution of heterogamety (Bull, 1983). On the latter connection, Haldane (1922) had argued from the genes outward: heterogamety causes tighter linkage on the autosomes. We argue the other way around: an initial sex difference in recombination across the autosomes predisposes the sex with tighter linkage to the appearance of heterogamety in order to preserve combinations of sex factors or genes having sex limited benefits (Bell, 1982; Charlesworth and Charlesworth, 1978). Increased selection for even tighter linkage on the incipient sex chromosomes induces further genetic isolation and, hence, divergence in self-interest leading to increased conflict between the X and the Y chromosomes and eventual reduction and nullification of the Y (Hamilton, 1967).

SEX DIFFERENCES IN RATES OF RECOMBINATION

Rates of recombination are affected by two variables. On the one hand, nonhomologous pairs of chromosomes assort themselves independently during meiosis, so that increasing chromosome number will, with overall length of the genotype held constant, increase rates of recombination. On the other hand, homologous chromosomes may exchange segments during meiosis so that linked genes (located on the same chromosome) show some frequency of recombination above zero. Typically the sexes do not differ in chromosome number, but in haplodiploid systems males have only half as many chromosomes as a female—a haploid set—and there is no recombination, while females freely recombine. In all haplodiploid species (and parahaplodiploid species—see Bull, 1983) the male is the haploid sex, or reproduces as if he were (parahaplodiploidy = paternal genome loss). Systems having related effects are multiple sex chromosomes and reciprocal translocation heterozygotes (see below). Likewise, in the XO/XX means of sex determination (which is common in many insect groups) the heterogametic sex has one less chromosome (an X) than the homogametic sex. This is usually the male.

In addition to the above factors, whose effects on total recombination are typically small (e.g., XX vs. XY), the sexes also usually differ in the degree to

which linked genes located on homologous pairs of autosomes recombine. As is now well known, this kind of recombination results from a process termed crossing over. Crossing over occurs in mid-prophase of meiosis, when homologous chromosomes form intimate connections seen cytologically as "chiasmata," after which the homologous chromosomes typically switch chromosegments located "below" the connecting point. More than one chiasma may form between two homologous chromosomes, but smaller chromosomes typically show a single chiasma. Were this true for all the chromosome sets in an individual, and were the chiasmata always located in the exact midpoint of each chromosome, then this degree of crossing over would be exactly equivalent to cutting all the chromosomes in half at the midpoint and thereby doubling chromosome number.

Sex differences in rates of crossing over can be detected in two ways. They may be seen directly by counting chiasmata in the two kinds of sex cells photographed at parallel stages in meiosis, or they may be detected by counting the frequencies of various combinations of linked markers among progeny depending on the sex bearing the markers (for both methods applied to a marsupial, see Bennett et al., 1986). Each method has shown that there may be striking heterogeneity in sex differences in recombination depending on the section of the genotype being compared. For example, in the mealworm *Tribolium castaneum* studies of genetic markers reveal three linkage groups, in which adjacent sections show a reversal in the sex with tighter linkage (Dawson and Berends 1985). Likewise, cytological studies of chromosomes forming chiasmata show that these are sometimes distributed in a complementary fashion between the two sexes. For example, in the grasshopper *Stethophyma grossum*, chiasmata in males are procentrically located (that is, close to the centromeres), while in females only 6 percent of chiasmata are procentrically located, the remainder being interstitial or distal (White, 1973; Perry and Jones, 1974).

A remarkable feature of complementary sex differences in recombination is that the complementarity may differ markedly in closely related species. For example, in newts (*Triturus helveticus*), males show proterminal chiasma localization: almost all chiasmata are found at the ends of chromosomes (Watson and Callan, 1963). Although chiasmata frequency is similar in females, these are not localized. A partial reversal of this pattern is found in *Triturus cristatus*, in which chiasmata in male meioses are spread along the length of the chromosomes, while chiasmata in females are located near the centromeres.

In spite of this heterogeneity, in many species there are clear *average* differences between the sexes in rate of recombination across the autosomes. For example, across a wide variety of taxa, crossing over may be regular in one sex but completely absent in the other (meiosis achiasmatic: e.g., male *Drosophila*, female butterflies and moths). In species with crossing over in both sexes, chiasmata *number* may differ consistently between the sexes or the majority of linkage groups may show similar sex differences in linkage rates. For example, in the house mouse, 23 intervals measured across 10 linkage groups showed

significant sex differences in recombination. These were distributed within linkage groups such that nine groups always showed tighter linkage in males, while one group always showed tighter linkage in females (Dunn and Bennett, 1964). Even when the distribution of chiasmata within one sex differs from the other, average differences in chiasmata number often vary in the same direction. For example, in *Triturus*, chiasmata number are always slightly higher in the sex showing nonlocalized chiasmata, so that on both counts this sex shows higher rates of recombination.

The facts concerning average sex differences in autosomal recombination form an interesting pattern, as revealed by the valuable reviews of Dunn and Bennett (1964), Perry and Callan (1977), and Bell (1982). The most striking generalizations are these: linkage is typically tighter in males or in male tissue (a rule to which there are numerous exceptions); there is a strong—but not universal—tendency for the heterogametic sex to show tighter linkage; and only a few species are known to show no sex difference at all.

1. *Recombination is typically greater in females than in males.* This is true of most mammals, such as humans, laboratory mice, and horses, all of which typically show about 30 percent higher recombination across linkage groups in females than in males (Dunn and Bennett, 1964; Andersson and Sandberg, 1984). Achiasmatic meiosis is in the majority of insect orders confined to the male sex (Bell, 1982). Besides the well known case of *Drosophila* and some related Diptera, achiasmatic male meiosis is also found in various grasshoppers (White, 1965), in some bugs (Nokkala and Nokkala, 1983, 1984), in a beetle (Serrano, 1981) and in a mantid (White, 1938).
2. *This rule extends to male gonadal tissue compared to female in hermaphrodites.* This is true of the majority of plants that have been tested, e.g., *Lilium* spp. and species of *Allium* (Ved Brat, 1966). It is also true of a turbellarian worm (Pastor and Callan, 1952). The importance of this finding is that it suggests that the *phenotype* of the individual may control rates of recombination more than the genotype of the cells undergoing meiosis (since genotype in the two kinds of cells is identical). This supposition is strengthened by the remarkable finding of Yamamoto (1961): by hormone treatment he turned XY male fish (*Oryzias latipes*) into phenotypic females. When such a male reproduced, he showed five times as much crossing over between his X and Y chromosomes as did phenotypic males. For our purposes, it would have been more striking if Yamamoto had shown an effect on recombination across the autosomes, since it can be argued that an X in a female normally undergoes strong recombination with another X and is "fooled" in the XY female into thinking she is recombining with another X chromosome. In either case, however, a controlling role of the phenotype is established.
3. *There are numerous exceptions.* For example, unlike the placental mammals mentioned above, the marsupial fat-tailed insectivore *Sminthopsis crassicaudata* shows tighter linkage in females (Bennett et al., 1986). The salamander

Triturus cristatus shows tighter linkage in females. Although six orders of insects show achiasmatic male meiosis in at least some species, the Lepidoptera and Trichoptera (themselves closely related) invariably show achiasmatic meiosis limited to females (e.g., Turner and Sheppard, 1975; Suomalainen et al., 1973; Suomalainen, 1966). Among other invertebrates a few scorpionids and acarines are known to have achiasmatic meiosis limited to males, but several copepods show achiasmatic meiosis limited to the female. In hermaphroditic plants, *Pinus radiata* shows 43% higher recombination across one linkage group in male tissue compared to female and a similar (but not significant) trend in a second linkage group (Moran et al., 1983). These exceptions are critical to a test of any theory. I will argue that the exceptions will be associated with (1) substantial male parental investment (Lepidoptera/Trichoptera); (2) substantial male mating cost (copepods, *Triturus*); or (3) a sharp reduction in the efficacy of sexual selection (*Pinus*).

4. *There is a strong—but not universal—association between heterogamety and tighter linkage.* For example, copepods and Lepidoptera/Trichoptera are female heterogametic and achiasmatic in female meiosis. Indeed, there is a one-to-one correspondence between the sex showing achiasmatic meiosis (when only one sex does; see below) and the sex which is heterogametic (Bell, 1982). When both sexes show crossing over, however, exceptions begin to appear. Male marsupials are XY, yet *Sminthopsis* females show tighter linkage. Chickens are female heterogametic, yet show tighter linkage in males (Fisher and Laundauer, 1953; Warren and Hutt, 1936; Warren, 1940). *Triturus helveticus* is female heterogametic, yet shows tighter linkage in males. Nevertheless, in species in which chiasmata occur in both sexes, the general pattern is for heterogamety to be associated with tighter linkage.
5. *Cases in which no sex difference in recombination exists are relatively rare.* Although seven genera of oligochaete worms are known to have achiasmatic meiosis in both sexes (thus, no sex difference in recombination: Christiansen, 1961), all other cases of achiasmatic meiosis are restricted to one sex (Bell, 1982). Likewise, although scattered species, both hermaphroditic and dioecious, have failed to show a sex difference in recombination (Callan and Perry, 1977; Bell, 1982), the great majority of species investigated show consistent, significant differences.

SEX DIFFERENCES IN RECOMBINATION ACROSS THE SEX CHROMOSOMES

The sex difference in recombination across the autosomes is supplemented by a sex difference in rates of recombination across the sex chromosomes. The X and the Y chromosomes typically show low rates of recombination. Although especially true for X's and Y's that differ markedly in size and structure, a sharp reduction in recombination sometimes precedes structural divergence of the

incipient X and Y (Schmidt et al., 1979). When one sex is XO, there is no recombination across the sex chromosomes, while the XX may show normal recombination. Haplodiploid and parahaplodiploid species (Bull, 1983) are extreme examples of this. Intermediate examples are systems of multiple sex chromosomes, either multiple X's or multiple Y's; the former tend to increase the proportion of the genotype inheriting in a haplodiploid fashion (see White, 1973). Another intermediate case is sex-linked translocation heterozygotes limited to the male sex, e.g., many species of termites (Luykx and Syren, 1979) and possibly all of the monotremes (Murtagh and Sharman, 1977). Similar translocation heterozygotes have been described for staminate mistletoes (*Viscum*: Wiens and Barlow, 1979). These facts can be summarized by saying that the mode of inheritance typically causes at least a small sex difference in recombination (measured across the entire genotype) according to the rule that the heterogametic sex shows little or no crossing over on the sex chromosomes (entire genotype for haplodiploid species).

Haldane (1922) was the first to produce a logic linking sex differences in recombination on the sex chromosomes to sex differences in recombination on the autosomes. He argued that the sex chromosomes were primary; that selection had favored a sharp reduction in crossing over (for example, to bind two independently segregating sex factors to each other); and that this had had a spill-over effect onto the autosomes, either because an absence of recombination on the sex chromosomes mechanically interfered with smooth recombination across the autosomes, or because selection to reduce recombination on the sex chromosomes had often acted on the genome at large, thereby increasing linkage on the autosomes. In hindsight, this argument has the form of the evolutionary tail wagging the evolutionary dog. There is no evidence for a mechanical interference, and there is no a priori reason for supposing that natural selection could not separate the two variables as, indeed, in exceptional cases we know it has.

The reverse logic seems more appealing (Bull, 1983): that an initial sex difference in recombination across *all* chromosomes predisposed the sex with tighter linkage to evolve heterogamety. This would occur if sex were determined by two or more independent loci located along the incipient sex chromosomes (as in plants: Charlesworth and Charlesworth, 1978), or if there existed genes with strong sex-specific effects on reproductive success along the incipient sex chromosomes which would benefit from tight linkage to the appropriate sex factor. At the same time, the frequent evolution of haplodiploidy, parahaplodiploid systems, and intermediate systems involving multiple sex chromosomes or translocation heterozygotes suggests that there has been selection to increase the proportion of the genotype undergoing little or no recombination in the heterogametic sex.

At this point it is worth emphasizing that this haplodiploid portion of the genotype, in addition to restricting recombination in one sex, also generates a novel pattern of degrees of relatedness among offspring and, perhaps more

relevant to this discussion, increases the proportion of genes in the heterogametic sex which are inherited in the first generation only by the homogametic sex. In the typical case, that is, *it increases the proportion of genes in males which are inherited only by daughters*. This, as we shall see, may become important under certain kinds of female choice and sexual selection.

SEXUAL SELECTION AND SEX DIFFERENCES IN AUTOSOMAL LINKAGE

Two facts are worth noting at the outset. First, the autosomes are equally inherited by sons and daughters and are, except where linked to the sex chromosomes, distributed at random to the two sexes. This means that being male or female predicts nothing about the sex in which one's autosomes will reside in the future. Second, an individual's autosomes are equally likely to have been inherited from either parent. Thus being a male or female implies nothing about one's past life as either. Hence, if natural selection has molded different optimal rates of recombination in the two sexes, this can only reflect the intensity and direction of selection that has occurred in one generation: from the moment of conception to the moment of breeding. The chief variable affecting this will be sexual selection. Conceptually, it is valuable to distinguish three components of sexual selection: differential mortality by sex, intrasexual competition, and intersexual choice. For the great majority of species—that is, those lacking male parental investment—these typically translate into differential male mortality, male–male competition, and female choice (for a recent review see Trivers, 1985).

In species lacking male parental investment we expect to find greater variation in male reproductive success compared to that of the female (Bateman, 1948; Trivers, 1972). This means that the autosomal genes enjoying reproductive success on the male side are a more restricted sample of the original set of genes with which the generation began than are the genes in breeding females. If this additional restriction in males is in the right direction from the standpoint of resulting offspring—that is, if it is in the same general direction as the original restriction—then the genes and combinations of genes being passed in males will be superior on average, compared to genes passed in females. (Superior, that is, as measured by their effects on survival and reproductive success of the resulting offspring.) Insofar as the actual *combinations* in which a male's genes appear are important to their success, then he will be selected to reduce rates of recombination (compared to females) in order to preserve these beneficial combinations. This explains the primary rule in nature regarding rates of recombination: in dioecious species with no male parental investment, these are commonly lower in males. Note that the argument depends crucially on the assumption that sexual selection improves the quality of genes being passed in males, an assumption that is consistent with a growing body of evidence from

both plants and animals (see below) but is by no means a necessary feature of nature.

At first glance, the facts concerning sex differences in recombination in hermaphroditic individuals would appear to contradict the theory being proposed for dioecious species. A hermaphroditic plant passes the same genes through pollen as through ovules; why not link them equally tightly within individuals? If sexual selection is operative, we might expect a hermaphrodite who was of high female quality to be even more favored as a male. The plant should show equally tight linkage, however, in its two kinds of gonadal tissue because they are genetically identical. We might expect variation in quality of hermaphrodite to be matched by variation in frequency of recombination, but within each individual we would expect both kinds of sex cells to receive the same amount of recombination. But in fact, recent evidence from plants strongly suggests that sexual selection acts in hermaphroditic species, and that an individual's success as a male and as a female may not be tightly coupled (Stephenson and Bertin, 1983; Bertin, 1982).

If the organism is to some degree uncertain about the quality of its genes, it could reason as follows: "If I have high success as a male, I should assume that the quality of my genes is good and, since I'm unable to predict this in advance, I will link my genes more tightly in pollen production than in ovule production. If I have low quality genes, my pollen will not be used much in any case, so that the inappropriately tight linkage will not be an important factor overall." Evidence regarding this interpretation of plants will be reviewed below; the comparable situation in hermaphroditic animals is much less clear, though cases of reciprocal egg trading such as has been discovered in sea bass in which an individual's success as male and female are presumably tightly linked are probably the exception and not the rule.

Cases of tighter linkage in females appear to be associated with male parental investment (e.g., birds, Lepidoptera/Trichoptera) or with male mating effort (salamanders, copepods). The argument for these correlations runs as follows: in species with high male parental investment, one might initially expect rates of recombination to be similar in the two sexes. But the situation in birds suggests that when male parental investment is substantial (e.g., more than one-half female parental investment) but is less than female parental investment, there may be a sharp reduction in sexual selection on males while the higher female parental investment induces differential *female* mortality. Thus, we expect breeding females to be the more restricted sample and, providing this restriction is in the right direction (improves the genetic quality of offspring), they will wish to recombine their genes less. Although the evidence is less clear from butterflies and moths (see below), differential *female* mortality is at least more common in this group than other groups of animals (with the exception of birds), so that a similar explanation may apply.

Variation in male reproductive success may also be reduced when copulations are expensive for males, even though males invest nothing in offspring.

For example, in salamanders costly spermatophores are produced which are, however, low in proteins and lipids and are not digested by females. Likewise, in copepods and some other marine invertebrates, expensive spermatophores are produced which appear to function as a mechanism of sperm transport in an aqueous medium (Mann, 1984). These are not digested by the female, but are still expensive for the male to produce. In any case, as noted above, there seems to be a crude correlation between the production of such expensive but nondigested spermatophores and female heterogamety.

EVIDENCE THAT SEXUAL SELECTION MAY OFTEN ACT ADAPTIVELY FROM THE FEMALE'S STANDPOINT

The only direct experimental evidence regarding the adaptive value of sexual selection in animals comes from the work of Partridge (1980) on *Drosophila melanogaster*. When females were assigned mates at random, their larvae suffered 2–4% greater mortality in competitive circumstances in the lab, compared to the larvae of females housed in large enclosures with many adult males. Direct evidence from the wild comes from a study by Clutton-Brock (1983) on red deer. He showed that those males who defended larger harems during their years of rutting survived longer than those males that defended smaller harems, suggesting that sexual selection and selection for survival were aligned in the same direction.

More convincing experimental evidence comes from the study of plants (see especially Stephenson and Bertin, 1983; Willson and Burley, 1983). In angiosperms, at least, pollen competition acts in an adaptive fashion, selecting for genes which result in plants that survive and grow better (Mulcahy and Mulcahy, 1987; Mulcahy, 1983). Selective fruit abortion is often aligned in such a way as to compound the positive effects of pollen competition: greater pollen competition results in more seeds set per fruit and fruits with relatively few seeds are preferentially aborted (e.g., Winsor et al., in press; Stephenson and Winsor, 1986; Bookman, 1984; see also Marshall and Ellstrand, 1986). There is growing evidence that flower coloration and size in hermaphroditic plants works primarily to favor pollen dispersal (Bell, 1985; Schoen and Clegg, 1985). Unlike sperm which express only paternal gene effects, more than 50% of the haploid genotype is expressed during pollen tube competition (e.g., Tanksley et al., 1981) so that there is ample opportunity for much of the genotype to be tested during pollen competition.

On the theoretical side, two advances are notable. Seger and Trivers (1986) showed that there is an intrinsic defense to runaway selection favoring traits beneficial to males but disadvantageous to females, in systems guided by female choice: in such systems males will more easily evolve structures and behavior that reveal the quality of their genes for *daughters* than their sons, because female choice genes benefitting daughters spread more quickly than similar genes benefitting sons. The reason for this is that female choice genes are

expressed only in females and when they benefit daughters they increase in frequency in the choosing sex (females) within one generation. This increase, in turn, increases the reproductive success of males revealing high quality genes for daughters, and such males have a disproportionate number of the female choice gene for these traits. In short, a female choice gene benefitting daughters more quickly reinforces its own spread. By contrast, a similar female choice gene benefitting sons will increase in frequency among males in the first generation only, where it will fail to be expressed. Only in the following generation will we see an increase in the number of females choosing in this fashion. Thus, such genes lag in their effects a full generation behind those choice genes benefitting daughters. Some evidence in support of these assertions is reviewed in Trivers (1985).

A second advance was the appreciation that parasites may have played an important role in selecting for sexual reproduction (Hamilton et al., 1981) and molding mate choice (Hamilton and Zuk, 1982). Specifically, it was shown that species of birds which suffer higher loads of brood parasites tend to be more brightly colored in both sexes (especially in males) and to show more complex male song. Since bright coloration and complexity of song were assumed to reveal absence of parasites, and since mate choice in most birds is expressed by both sexes, the positive correlations were taken to support the assumption that species more heavily parasitized emphasized bright coloration and complexity of song more strongly in choice of mates. As noted by Hamilton and Zuk (1982) parasite selection, insofar as it conforms to certain cyclical features, will tend to maintain considerable genetic variability even while showing strong selection on the variants.

OTHER CORRELATES OF CROSSING OVER

If tighter linkage in males is really explained by sexual selection, then it follows that *within* a sex individuals may be able to rank themselves according to relative expected reproductive success and adjust their level of recombination accordingly. Remarkable evidence consistent with this expectation has been presented by Tucic et al. They showed that female fecundity in *Drosophila melanogaster* was inversely correlated with recombination rates: those females laying 40 or more eggs during a 48-hour period reduced by about 30% their rates of recombination in producing these offspring, compared to females who produced between 0 and 10 eggs during the same period of time. Fertility estimates in males were also inversely associated (with recombination rates in females of similar genotype) but the trend was not significant, probably because in this experimental design, male–male competition and female choice was sharply reduced (male fertility being estimated as the number of females out of ten showing sperm after enclosure with a single male for 48 hours). So far as I know, these are the only data available from any organism linking recombination rates with variation in reproductive success. Unfortunately, these results have

not yet been repeated and are subject to the criticism that they may be a secondary consequence of variation in degree of hybrid dysgenesis, a condition affecting some laboratory strains when exposed to matings with wild-caught individuals in which, among other traits, low fertility co-occurs with high recombination rates. (For a recent paper on hybrid dysgenesis see Simmons, 1986.)

Other known correlates of recombination show confusing patterns which, at this stage in our knowledge, defy explanation. For example, in the tulip, frequency of chiasmata vary in pollen mother cells according to position on the anther, those developing first, at the base of the anther, showing fewer chaismata per cell (Couzin and Fox, 1974). Since there is only a trivial age difference in the plant at which different sections of the anther mature, an adaptive explanation for the facts would have to consider the form of sexual selection operating at different points along the anther. Is it possible, for example, that different intensities of sexual selection are associated with different sections on the anther, so that a cell could logically adjust its chiasma frequency to position on the anther based upon assumptions about its genetic quality if it is successful at that position? A large literature on chiasma frequency in relationship to temperature shows many of the conceivable patterns without any clear logic for the variation (Yanney and Wilson, 1959). Likewise, correlations with age fail to show a consistent pattern within species (Valentin, 1972) or across species (Mayo, 1974). For additional correlates, see Bell (1982).

THE EVOLUTION OF HETEROGAMETY

Let us begin by reviewing the facts that need to be explained.

1. *The distribution of heterogamety by sex.* It is a striking fact of biology that sex at the chromosomal level is oppositely determined in a variety of organisms. In general, species are male heterogametic (XY or XO). In dioecious plants most species are male heterogametic, but some are female heterogametic (Bull, 1983). Mammals and most fish and frogs are male heterogametic, but birds, some fish, and all snakes are female heterogametic. Most insects are male heterogametic, but the Lepidoptera and Trichoptera are female heterogametic. And so on. We must first explain the distribution of heterogamety by sex. It would also be nice if we could explain why vertebrates are usually XY, while insects are usually XO.
2. *Degeneration of the* Y. Relative to the X chromosome and to the autosomes, and whether the female or the male is heterogametic, the Y appears often to have degenerated: it is often sharply reduced in size, in gene content, and has been invaded by repeat elements (Bull, 1983). Why has this occurred?
3. *Conservation of the* X. In contrast to the Y, the X chromosome is conserved in both size and genetic content (Bull, 1983; Ohno, 1967). The X often seems to be slightly larger than the average autosome (in mammals it is 5%

of the total genome, in birds 10%). Why has the X done relatively well compared to the Y, and even compared to the autosomes—prospering, so to speak?

4. *Haldane's rule.* In the same paper in which he first drew attention to the association between tighter linkage on the autosomes and heterogamety, Haldane (1922) enunciated a famous rule: when in a distant cross within a species or a cross between closely related species one sex is absent, rare, or sterile, this is the heterogametic sex. Why should this be true?
5. *X–Y heteromorphism and rates of morphological evolution.* In snakes there appears to be an association between rates of morphological evolution (and hence taxonomic status: "primitive" or "advanced") and degree of dissimilarity between the X and the Y (Jones and Singh, 1985). The Boidae are least advanced taxonomically and have morphologically indistinguishable X's and Y's (females heterogametic). The Elapidae and Viparidae, by contrast, are highly advanced and show a strongly degenerate Y. The Colubridae are morphologically intermediate and are also intermediate in X–Y differentiation: the X and the Y differ often in a single pericentric inversion, so that they are similar in length but differ in the position of the centromere. There is weaker evidence of a similar trend in birds: the "primitive" ratites have morphologically similar X's and Y's while more advanced birds show a degenerate Y (female heterogametic; see, for example, de Boer, 1980). Why should this be true?
6. *Modes of achieving dosage compensation.* Since the homogametic sex will have two X's and the heterogametic sex only one, some adjustment is necessary so that development will proceed smoothly in both even though one sex has two doses of each gene on an X while the other has only one. In placental mammals, this is achieved by the random inactivation of one X cell in each of the somatic cells of the female. This, in itself, is a surprising fact, because it means that female development is, in effect, being adjusted to male development; given the primacy of females in evolution, one would have expected the reverse. In *Drosophila* the more logical adjustment appears to take place, single X's producing as much gene product as two X's do, yet in birds (female heterogametic) there appears to be no dosage compensation, males producing roughly twice the gene products of their X's as females do. Even among mammals there is unexplained variation: in marsupials, females inactivate one X in their somatic tissue but, unlike placental mammals, this is always the *paternal* X (Cooper et al., 1977).
7. *Kinds of genes located on the sex chromosomes.* There is evidence that genes involved in spermatogenesis are disproportionately located on the Y and *also* the X (e.g., Livak, 1984; Lifschytz and Lindsley, 1974). Why should this be so?

In attempting to explain the above facts by reference to the notion of conflict within the genotype (as first outlined in this context by Hamilton, 1967), I

emphasize that some facts remain completely unexplained, e.g., the comparative facts concerning dosage compensation and the genetics of sex determination. I believe the next round of progress in understanding sex chromosome evolution will come from understanding these two areas. In the meantime, I suggest that the key variable in explaining the facts outlined above is an initial sex difference in rates of crossing over on *all* chromosomes due to sexual selection. Notice that this sex difference should evolve in an otherwise genetically homogenous world, lacking sex chromosomes but possessing two sexes, whether monoecious or dioecious. Note also that the efficacy of sexual selection (= tendency to benefit offspring) may have improved slowly at first, so that selection for tighter linkage likewise grew slowly. In any case, once tighter linkage appears in one sex, that sex should show a predisposition toward heterogamety. This should occur whenever two or more linked loci determine sex, or whenever genes at one or more sex linked loci show strong sex-specific effects on reproductive success (Charlesworth and Charlesworth, 1978; Bull, 1983). Either of these two causes leads to further selection for tight linkage across the incipient Y and X. This additional selection for linkage effectively isolates the X and Y from each other in subsequent evolution: insofar as allelles are no longer exchanged via recombination, the incipient X and Y diverge in (1) function, (2) mode of inheritance, and (3) shared self-interest.

Regarding function, isolation means that new mutations (including translocations, transposable elements, et cetera) appearing on one sex chromosome do not, within a few generations, automatically appear on the other. With divergence in genetic content comes the breakdown of loci themselves, so that in the heterogametic sex, there is an increasing proportion of loci on the X and Y which are unguarded or hemizygous.

With lack of recombination, all genes on the Y chromosome are passed from father to son only. Thus the Y should become a repository of genes beneficial only to males, such as those involved in sperm function. (This, however, poses a threat to the X—see below—which may generate genes for sperm function of the X.) By contrast, genes on the X chromosome of a male are all passed to daughters only. Thus, the X is a perfect repository for genes benefitting daughters preferentially. Insofar as sexual selection is oriented in this direction, the X may be selected to grow in size. This possibility is important to emphasize, because conflict between X and Y may lead to selection for the diminution in size of each (Y more so than X—see below; Hamilton, 1967), yet comparative evidence suggests that the X chromosome is often average or larger than average in size.

With lack of recombination, there also comes a striking reduction in shared self-interest. With each generation X and Y will view each other as less related. Although inbreeding will increase relatedness between paired autosomes, it leaves r between X and Y at or near zero. With lack of shared self-interest comes increasing conflict over representation among offspring. Genes on a recently separated Y chromosome might reason as follows: "None of us are

found on the X, nor will we be in the future. We best act together to favor our own spread, even if this means the destruction of the X chromosome." Of course, genes on the X in a heterozygote are expected to "reason" in a similar way, but such genes spend two-thirds of their evolutionary life in females (who are XX), while genes on the Y invariably appear in males only. Thus selection for narrowly selfish behavior on the Y should be three times as intense as similar selection on the X (Hamilton, 1967). In addition, recombination between X's in females may occur at normal levels, while the Y shows at best very low levels of recombination.

To state the matter again, in the heterozygous individual genes on either sex chromosome will see little relatedness to the other sex chromosome and virtually no chance of being associated in progeny, so that all genes on a sex chromosome will be selected to act in concert to bring about preferential inheritance of that chromosome (meiotic drive). By contrast, genes on autosomes—while also tempted towards meiotic drive—will be restrained by some past relatedness (which, through variation in degree of inbreeding, may be high or very low) and *by a future chance, through recombination, of being associated together in progeny*. If these two forces are strong enough, we are led to clear expectations regarding the frequency of meiotic drive in nature: most frequent among sex chromosomes (especially Y), less frequent among autosomes, and least so in the sex having highest rates of crossing over. For fruit flies and most mammals this means that we would expect frequency of meiotic drive to decrease as follows: Y > X > autosome in male > autosome in female.

Although cases of meiotic drive are by their nature difficult to detect (requiring the detection of biased frequencies of genes among progeny), many well-studied cases are now known from animals (Crow, 1979). Cases of sex chromosome drive are, as expected, especially common, e.g., in *Aedes aegypti*, *Drosophila pseudoobscura*, *Nasonia vitripennis*, as well as a butterfly and two species of lemmings (see Trivers, 1985 for a review and references). Autosomal drive has been less frequently reported, as expected, but it is much more difficult to detect (see below). Famous cases are segregation distortion on the fourth chromosome of *Drosophila melanogaster* and the + system on the seventeenth chromosome in mice (*Mus musculus* and *M. domesticus*). By contrast, cases of meiotic drive in *female* mammals and *Drosophila* are nonexistent. The only case of meiotic drive in female animals occurs on the X of a butterfly *Aoraea acedon*. Of course, in butterflies females are XY, so this exception is another confirmation of the general pattern. Although exactly as expected, the facts themselves are still very few from animals, and I doubt that the facts concerning meiotic drive in plants can as easily be explained.

Meiotic drive operating on sex chromosomes in the XY individual is special in several ways. Such drive is easier to detect, because the sexes themselves are usually easy to distinguish and strong deviations among progeny (from, for example, an expected value of 1:1) should stand out. By contrast, the *t* haplotype in wild mice, though known to have an antiquity of at least a few million years

and to have undergone a complex evolution, is classically detected in phenotype (by humans) only when paired with a laboratory-maintained dominant marker gene for tail length (short in non-*t* mice, absent when paired with *t* haplotype). Of course, relative ease of detection may also affect the creatures themselves, though such abilities are expected to evolve: the mice themselves detect *t* haplotypes via smell and make very subtle discriminations. Genes for altering the sex ratio will change the relative frequency of two well-developed and co-adapted morphs, the two sexes. Initially counterselection is slight (since effect on the population sex ratio is small). By contrast, the *t* haplotype appears to be otherwise disadvantageous at *all frequencies* and to be maintained only because of a very strong meiotic drive in males (inherited by about 95% of the offspring of a heterozygous male). In this context, it is also worth noting tht the sex ratio may, especially under conditions of sib-mating, be selected to deviate from 1:1, usually towards an excess of females. Under such conditions, genes inducing the appropriate meiotic drive will, initially at least, be favored at both levels of selection. This appears to have been the case in the lemmings and butterfly mentioned above.

A most intriguing correlation—should it hold true—is that between degree of sex chromosome heteromorphism (degeneration of Y) and degree of morphological specialization (taxonomic rank). Cause and effect could go in either direction. One possibility goes as follows: the more sexual selection acts adaptively, the faster should be the rate of adaptive evolution. At the same time, chromosome degeneration will proceed more quickly because selection will be more intense for tight linkage. Against this view is the fact that it appears to be most valid only early in sex chromosome evolution. Put another way, given that a sharp reduction in crossing over occurs early in sex chromosome differentiation, is it really true that subsequent changes reflect differences in overall selection for linkage due to differences in the adaptive orientation of sexual selection?

SUMMARY

In almost all species examined there is a sex difference in rates of recombination across the autosomes. In dioecious species males usually show tighter linkage and in hermaphrodites cells giving rise to sperm (or pollen) typically show in meiosis tighter linkage than cells giving rise to eggs (or ovules). There are numerous exceptions, most notably in Lepidoptera/Trichoptera and in copepods, in all of which meiosis is achiasmatic in females while showing crossing over in males. These species are female heterogametic and, in fact, in species showing achiasmatic meiosis in one sex, this sex is invariably heterogametic. However, examples are known (e.g., *Triturus helveticus*) in which the heterogametic sex shows *more* recombination across its autosomes than the homogametic sex.

These are explained by arguing that sexual selection often acts adaptively from the female's standpoint and, if so, males are selected to link their genes more tightly in order to preserve their more highly selected beneficial combinations. This also applies to hermaphroditic species, as long as sexual selection is operating, for which there is good evidence in plants. Exceptions are explained by (1) substantial male parental investment, (2) the high cost of male copulation, or (3) the relative weakness of sexual selection.