Evolution, 38(4), 1984, pp. 735–742

SEX CHROMOSOMES AND THE EVOLUTION OF SEXUAL DIMORPHISM

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Received September 26, 1983. Revised December 11, 1983

The evolutionary significance of sex chromosomes has generally been associated with their effect on sex determination (see Mittwoch, 1967; Ohno 1967, 1979 for review). Here I will evaluate the idea that sex chromosomes also play an important role in the evolution of sexually dimorphic traits. As a premise I will assume that sexual dimorphism results from natural selection that favors different phenotypic characteristics in the two sexes. For example, in bighorn sheep (*Ovis canadensis*) horn size and shape are sexually dimorphic. The massive recurved horns of males are presumably an adaptive compromise between the need to blunt head-to-head collisions between fighting males and to act as weapons against predators. The smaller dagger-like horns of females are favored in this sex since their sole function is defense against predators (Geist, 1971). Many less obvious characteristics are also likely to differ in their selective value between the sexes. In an extensive review of sexual dimorphism in mammals, Glucksmann (1981) suggested that traits such as growth rate, thermoregulation, metabolic rate, biorhythms, sensory modality, and a wide variety of other traits differ between the sexes in their optimal value. Characteristics that are selectively favored in one sex but selected against in the other will be referred to as "sexually-antagonistic" traits.

Consider the evolution of a sexually dimorphic trait from a monomorphic state. For example, Geist (1971) used paleontological evidence and taxonomy to argue that the sexually dimorphic horns of bighorn sheep evolved from a sexually monomorphic ancestor. Because an exact record of this evolutionary change is unavailable, I will arbitrarily assume for the purpose of illustration that the size of female horns remained unchanged while the size of male horns increased. The evolution of sexual dimorphism in horn size could have proceeded in at least two ways: 1) The increase in frequency of genes that enhanced horn size in males but not in females, and 2) The increase in frequency of genes that enhanced horn size in both sexes followed by the evolution of modifier genes that restricted the expression of increased horn size to males. The first way will be referred to as the "pleiotropy-mechanism." It requires genetic variability that simultaneously produces the sexually-antagonistic trait (increased horn size) and is sex-limited in its expression. The second way will be referred to as the "modifer-mechanism." It requires genetic variability for both the sexually-antagonistic trait (increased horn size) and sex-limited expression of this trait. A similar classification was previously proposed by Turner (1978) for the evolution of sex-limited traits in butterflies.

Most mutations that have been studied carefully in the laboratory have not been found to be completely sex-limited in their expression. Thus it seems reasonable to assume that most of the genetic variability available for the evolution of sexual dimorphism would be initially expressed in both sexes. This assumption may be unreasonable when considering the enhancement of an established sexually dimorphic trait since developmental canalization (Waddington, 1962) may facilitate sex-limited gene expression. However, during the initial evolution of sexual dimorphism from a monomorphic state, a feasible sequence of events would be the "modifier-mechanism" described above.

In the following model I will suggest how the sex chromosomes can facilitate

the initial buildup of sexually-antagonistic genes (i.e., genes producing a sexually-antagonistic phenotype) within the gene pool and initiate the evolution of a sexually dimorphic trait. Consider a large random-mating population with nonoverlapping generations and constant fitness values. Assume that the A-locus codes for a rare sexually-antagonistic allele ($A1$) and an established allele ($A2$) which produces a trait with unit fitness in both sexes. The $A1$ allele increases the fitness (survivorship) of one sex by an increment S when homozygous (or hemizygous in the heterogametic sex) and hS when heterozygous (see, for example, Table 1). The parameter h is a dominance scaler. When $h = 0$ the sexually-antagonistic allele is fully recessive and when $h = 1.0$ the allele is fully dominant. The same allele ($A1$) when expressed in the opposite sex reduces fitness by an increment of T when homozygous (or hemizygous) and hT when heterozygous. The values of S, T, and h are constrained to the interval [0, 1]. The zygotic frequency of $A1$ will be denoted by p and the frequency of $A2$ by q, with $p + q = 1$. The subscripts f and m will be used to refer to the homogametic and the heterogametic sex respectively.

The Autosomal Case

First consider the case in which the A-locus is autosomal (Table 1). The frequency (p) of the allele $A1$ in the gene pool will be the arithmetic mean of p_f and p_m. The change in p per generation is given by,

$$\Delta p = \frac{p_m p_f(1 + S) + \left[\frac{p_m q_f + p_f q_m}{2}\right](1 + hS)}{\bar{W}_m} + \frac{p_m p_f(1 - T) + \left[\frac{p_m q_f + p_f q_m}{2}\right](1 - hT)}{\bar{W}_f}. \quad (1)$$

When p is small, $\bar{W}_m \simeq \bar{W}_f \simeq 1.0$, and Δp is approximated by

$$\Delta p = \frac{S - T}{2}[p_m p_f + (h/2)(p_m q_f + p_f q_m)]. \quad (2)$$

Since Δp is only positive for $S > T$, an autosomal gene that produces a sexually-antagonistic phenotype will only increase in frequency when rare if the advantage to one sex is larger than the disadvantage to the other sex. This result also can be deduced from the more general treatment of sex-specific genes described by Mandel (1971).

The Sex-linked Case

Alternatively, when the sexually-antagonistic gene is located on the X-chromosome (or the Z-chromosome in a WZ system), the conditions for increase when rare are far less stringent. It will be assumed here that the Y-chromosome (or the W-chromosome in a WZ system) is missing all or most of the loci found on the X-chromosome. It also will be assumed that both X-chromosomes are biochemically active in the homogametic sex. This last assumption need not require that both X-chromosomes be active in each cell of the organism, but that both chromosomes contribute to the phenotype of the organism in such a way that dominance between alleles can be expressed at the organismal level.

Advantage to the Heterogametic Sex

Consider a fully recessive X-linked gene ($A1'$) that benefits the heterogametic sex while being disadvantageous in the homogametic sex (Table 2, row W[1], $h = 0$). An example of such a gene would be

TABLE 1. Summary of modeled parameters for an autosomal locus segregating for a sexually-antagonistic allele.

	Males*			Females		
Genotype	*A1A1*	*A1A2*	*A2A2*	*A1A1*	*A1A2*	*A2A2*
Zygote frequencies	$p_m p_f$	$p_m q_f + p_f q_m$	$q_m q_f$	$p_m p_f$	$p_m q_f + p_f q_m$	$q_f q_m$
Fitness (W)	$1 + S$	$1 + hS$	1	$1 - T$	$1 - hT$	1

$\bar{W}_m = 1 + S(p_m p_f + h(p_m q_f + p_f q_m))$
$\bar{W}_f = 1 - T(p_m p_f + h(p_m q_f + p_f q_m))$

* The assignment of sex in this model is arbitrary.

one that increased horn size in both sexes of bighorn sheep during the period when sexual dimorphism in horn size was just starting to evolve. Because individuals of the heterogametic sex (males in bighorn sheep) are hemizygous for sex-linked genes and individuals of the homogametic sex are either homozygous or heterozygous, the ratio of the level of expression of the sexually-antagonistic trait in the two sexes would be,

$$\frac{\%\text{ heterogametic individuals expressing } A1'}{\%\text{ homogametic individuals expressing } A1'} = \frac{p_f}{p_f p_m} = p_m^{-1}.$$

When p_m and p_f are small the level of expression in the heterogametic sex will be much larger than that in the homogametic sex. For example, if $p_f = p_m = .001$, then the proportion of heterogametic individuals expressing a recessive sexually-antagonistic trait would be 1,000-times the proportion of homogametic individuals expressing the same trait. As shown below, this sex-specific difference in the level of expression of a recessive sexually-antagonistic gene can greatly enhance the ability of an *X*-linked gene to increase when rare.

The equilibrium, convergence, and stability of the frequency of *X*-linked genes have been analyzed by Haldane (1926), Bennett (1958), Parsons (1961), Haldane and Jayakar (1964), Cannings (1967), and others. Adapting the formulation of Parsons (1961 p. 105) to the fitness model in Table 2 (row [*W*1]) a sexually-antagonistic gene will increase when rare whenever the following inequality is met,

$$S > \frac{2hT}{1 - hT}. \tag{3}$$

The biological interpretation of this inequality is that when the gene *A1'* is at least partially dominant (i.e., $h > .5$) then the conditions required for an *X*-linked gene to increase when rare are more restrictive than those for an autosomal gene, i.e., $2hT/(1 - hT) > T$. If the gene is sufficiently recessive (i.e., $h < 1/(2 + T)$), however, then *X*-linkage enables a sexually-antagonistic gene to increase when rare even if the fitness cost to the homogametic sex is larger than the gain to the heterogametic sex. When the sexually-antagonistic gene is fully recessive ($h = 0$), then inequality (3) reduces to $S > 0$. Thus an *X*-linked recessive gene that benefits the heterogametic sex will increase when rare, even if it is lethal when homozygous in the homogametic sex.

The equilibrium frequency ultimately achieved by a sexually-antagonistic gene depends on the values of *S*, *T*, and *h*. Because selection operates in a sex-specific manner, the equilibrium value of *p* is different between the sexes. Adapting equations (1) and (2) of Bennett (1958) to the fitness model specified in Table 2 (row *W*[1]), the equilibrium values of *p* are given by

TABLE 2. Summary of modeled parameters for an X-linked locus segregating for a sexually-antagonistic allele.

	Heterogametic sex (m)		Homogametic sex (f)		
Genotype	$A1$	$A2$	$A1A1$	$A1A2$	$A2A2$
Zygote frequencies	p_f	q_f	$p_m p_f$	$p_m q_f + p_f q_m$	$q_m q_f$
Fitness					
$W(1)$	$1 + S$	1	$1 - T$	$1 - hT$	1
$W(2)$	$1 - T$	1	$1 + S$	$1 + hS$	1

$$\hat{p}_f = \frac{S - hT(2 + S)}{2T[(1 + S) - hT(2 + S)]} \tag{4}$$

$$\hat{p}_m = \frac{S^2(1 - hT) + S(1 - 3hT) - 2hT}{S^2(1 - hT) + ST(2 - 4h) + T(2 - 4h)} . \tag{5}$$

Equations (4) and (5) can be used to examine the relationship between the equilibrium frequency of the gene $A1'$, the level of dominance (h), and the level of sexual-antagonism (Fig. 1). The level of sexual-antagonism is expressed as the ratio of the cost to the homogametic sex divided by the gain to the heterogametic sex, i.e., T/S. The value of $\hat{p}$ depends on the absolute values of S and T in addition to their ratio. For values of $S < .05$ the solid curves in Figures 1 and 2 represent a close approximation for $\hat{p}$. For larger values of S the equilibrium values of p are reduced (Fig. 1, dashed curves).

As illustrated in Figure 1, the equilibrium frequency of a completely recessive sexually-antagonistic gene will be substantial even when the cost to the homogametic sex is 50-times greater than the gain to the heterogametic sex. The gene $A1'$ need not be fully recessive to attain a relatively high equilibrium frequency. Even when h is as large as .2, $\hat{p}$ will exceed 2% for values of $T/S < 2$. I conclude from this analysis that X-linkage facilitates the initial buildup of recessive and partially recessive sexually-antagonistic genes that favor the heterogametic sex.

Advantage to the Homogametic Sex

X-linkage can also facilitate the increase in frequency of sexually-antagonistic genes that favor the homogametic sex. Consider the gene $A1''$ which I will initially assume to be fully dominant (Table 2, Row $W[2]$, $h = 1.0$). The proportion of individuals expressing the trait will be p_f in the heterogametic sex and $p_f + p_m q_f$ in the homogametic sex. The proportion of individuals expressing the trait is higher in the homogametic sex because ⅔ of the X-chromosomes reside in homogametic individuals compared to only ⅓ in heterogametic individuals. Thus dominant sexually-antagonistic genes that favor the homogametic sex are expressed at a higher rate in the sex where they are selectively favored.

The conditions required for $A1''$ to increase when rare can be obtained from inequality (3.6) of Parsons (1961),

$$S > \frac{T}{h(2 - T)} , \tag{6}$$

where S, T, and h are as defined in Table 1, row $W[2]$. This inequality indicates that when the gene $A1''$ is sufficiently dominant (i.e., $h > 1/(2 - T)$) the requirements for increase when rare are less stringent than those for an autosomal gene, i.e., $T/[h(2 - T)] < T$. When the trait is fully dominant ($h = 1.0$) and S and T are small, inequality (6) is approximated by $S > T/2$. Thus when S and T are small, the disadvantage to the

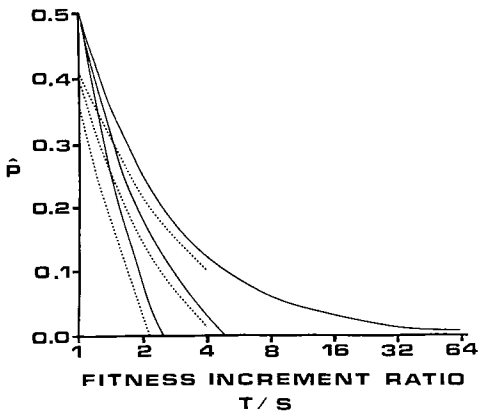

FIG. 1. The equilibrium frequency ($\hat{p}$) of a sexually-antagonistic gene ($A1'$) that increases the fitness of the heterogametic sex by an increment of S and decreases the fitness of the homogametic sex by an increment of T when homozygous and hT when heterozygous, plotted against the ratio T/S. The solid curves are appropriate when the value of S is small (i.e., < .05). The dashed curves illustrate the reduction in $\hat{p}$ when S is increased to .25. For each set of three curves $h = .0$ (top), $h = .1$ (middle), or $h = .2$ (bottom).

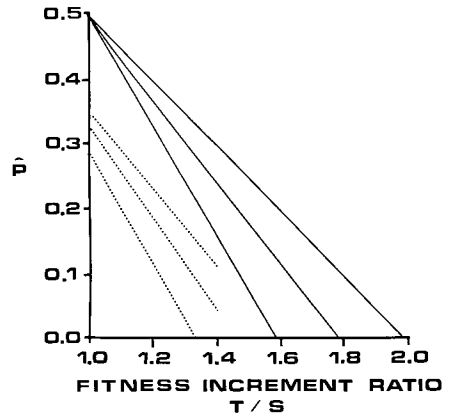

FIG. 2. The equilibrium frequency ($\hat{p}$) of a sexually-antagonistic gene ($A1''$) that increases the fitness of the homogametic sex by an increment of S when homozygous and hS when heterozygous and decreases the fitness of the heterogametic sex by an increment of T, plotted against the ratio T/S. The solid curves are appropriate when the value of S is small (i.e., < .05). The dashed curves illustrate the reduction in $\hat{p}$ when S is increased to .25. For each set of three curves $h = 1.0$ (top), $h = .9$ (middle), or $h = .8$ (bottom).

heterogametic sex can be almost twice as large as the advantage to the homogametic sex for the gene $A1''$ to increase when rare.

The equilibrium value of p is determined by adapting equations (1) and (2) of Bennett (1958) to the fitness model given in Table 1, row $W[2]$,

$$\hat{p}_f = \frac{2 - T - \left[\frac{2}{1 + hS}\right]}{4 - 2T - \left[\frac{2 + 2(1 - T)(1 + S)}{1 + hS}\right]} \tag{7}$$

$$\hat{p}_m = \frac{(1 - T)\left(2 - T - \left[\frac{2}{1 + hS}\right]\right)}{(1 - T)\left(2 - T - \left[\frac{2}{1 + hS}\right]\right) + 2 - T - \left[\frac{2(1 + S)(1 - T)}{1 + hS}\right]}. \tag{8}$$

As illustrated in Figure 2, the dominance of $A1''$ need not be complete to establish equilibrium values of $\hat{p}$ that are relatively large. Compared to the case of a recessive or partially recessive gene favoring the heterogametic sex, a dominant or partially dominant sexually-antagonistic gene favoring the homogametic sex requires smaller values of T/S to increase when rare. When dominance is nearly complete, however, the maximum value of T/S permitting an X-linked sexually-antagonistic gene to increase when rare is twice as large as the comparable value for a gene at an autosomal locus.

To summarize up to this point, X-link-

TABLE 3. Fitness values for all possible genotypes at a sex-linked sexually-antagonistic locus (A-alleles) and an autosomal, sex-limited modifier locus (M-alleles).

Genotype	Fitness	
	Heterogametic sex favored	Homogametic sex favored
Heterogametic sex		
— *A1 M1 M1*	$1 + S$	$1 - KT$
— *A1 M1 M2*	$1 + S$	$1 - (h'KT + (1 - h')T)$
— *A1 M2 M2*	$1 + S$	$1 - T$
— *A2 M1 M1*	1	1
— *A2 M1 M2*	1	1
— *A2 M2 M2*	1	1
Homogametic sex		
A1 A1 M1 M1	$1 - KT$	$1 + S$
A1 A1 M1 M2	$1 - (h'KT + (1 - h')T)$	$1 + S$
A1 A1 M2 M2	$1 - T$	$1 + S$
A1 A2 M1 M1	$1 - KhT$	$1 + hS$
A1 A2 M1 M2	$1 - (h'KhT + (1 - h')hT)$	$1 + hS$
A1 A2 M2 M2	$1 - hT$	$1 + hS$
A2 A2 M1 M1	1	1
A2 A2 M1 M2	1	1
A2 A2 M2 M2	1	1

age facilitates the initial increase in frequency of sexually-antagonistic genes when the disadvantage to one sex exceeds the gain to the other. These genes do not typically increase to fixation but have intermediate equilibrium gene frequencies. To produce sexual dimorphism, sexually-antagonistic genes ultimately must be fixed and their expression become sex-limited. The evolution of modifier genes, that restrict expression of sexually-antagonistic genes to the sex where they are selectively favored, can bring about both of these prerequisites.

SEX-LIMITED MODIFIERS

Once a sexually-antagonistic gene begins to increase in frequency there will be selection for sex-limited gene expression. The next step in the evolution of sexual dimorphism is the buildup of modifier genes that restrict the expression of sexually-antagonistic genes to the sex in which they are selectively favored. If such sex-limited modifiers were to evolve they would depress the average value of T, i.e., the fitness loss to the sex not favored by the sexually-antagonistic gene. Inspection of equations 4, 5, 7 and 8 indicates that as the value of T declines, the equilibrium frequency of a sexually-antagonistic gene increases, leading to fixation when T is sufficiently reduced.

An extensive series of simulations was carried out to determine: 1) Can sex-limited modifier genes, that reduce the expression of sexually-antagonistic genes in the sex where they are selected against, increase when rare? and 2) Can the increase in frequency of sex-limited modifier genes cause the fixation of sexually-antagonistic genes with intermediate equilibrium gene frequencies? The fitness model used in the simulations is given in Table 3. The results of the simulations were clear-cut. Whenever the equilibrium frequency of the sexually-antagonistic gene was >0, the sex-limited modifier gene (*M1*) increased when rare irrespective of the initial level of linkage disequilibrium between the A-alleles and the modifier alleles. Additional long-term simulations supported the idea that the increase in frequency of sex-limited modifier genes ultimately led to the fixation of sexually-antagonistic genes, provided that they had a non-zero equilibrium frequency in the absence of the

modifier. To save space the full details of the simulations have been deleted from this manuscript, but will be provided by the author upon request.

Conclusions and Predictions

I conclude from this analysis that the different frequency of expression of X-linked genes in the two sexes can facilitate the evolution of sexual dimorphism. If genes coding for sexually-antagonistic traits are dominant ($h > 1/[2 - T]$) and favor the homogametic sex or are recessive ($h < 1/[2 + T]$) and favor the heterogametic sex, then they will increase when rare even when the cost to one sex far exceeds the gain to the other. If genetic variation is present throughout the genome for a sexually-antagonistic trait, then X-linked genes will increase when rare under a much wider range of conditions compared to autosomal genes. As a result, X-linked sexually-antagonistic genes should be more likely to be involved in the evolution of sexually dimorphic traits. Once these genes begin to increase in frequency there will be natural selection for sex-limited modifiers that restrict the expression of sexually-antagonistic genes to the appropriate sex.

It is important to point out that I am not implying here that sex chromosomes are a prerequisite for the evolution of sexual dimorphism. Sexually dimorphic traits can evolve when sex chromosomes are absent via the "pleiotropy-mechanism" or via the "modifier-mechanism" when the advantage to one sex is greater than the disadvantage to the other. The effect of sex chromosomes is to expand the range of traits that can evolve via the "modifier-mechanism"

The major prediction from this analysis is that genes coding for sexually dimorphic traits should be located disproportionately on the X-chromosome. That is, the proportion of X-linked genes coding for sexually dimorphic traits will exceed the proportion of the total functional genome located on the X-chromosome. There are a number of empirical studies that support the prediction that X-linked genes play a dominant role in coding for sexually dimorphic traits (see, for example, Winge, 1927; Kallman, 1975; Templeton, 1977; Val, 1977; Grula and Taylor, 1980*a*, 1980*b*). For example, Grula and Taylor (1980*a*, 1980*b*), working with two species of hybridizing sulfur butterflies (*Colius eurytheme* and *C. philodice*), found that the majority of genetic variability controlling sexual dimorphism in body color, pheromones, and mating behavior was X-linked and sex-limited in expression. The X-chromosome in each of these species made up less than 3% of the haploid genome. This correlation as well as others, however, may be caused by factors unrelated to the ideas presented here. For example, Charlesworth and Charlesworth (1980) have shown that translocations of sexually-antagonistic autosomal genes to the sex chromosomes can be selectively favored under certain conditions. A direct test of the prediction could be achieved by artificial selection for sexual dimorphism in the laboratory coupled with testing to see if X-linked genes disproportionately produced the response to selection. The conclusion from the logical arguments presented here is that the evolutionary significance of the sex chromosomes probably involves not only the mechanism of sex determination but also the evolution of sexually dimorphic traits.

Summary

An analytical model was used to explore the effect of X-linkage on the evolution of sexual dimorphism. The model examined the requisite conditions for increase when rare, and the equilibrium frequency of genes that produce a "sexually-antagonistic" phenotype, i.e., a phenotype that is selectively favored in one sex but disfavored in the other sex. A simulation model was used to examine the evolution of sex-limited modifier genes that restrict the expression of sexually-antagonistic genes in the sex where they are selected against. The conclusion from this analysis is that sex chromosomes facilitate the evolution of sexual

dimorphism and that *X*-linked genes have a predominant role in coding for sexually dimorphic traits.

ACKNOWLEDGMENTS

I thank Brian Charlesworth, Steve Gaines and Michael Turelli for many helpful comments on an earlier draft of this manuscript. I also thank the Ecology Lunch Seminar Group at UCD for helpful comments on the ideas presented here. This work was supported in part by research funds provided by the Zoology Department of The University of California at Davis.

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