

Preferential Sex Linkage of Sexually Selected Genes: Evidence and a New Explanation

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Summary. Here, I review the evidence showing that the X chromosome has a disproportional share concerning the inheritance of sexually selected traits in animals with heterogametic males, and suggest a new explanation that relates this X bias with female choice of heterozygotic males. With numeric simulations I show that female choice of heterozygotic males is usually disadvantageous. Because this disadvantage cannot occur when females prefer X-linked male traits, preferential X linkage of sexually selected traits can be expected. As an alternative to fluctuating selection on sex-limited traits the disadvantage of heterozygotic choice may thus explain the X bias observed for sexually selected traits.

INTRODUCTION

INITIALLY BASED ON the inheritance of sexually selected traits in single species, some authors have proposed that genes coding for traits involved in speciation (Ewing, 1969) and sexual selection (Reinhold, 1994) are biased towards the X chromosome. In a qualitative literature review, however, Charlesworth *et al.* (1987) found no evidence of a special role of sex-linked genes in species recognition. Recently, two quantitative literature reviews have shown that there is a substantial X bias for sex- and reproduction-related genes in humans (Saifi & Chandra, 1999) and for sexually selected traits in animals with heterogametic males (Reinhold, 1998). Based on a database search, Saifi & Chandra (1999) concluded that in humans the proportion of loci related to sex or reproduction is significantly higher on the X chromosome than on the autosomes. For three groups of animals, *Drosophila*, other insects with heterogametic males and mammals, Reinhold (1998) compared the influence of X chromosomal genes on sexually selected male traits and on traits supposedly not under sexual

selection. Regarding sexually selected traits, about one-third of the difference between closely related taxa is coded by X chromosomal genes, whereas the X chromosomal contribution is negligible regarding traits classified as not under sexual selection. Those studies not included in these two reviews, for example because they appeared after these reviews or because some data were not included in the analysis, are also generally in accordance with these findings. The X chromosome has a disproportional effect regarding the inheritance of sexually selected song traits in the bushcricket *Ephippiger ephippiger* (Ritchie, 2000) and various *Drosophila* hybrids (Noor & Aquadro, 1998; Hoikkala *et al.*, 2000; but see Ritchie & Kyriacou, 1996; Boake *et al.*, 1998). Other sexually selected traits in *Drosophila*, the sex combs and cuticular hydrocarbon pheromones, also seem to be strongly sex linked (Khadem & Krimbas, 1997; Blows & Allan, 1998; Scott & Richmond, 1998). Male agonistic behaviour in the desert spider *Agelenopsis aperta* and male display behaviour in the fiddler crab *Uca*, traits likely to be under sexual selection, are largely determined by sex-linked loci (Salmon & Hyatt, 1979; Riechert & Maynard Smith, 1989). In accordance with the above reviews, no disproportional effects of X chromosomal genes on sexually selected male traits were detected in birds (*Philomachus pugnax*: Lank *et al.*, 1999) where males are homogametic.

An X bias such as the one observed by Saifi & Chandra (1999) and Reinhold (1998) can be expected for several theoretical reasons, for example sexually antagonistic selection of fluctuating selection. When traits are under sexually antagonistic selection (Rice, 1984), i.e. when one sex would benefit from an increased and the other sex from a decreased trait size, trait expression already differs between the sexes when X chromosomal genes are involved. Under such a selection regime, X chromosomal genes therefore provide the raw material for selection to work with and will therefore be particularly likely to contribute to traits under sexually antagonistic selection. However, most of the examined traits are sex limited in their expression, i.e. females do not express those sexually selected male traits, and the antagonistic selection explanation thus cannot easily be applied. Another possible explanation relates to the difference in expression of autosomal and X chromosomal sex-limited genes. As in heterogametic males one-third of the X chromosomal genes and one-half of the autosomal genes are exposed to selection, fluctuating selection should favour the invasion of X chromosomal mutations (Reinhold, 1999).

Here, I have tried to show that the observed X-bias might also result from selection against female choice of heterozygotic males. If heterozygote advantage is frequent and if female choice is disadvantageous, there will be selection against female choice of males when attractiveness of males is correlated with heterozygote advantage. As heterozygote advantage cannot occur for X chromosomal traits, this disadvantage will not occur for X chromosomal traits and under this scenario one can thus expect to find an X bias for sexually selected traits.

In many animal species, females choose healthy males with high growth rates and developmental stability (reviewed by Andersson, 1994). On the other hand, heterozygosity has been shown to be associated with superior survival, disease resistance, high growth rates and developmental stability (Allendorf & Leary, 1986). Inspired by this pattern, Borgia (1979) and Brown (1997) suggested that females might benefit from choosing heterozygotic males. One problem for this hypothesis is that, at equilibrium, offspring viability does not increase when females preferentially mate with heterozygotic males having superior viability (Partridge, 1983). Offspring viability does not increase at equilibrium because heterozygosity is, as Brown (1997) correctly noted, not directly heritable (but see Mitton *et al.*, 1993). In accordance with the hypothesis that choice of heterozygotic males increases offspring fitness, a theoretical analysis by Charlesworth (1988) suggested that female choice for heterozygotic males might evolve when environmental conditions fluctuate. In these simulations underdominance of viability was assumed and heterozygotes were modelled to be almost as viable as the better one of the homozygotes. Viability values were switched between the homozygotes at regular intervals to mimic temporal environmental fluctuations, and the relative viability of the heterozygotes was kept constant (Charlesworth, 1988). As a result of the higher geometric mean fitness of heterozygotes, an increase in the frequency of the allele causing females to choose heterozygotic males was observed in most but not all simulations. In similar simulations, Mitton (1997) assumed overdominance of heterozygotic males and showed that female choice for heterozygotic males can evolve under temporally fluctuating selection. An allele causing female choice for heterozygotic males is, in contrast, unlikely to increase in frequency when constant viabilities are assumed and when the allele occurs in initially low frequency (Heisler & Curtsinger, 1990). In a recent analytical model, Irwin & Taylor (2000) showed that fluctuating selection is a necessary condition for the evolution of heterozygotic choice.

With the following model I also examined whether a female choice allele can increase in frequency when it causes females to choose heterozygotic males that have superior viability due to overdominance. The simulations show that female choice for heterozygotic males with superior viability will only evolve under restricted environmental conditions.

METHODS

The evolution of female choice for superior heterozygotic males was examined by a population genetic model for a diploid organism with discrete generations and a population of infinite size. Two alleles, A and B at one locus, were assumed to influence male viability before mating, and heterozygotic males

were assumed to have higher viability than both types of homozygotic males. The relative viability of AA males was assumed to be $V_{AA} = 1 - s$, the relative viability of BB males was assumed to be $V_{BB} = 1 - t$ ($0 < s, t \leq 1$), and the relative viability of heterozygotic males was assumed to be $V_{AB} = 1$. Female viability was assumed to be independent from the male viability allele. A female choice allele, C, that causes absolute choice of heterozygotic males was introduced in low frequency in linkage equilibrium with the viability alleles so that 1% of all females showed preference for heterozygotic males. All other females were assumed to mate at random with the available males. The choice allele and the trait alleles were assumed to be unlinked, and the allele causing female choice was both modelled to be dominant and recessive against the no-choice allele. The evolution of female choice of superior heterozygotic males was examined for three different types of environmental variability: (1) stable environmental conditions leading to stable viability disadvantages of the homozygotic males; (2) environmental fluctuations leading to random fluctuations of the viability disadvantages of homozygotic males within a given interval; (3) the size of the viability disadvantage of homozygotic males was assumed to vary randomly as an effect of environmental fluctuations.

Stable environment

The evolution of a rare choice allele was modelled for a range of different values ($0.01 \leq s \leq 0.9$) for the viability of homozygotic AA males, while the viability of homozygotic BB males was assumed to be 0.5 or 0.8 times the viability of heterozygotic males. The relative change in the frequency of the C allele was calculated as the frequency of the C allele at generation 150 divided by the frequency of C at generation 50. The logarithm of this relative change in the frequency of the choice allele is given as the result. Linkage disequilibrium between the choice and viability alleles was calculated as D/D_{\max} (Maynard Smith, 1989).

Environmental fluctuations within a given interval

In these simulations, viability of homozygotic AA males was assumed to vary as an effect of temporal fluctuations in environmental conditions. Viability was modelled to vary randomly within an interval of ± 0.1 around a given mean (i.e. values were chosen from a uniform distribution each generation). Viability of homozygotic BB males was assumed to vary in the same way between 0.4 and 0.6 times (or 0.7 and 0.9 times) the viability of heterozygotic males. The influence of these fluctuations on the frequency of the choice allele was examined. Viabilities of AA males were assumed to vary independently from the viability of BB males. The logarithm of the relative change in the frequency of the

choice allele between generations 50 and 1050 is given as the result. In comparison to a stable environment, a 10-fold number of generations was used, otherwise chance would have a large influence on the frequency of the choice allele due to the stochastic nature of the modelled environmental fluctuations.

Environmental fluctuations of different variance

Viabilities of homozygotic AA males were assumed to vary around a mean value of $1 - s$. The influence of environmental fluctuations on the viability of AA males was modelled by $V_{AA} = (1 - s) + s * X * R_1$, where R_1 is a number between -1 and 1 chosen randomly each generation, and where X , a value between 0 and 1 , gives the strength of temporal fluctuations influencing the viability disadvantages of the homozygotes. With a maximum X of 1 the viability of homozygotic AA males was assumed to vary between 1 and $2s$, and the viability of heterogametic males was assumed to be 1 . With a minimum X equal to 0 , the viability of AA males was assumed to be constant at $1 - s$. Viabilities of homozygotic BB males were accordingly assumed to vary around a mean value by $V_{BB} = (1 - t) + t * X * R_2$. For the simulations shown in Figure 3, AA males were assumed to have a mean viability of 0.5 and BB males were assumed to have a mean viability of 0.8 . The viabilities of AA males were assumed to vary independently from the viability of BB males. The logarithm of the relative change in the frequency of the choice allele between generations 50 and 1050 is given as the result. To account for the stochastic nature of the modelled environmental fluctuations, 1000 generations were again used to estimate the change in the frequency of the choice allele.

RESULTS

The female choice allele was neutrally stable when both types of homozygotic males were assumed to have the same viability (Figure 1). For all other viability values the frequency of the female choice allele causing mate choice of heterozygotic males decreased markedly (Figure 1). The observed decrease was largest for extreme differences in viability between the two types of heterozygotic males. The size of the linkage disequilibrium between the choice allele and the viability alleles showed a similar dependence on the difference between homozygote viabilities. Linkage disequilibrium was zero when the homozygotes had identical viabilities, and increased monotonically when larger differences between homozygote viabilities were assumed (Figure 2). For all assumed viabilities of males, the observed decrease in the frequency of the female choice allele was larger for a dominant choice allele than for a recessive choice allele (Figure 1).

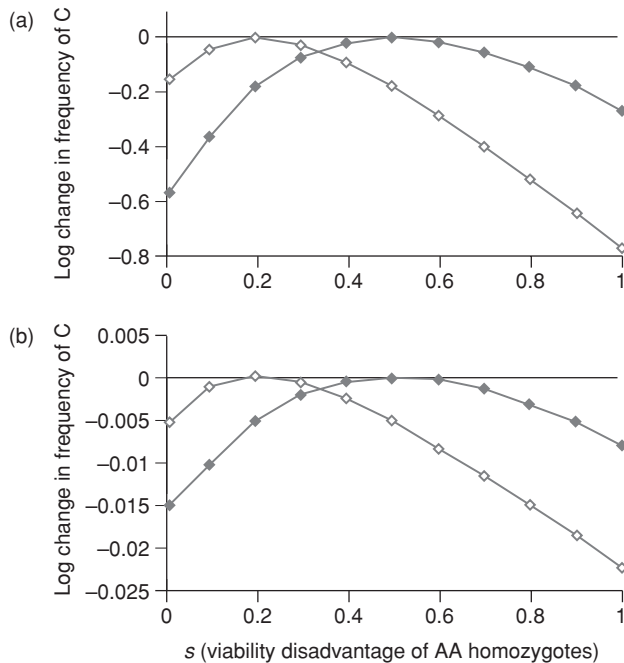


Figure 1. Effect of viability of homozygotic AA males on the frequency of the choice allele C under the assumption of stable viabilities (viability of BB males: open diamonds, $1 - t = 0.8$; closed diamonds, $1 - t = 0.5$). The relative change in the frequency of C is given as logarithm of the ratio between the frequency at generation 150 and the frequency at generation 50, (a) for a dominant choice allele, (b) for a recessive choice allele.

Assuming random variation in the viability of homozygotic males within a given interval, a small increase in the frequency of the choice allele could be observed when the average viabilities of the two types of homozygotic males were assumed to be similar (Figure 3). The frequency of the choice allele decreased when the average viabilities of the two types of males differed by about 0.1 or more. The disadvantage of the choice allele increased with increasing difference between the mean viabilities of AA and BB males (Figure 3). Given mean viabilities of AA and BB males differing by 0.2 or more, the observed decrease in the frequency of the choice allele was large compared with the observed increase in the frequency of the choice allele when the viabilities of the homozygotes were assumed to be similar. This pattern, with a small increase in the frequency of the choice allele when homozygotes had similar viabilities and a large decrease in the frequency of the choice allele when homozygotes had dissimilar viabilities, occurred both with a dominant and with a recessive choice allele. The change in the frequency of the choice allele was smaller when the choice allele was assumed to be recessive against the no-choice allele.

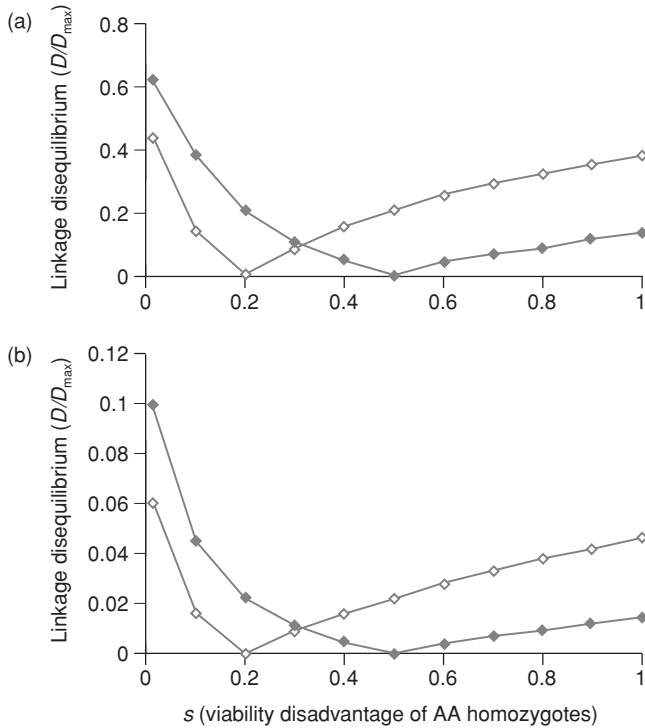


Figure 2. Effect of the viability of homozygotic AA males on the linkage disequilibrium between choice allele and viability alleles under the assumption of stable viabilities (viability of BB males: open diamonds, $V_{BB} = 1 - t = 0.8$; closed diamonds, $V_{BB} = 1 - t = 0.5$), (a) for a dominant choice allele, (b) for a recessive choice allele.

The outcome of the simulations was similar when temporal fluctuations of different strength were assumed. When mean viabilities of homozygotic males were assumed to differ substantially, the frequency of the choice allele decreased even under maximum variability of homozygotic viabilities (Figure 4). However, the disadvantage of the choice allele decreased with increasing strength of temporal fluctuations (Figure 4). As in the other simulations the decrease in the frequency of C was smaller for a recessive choice allele than for a dominant choice allele.

CONCLUSIONS

It was suggested that females should be able to increase offspring fitness by choosing heterozygotic males with superior viability for mating (Borgia, 1979;

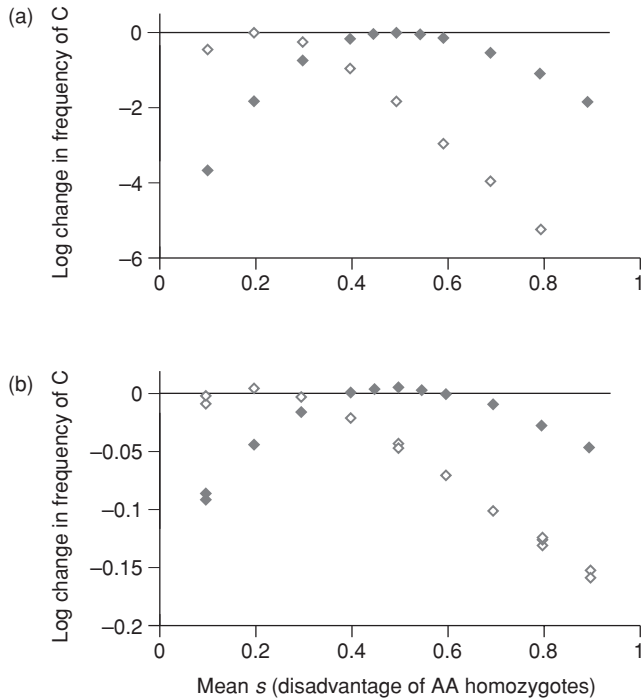


Figure 3. Effect of mean viability of homozygotic AA males on the frequency of the choice allele C under the assumption that viabilities vary randomly by ± 0.1 around the mean (mean viability of BB males: open diamonds, $V_{BB} = 1 - t = 0.8$; closed diamonds, $V_{BB} = 1 - t = 0.5$). The relative change in the frequency of C is given as the logarithm of the ratio between the frequency at generation 1050 and the frequency at generation 50. For each value of the parameter s the results of three replicate simulations are shown, (a) for a dominant choice allele, (b) for a recessive choice allele.

Charlesworth, 1988; Brown, 1997). With overdominance and constant relative viabilities of homozygotic ($V_{AA} = 1 - s$; $V_{BB} = 1 - t$) and heterozygotic ($V_{AB} = 1$) males, there is an equilibrium frequency for the viability alleles. For any values of $s > 0$ and $t > 0$, maximum average viability of males is $V_{\max} = 1 - (st/(s + t))$ when the frequency of the A allele is equal to $t/(s + t)$ (Partridge, 1983). At equilibrium the viability of male offspring of females choosing heterozygotic males is equal to $1 - (st/(s + t))$ (Partridge, 1983). Thus, offspring viability does not change when females choose heterozygotic males for mating, and female choice seems to be neutral under the assumption of a constant environment. However, these simulations show a disadvantage for females choosing heterozygotic males with superior viability. This disadvantage is caused by a linkage disequilibrium between the choice allele and the less frequent viability allele that is built up by female choice for heterozygotic males. Female choice of

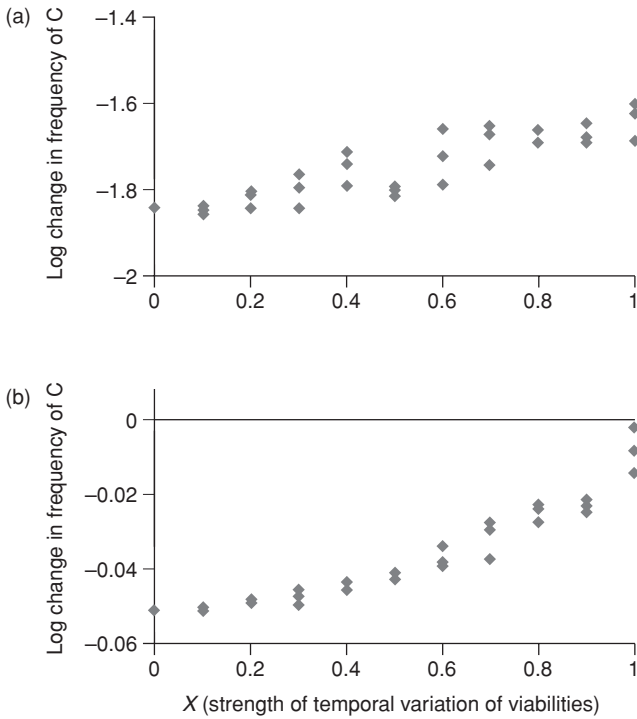


Figure 4. Effect of temporal fluctuations of different strength on the frequency of the choice allele under the assumption that viabilities of homozygotic males are influenced by these fluctuations. The extent of temporal fluctuation of homozygote viabilities is given by X , and homozygote viabilities are assumed to vary randomly by $(1 - s) \pm s^*X$ and $(1 - t) \pm t^*X$ around the mean ($s = 0.5, t = 0.2$). The relative change in the frequency of the choice allele is given as logarithm of the ratio between the frequency at generation 1050 and the frequency at generation 50. For each value of X the results of three replicate simulations are shown, (a) for a dominant choice allele, (b) for a recessive choice allele.

heterozygotic males gives an advantage to the less frequent viability allele, because the frequency of the rare allele is higher in the preferred males than in the overall population. As a result, the female choice allele becomes associated with the less frequent allele. This association, together with the reduced viability of the offspring homozygous for the less frequent viability allele, leads to a selection disadvantage of the choice allele compared with the no-choice allele.

When females choose heterozygotic males, the frequency of the less frequent viability allele increases because heterozygotic males have one copy of this allele. When the choice allele occurs at substantial frequency, female choice will cause a deviation of the viability alleles away from the equilibrium frequency towards a 1 : 1 ratio. This is an additional disadvantage for female

choice of heterozygotic males because any deviation in the frequency of the A allele by ε from equilibrium frequency reduces mean male viability by $\varepsilon^2/(s + t)$.

When s and t are assumed to be equal, the equilibrium frequency of allele A is 0.5 and female choice of heterozygous males cannot cause a linkage equilibrium or a deviation from the equilibrium allele frequencies. For this reason the decrease of the choice allele was smallest for those cases where s and t were similar. An increase in the frequency of the choice allele could not be observed for any combination of homozygote viabilities when a stable environment was assumed.

The pathway for the evolution of female choice for heterozygotic males suggested by Brown (1997) thus seems to be impossible for temporally stable viabilities. Charlesworth (1988) showed that female choice for heterozygotic males may evolve when environmental conditions fluctuate. However, he only used symmetrical viabilities of the homozygotes in his simulations and the long-term fitness of the two types of homozygotes was thus modelled to be identical. According to the simulations presented here, female choice for heterozygotic males may only be advantageous when homozygote viabilities are similar. Inferred from data on allele frequencies, similar viabilities of the homozygotes seem to be rare in cases with overdominance (Mitton *et al.*, 1993). If temporal fluctuations in the viabilities of homozygotic males were assumed together with similar average viabilities of the two types of homogametic males, the allele causing female choice of heterozygous males increased in frequency. But even in these cases the increase in the frequency of the choice allele was small compared with the decrease that was observed when viabilities of the two types of homozygotes differed. Thus, the evolution of female choice for heterozygotic males with superior viability seems to be restricted to special environmental conditions.

According to the above theoretical analysis, female choice of heterozygotic males should be rare because it is restricted to special environmental conditions. Does the empirical evidence concerning female choice for heterozygous males fit this expectation? There are several studies that are often cited as evidence for female choice of heterozygous males (*Littorina* snails: Rolán-Alvarez *et al.*, 1995; *Danaus* butterflies: Smith, 1981; *Colias* butterflies: Watt *et al.*, 1986; *Artemia* shrimps: Zapata *et al.*, 1990). All these studies have shown that heterozygous males have a mating advantage but they have not shown that females prefer heterozygous males. The mating advantage of heterozygous males shown in these studies can also be explained by an advantage of heterozygous males in finding or acquiring mating partners due to their superior physiology or behaviour resulting from heterosis (Brcic & Koref-Santibañez, 1964). There is thus no clear evidence that the observed mating advantage involved female choice for heterozygotic males. Empirical evidence for female choice of heterozygotic males is therefore at least equivocal and female choice for

heterogametic males may be rare, in accordance with the results of the model presented here.

Due to the simplistic conditions used in the simulations, some outcomes of the model may not hold for the more complicated conditions that might occur in nature. The main assumption of the model examined here, choosy females mating only with males that are heterozygotic at a single locus, is clearly unrealistic with respect to female choice in nature. Females have been proposed to select males for their overall heterozygosity at a large number of loci (but see Watt *et al.*, 1986). One should be careful to extrapolate the results of a one-locus simulation to a multilocus system. However, the cause of the disadvantage of female choice in the analysed one-locus system (the linkage disequilibrium) should also occur in a multilocus system. Female choice of heterozygotic males favours rare alleles because rare alleles occur in higher frequency among heterozygotic males than in the overall population. A linkage disequilibrium will consequently build up between choice allele and rare trait alleles. In multilocus systems, the size of the linkage disequilibrium and therefore the disadvantage of female choice of heterozygotic males might, however, be much smaller than in one-locus two-allele systems. When females have a less strong preference for heterozygotic males (for example because they make mistakes in identifying heterozygotic males) or have some intermediate preference for one type of homozygotic males, this will change the size of the linkage disequilibrium and therefore the strength of selection against female choice. But, it will not alter the direction of selection, and female choice can, at best, be expected to be neutral when the chosen males have on average the same allele frequencies as the whole population. It should also be noted that the model rests on the assumption that the population is at equilibrium for the alleles determining male viability, and the results of the simulations will not be applicable when this condition is not met. Considering the limitations of the simulations used, I conclude that the selective disadvantage of female choice of heterozygotic males is also likely to occur under more realistic conditions.

Even when not actively choosing heterozygotic males, females might for some other reason prefer healthy and viable males, in accordance with the good genes hypothesis (for references see Andersson, 1994). If some part of the above-average viability of these attractive males is due to heterozygote advantage, selection against heterozygotic choice can decrease the benefit of choosing viable males. For the good genes mechanism to work, either the benefits of choosing viable males have to be larger than the cost of choosing heterozygotic males, or fluctuating selection is necessary to render heterozygotic choice adaptive. Let us assume that female choice of viable males is adaptive, heterozygotic choice disadvantageous, and that there is variation between females in the male traits they use to recognise male viability. Let us further assume that some females base their preference on traits that are largely determined by X

chromosomal genes, and some other females use autosomal traits as viability indicators. If heterozygote advantage is frequent and if the X chromosomal traits are as good as viability indicators as the autosomal traits, selection against heterozygotic choice should lead to an increase in the frequency of females preferring X chromosomal male traits. For those traits, the disadvantage of heterozygotic choice cannot occur because in heterogametic males there is only one X chromosome. Selection against heterozygotic choice should thus lead to an increased influence of X chromosomal genes on sexually selected traits, as has been observed empirically (Reinhold, 1998). Together with sex differential selection (Rice, 1984) and fluctuating selection on sex-limited traits (Reinhold, 1999), the disadvantage of heterozygotic choice thus provides an additional possible explanation of the X-bias observed for sex- and reproduction-related traits (Reinhold, 1998; Saifi & Chandra, 1999).

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DISCUSSION

Questioner: Does assortative mating come into this story? Humans have all sorts of assortative mating patterns, things that don't look like sexual selection at first sight, but clearly each sex is looking for something in the other.

Reinhold: Why should this be related to the X chromosome?

Comment: It may have nothing to do with the X chromosome and could be on autosomes.

Questioner: Could it be that there are two copies of the X in the female?

Reinhold: Yes, and therefore the expression is different in females than in males.

Questioner: What are the sex chromosomes in *Drosophila*. Do they have an X and Y as in humans?

Reinhold: Males are X0 and females are XX and there is no X inactivation. In the male; the single X is upregulated.

Comment: I wonder if you have considered imprinting, that is genes would be expressed when they are inherited from the mother, but not from the father. In the latter they would be silent.

Reinhold: There has not been any modelling of imprinting. It should be done. Up to now, the information is limited to mammals. However, I don't think this is likely to be an overall explanation for sexual selection.

Questioner: What is the relationship between speciation and sexual selection? Do you think the evidence is substantial?

Reinhold: Many characteristics are the same between species except for those that are sexually selected. These appear to be species specific. There is much evidence showing that sexual selection and speciation come together. For example in the cichlids, in African lakes, if there is a different colour pattern they don't mate. But if the colour can't be seen because the water is too turbulent, then they mate between the species and the species disappear. So sexual signals are very important in keeping species apart.

Comment: One consequence of having a gene on the X chromosome is that it is dominant in the males and recessive in females. So that it produces greater variation in the males than females. Is this advantageous for sexual selection?

Reinhold: Yes, definitely.

Comment: This difference in sex could have to do with the unequal distribution of language disorders, i.e. more often occurring in males. There are all sorts of sex differences in other disorders such as autism and schizophrenia.

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