

This allowed us to show, unequivocally, that normal participants may produce confabulatory reports when asked to describe the reasons behind their choices. More importantly, the current experiment contains a seed of systematicity for the study of choice and subjective report. The possibility of detailing the properties of confabulation that choice blindness affords could give researchers an increased foothold in the quest to understand the processes behind truthful report.

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# Sexual Selection Can Resolve Sex-Linked Sexual Antagonism

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Sexual selection is a potent evolutionary force. However, very few models have considered the evolution of female preferences for traits expressed in both sexes. Here we explore how female preferences coevolve with sexually antagonistic traits, which involve alleles that are beneficial to one sex but harmful to the other. We show that with a sexually antagonistic trait on the X chromosome (males XY, females XX), females evolve to prefer mates carrying alleles beneficial to daughters. In contrast, with a Z-linked trait (males ZZ, females ZW), females more often evolve mating preferences for mates carrying alleles beneficial to sons (that is, flashy displays).

Evolutionary biologists have long puzzled over how and why female preferences drive the evolution of exaggerated male traits. Generally, female preferences are thought to enhance a female's long-term fitness by increasing her offspring's fitness, either directly or through genetic associations between preference and trait loci (1, 2). Nearly all models assume that females do not initially express the male display trait, or else they assume that the fitness effects are the same in males and females (3). However, traits subject to sexual selection will often be sexually antagonistic, for example, with "sexy" male traits benefiting males but reducing female fitness (4-6). If a trait increases male reproductive success at the cost of female viability, females must then choose between having attractive sons (and unfit daughters) and having ugly sons (but unencumbered daughters). As long as females can detect the genotypic differences among males at sexually antagonistic loci, we expect mating preferences to evolve as described by the models explored here.

Theory predicts that sexually antagonistic loci are more likely to remain polymorphic on

the sex chromosomes (4, 5). Furthermore, recent empirical work suggests that many sexually selected traits in animals are located on the X chromosome (7, 8) and that most polymorphic sexually antagonistic traits are located on the X chromosome in *Drosophila* (5, 9). There is also evidence to suggest that a female's mate choice may result in a tradeoff in the fitness between her daughters and sons (10). Several recent theoretical examinations of the evolution of female preferences have explored sex linkage of the trait and/or preference (11-14). However, these models, with the exception of Reeve and Pfennig's (12),

assume that sexually selected traits have male-limited expression and therefore no fitness consequences when in females, and none has addressed sexual antagonism. Here we address the question of how female preferences evolve for traits that have contrasting fitness effects in each sex.

With sexual antagonism, chromosomal location should strongly affect the evolution of female preferences. Simply put, an X-linked male trait is never passed on from an attractive father to his sons, whereas his daughters suffer the cost of carrying the display trait (5, 9). Offspring in XY species therefore do not gain a fitness benefit from females preferring males with a more extreme X-linked display trait. In contrast, both males and females contribute a Z chromosome to sons in ZW species. Thus, females preferring a Z-linked display trait receive the fitness benefit of sexy sons, even though their daughters suffer a fitness cost (5, 9). This cost is lessened by the fact that daughters inherit only one of their father's Z chromosomes. With autosomal inheritance, these asymmetries in inheritance are absent.

To verify the verbal argument laid out above, we present the results of two-locus models that follow the fate of a newly arisen preference allele *p* in a population that is at a polymorphic equilibrium at a trait locus. We assume that

**Table 1.** Male and female fitness components in male heterogametic (XY) and female heterogametic (ZW) species.

	X-linked trait			Z-linked trait		
	<i>Male trait</i>					
		<i>T</i>	<i>t</i>	<i>TT</i>	<i>Tt</i>	<i>tt</i>
Female preference	<i>PP</i>	1	1 + $a_{pp}$	<i>P</i>	1 + $da_p$	1 + $a_p$
	<i>Pp</i>	1	1 + $a_{pp}$	<i>p</i>	1 + $da_p$	1 + $a_p$
	<i>pp</i>	1	1 + $a_{pp}$			
Male viability		1 - $s_y$	1	1 - $s_z$	1 - $hs_z$	1
	<i>Female trait</i>					
		<i>TT</i>	<i>Tt</i>	<i>tt</i>	<i>T</i>	<i>t</i>
Female viability		1	1 - $hs_x$	1 - $s_x$	1	1 - $s_w$
	<i>Female preference</i>					
		<i>PP</i>	<i>Pp</i>	<i>pp</i>	<i>P</i>	<i>p</i>
Female cost		1 - $ a_{pp} k$	1 - $ a_{pp} k$	1 - $ a_{pp} k$	1 - $ a_p k$	1 - $ a_p k$

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allele  $T$  is most fit in females (“female-benefit allele”) and allele  $t$  is most fit in males (“male-benefit allele”), with this tradeoff mediated by natural selection, sexual selection, or both. In the X-linked model, females with a  $tt$  genotype suffer a fitness disadvantage of  $1 - s_x$  relative to  $TT$  females, where  $s_x$  is the strength of selection in females, and the fitness of  $Tt$  females is given by  $1 - hs_x$ , where  $h$  is a dominance coefficient. In the absence of sexual selection, males carrying the  $T$  allele have a relative fitness of  $1 - s_y$  relative to males with the  $t$  allele (Table 1). Note that  $s_y$  can be positive or negative, allowing for natural selection either for or against the trait allele  $t$  in males. The mating preference of a female of genotype  $i$  at the preference locus is given by  $a_p$ , which describes the relative increase (or decrease) in the female’s probability of mating with males carrying allele  $t$  (Table 1) (3). We also allow for selection against the female preference by reducing fe-

male fitness by an amount proportional to her choosiness and a cost parameter,  $k$  (15). The fitness scheme in the Z-linked model is similar (Table 1). However, an additional parameter,  $d$ , is required to describe female preferences for heterozygous males when the preference locus is Z-linked. When both trait and preference loci are sex-linked, recombination occurs at a rate  $r$  between them; autosomal preference loci were also considered, in order to examine the influence of physical linkage on the results. Finally, both autosomal preference and trait loci were modeled. The analytical solutions for the polymorphic equilibria and the invasion rate of a new preference allele,  $p$ , are approximated assuming weak selection ( $s_x, s_y, a_p$ , and  $k$  are small).

There are three ultimate fates for sexually antagonistic genes: (i) the fixation of the allele with the higher fitness across both sexes, (ii) the evolution of sex-specific expression, or (iii) polymorphism (4, 5). A polymorphic equilib-

rium for an X-linked trait is maintained by sexually antagonistic selection in the X-linked model with allele  $P$  fixed as long as

$$2hs_x < (a_{pp} + s_y) < 2s_x(1 - h) \quad (1)$$

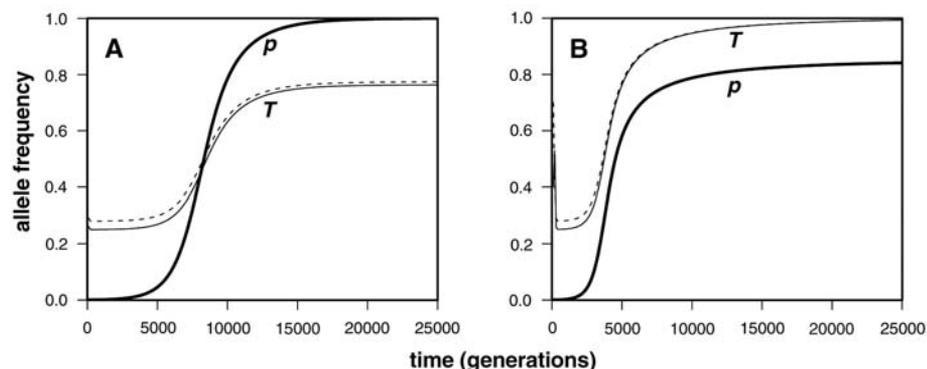
which requires that the male-benefit allele,  $t$ , be wholly or partially recessive in females ( $h < 1/2$ ). As long as Eq. 1 holds, the model allows for the maintenance of polymorphism either by sexually antagonistic natural selection ( $s_y > 0$ ) and/or by sexual selection opposing natural selection ( $a_{pp} > 0$ ). Similar criteria for maintaining a polymorphic equilibrium at a Z-linked trait locus are presented in (15).

Performing a stability analysis, a new preference allele,  $p$ , invades a population that is polymorphic for an X-linked trait locus whenever it confers a stronger preference for males bearing the female-benefit allele,  $T$  ( $a_{pp} < a_{pp}$ ). This result holds regardless of the physical location of the preference locus. Whereas recombination breaks apart genetic associations, it also places preference alleles on the same chromosome as the trait alleles that have been preferred and is therefore critical to the development of genetic associations. These two factors balance, causing the level of genetic associations to be fairly insensitive to the recombination rate.

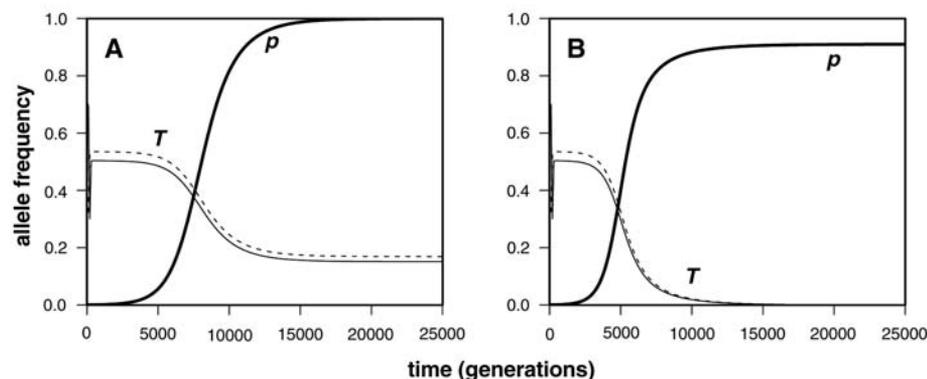
In contrast to the X-linked case, when both the trait and preference loci are Z-linked, allele  $p$  can invade a population only when it confers a stronger preference for the male-benefit allele,  $t$  ( $a_p > a_p$ ). Again, the recombination rate does not alter the range of conditions under which invasion occurs. However, when the trait locus is Z-linked and the preference locus is autosomal,  $p$  invades only when it increases the preference for the female-benefit allele,  $T$  ( $a_{pp} < a_{pp}$ ), as in the X-linked case. Adding a cost of female preference to both the X- and Z-linked models just slows the spread of stronger preference alleles, as long as the costs are not too strong relative to selection acting on the trait locus (15).

Finally, if all of the loci are autosomal, there is no longer any selection for females to prefer traits that help only one sex, and the invasion of any particular preference allele depends crucially on the level of linkage (15). This is consistent with a previous model (16), which found that sexual selection could resolve a polymorphism of a male-limited trait either in favor of  $T$  or  $t$ .

To understand the selective forces at work, we performed a quasi-linkage equilibrium analysis (17). When females carrying a new allele  $p$  preferentially mate with males carrying  $T$ , for example, a positive genetic association develops between the  $p$  and  $T$  alleles. In the homogametic sex (either XX females or ZZ males), both cis and trans linkage disequilibrium are present and approximately equal in magnitude, but only cis disequilibrium can be



**Fig. 1.** Simulation results for the evolution of female preferences in male heterogametic species (XY). All simulations were started under conditions allowing a polymorphic equilibrium at an X-linked trait locus with  $P$  fixed. (A) A new allele  $p$  that prefers males carrying the female-benefit allele,  $T$ , is introduced and sweeps to fixation, leaving  $T$  polymorphic at a higher frequency and improving the fitness of daughters ( $a_{pp} = 0.1$ ,  $a_{pp} = 0$ ,  $a_{pp} = -0.1$ ). (B) A preference allele favoring the  $T$  allele sweeps to high frequency, driving  $T$  to fixation. Sexual antagonism is thus completely resolved in favor of females ( $a_{pp} = 0.1$ ,  $a_{pp} = -0.1$ ,  $a_{pp} = -0.2$ ). Both loci are X-linked, with  $r = 0.5$ ,  $h = 0.1$ ,  $s_x = 0.2$ , and  $s_y = 0.2$ . The frequency of  $T$  is shown in thin curves, and males are shown with dashed curves. The frequency of  $p$  is in bold, with indistinguishable curves for males and females.



**Fig. 2.** Simulation results for the evolution of female preferences in female heterogametic species (ZW). (A) A new allele  $p$  that prefers males carrying the male-benefit allele,  $t$ , sweeps to fixation, driving  $t$  to high frequency and improving the fitness of sons ( $a_p = -0.1$ ,  $a_p = 0.08$ ). (B) A preference allele  $p$  favoring the  $t$  allele sweeps to high frequency, driving  $t$  to fixation ( $a_p = -0.1$ ,  $a_p = 0.2$ ). Sexual antagonism is thus completely resolved in favor of males. Both loci are Z-linked, with  $r = 0.5$ ,  $d = 0.5$ ,  $h = 0.2$ ,  $s_w = 0.224$ , and  $s_z = 0.3$ . The curve types are the same as in Fig. 1.

present in the heterogametic sex (either XY males or ZW females when the preference is Z-linked). Consequently, the new preference allele rises by association with *T* in XX females (in whom *T* is favored) by twice the amount that it goes down in XY males (in whom *t* is favored), explaining the spread of female preferences for the trait favored in females among XY species. Conversely, when both the trait and preference are Z-linked, the new preference allele declines by association with *T* in ZZ males (in whom *t* is favored) by twice the amount that it goes up in ZW females (in whom *T* is favored), explaining why sexual selection does not favor preferences for the allele beneficial to daughters.

When the trait is Z-linked but the preference locus is autosomal, the situation is slightly more complex. Both sons and daughters now inherit two copies of the preference allele, but daughters inherit only one copy of the trait allele. Because daughters' trait alleles are only inherited from their father, stronger trans disequilibrium develops between the preference allele inherited from their mothers and the trait allele inherited from their fathers. The stronger genetic associations that develop in daughters than in sons again favor the spread of preferences for the female-benefit allele, *T*.

The conditions for the invasion of a new preference allele were determined assuming weak selection. How robust are these results to stronger selection? Simulations with selection coefficients on the order of 10 to 30% were explored; selection coefficients in this range are not uncommon (18). Invasion depends only on how the new preference allele changes female mating preferences and not on the strength of selection, confirming our analytical results (Figs. 1 and 2)

Our results point to a potentially large effect of the sex-determination mechanism on how female preferences evolve for sexually antagonistic traits. Over long time scales, evolutionary changes in female preferences will lead to the fixation of the trait alleles most fit in females in XY systems and ZW systems when the preference is autosomal, but the trait allele most fit in males in ZW systems with Z-linked preferences. Thus, sexually antagonistic selection is always resolved in favor of females in XY species (Fig. 1B), but in favor of males in ZW species when the preference is Z-linked (Fig. 2B). This process can occur very rapidly if the new allele has a strong effect on preferences.

Assuming the conditions of our model, we predict that the difference in fitness between daughters and sons should be greater for females that choose mates relative to females mating at random. Choosy females should produce daughters that are more fit than sons in XY systems and vice versa in ZW systems. Another prediction of our model is that female preferences can evolve more easily for male-benefit alleles in ZW species, which is con-

sistent with the greatly exaggerated displays observed in groups such as birds and butterflies (12). This prediction calls for phylogenetic analyses of the association between flashy displays and sex determination. Finally, our model predicts that sex-linked polymorphisms maintained by sexually antagonistic selection should disappear faster when sexual selection is present and as long as the females can detect the polymorphism. Once female preferences have resolved sexually antagonistic selection and become established within a species, they could cause the further evolution of flashy male displays at loci throughout the genome (11).

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#### Supporting Online Material

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Materials and Methods

Tables S1 and S2

References

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## *Arabidopsis* H<sup>+</sup>-PPase AVP1 Regulates Auxin-Mediated Organ Development

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The transport of auxin controls developmental events in plants. Here, we report that in addition to maintaining vacuolar pH, the H<sup>+</sup>-pyrophosphatase, AVP1, controls auxin transport and consequently auxin-dependent development. AVP1 overexpression results in increased cell division at the onset of organ formation, hyperplasia, and increased auxin transport. In contrast, *avp1-1* null mutants have severely disrupted root and shoot development and reduced auxin transport. Changes in the expression of AVP1 affect the distribution and abundance of the P-adenosine triphosphatase and Pinformed 1 auxin efflux facilitator, two proteins implicated in auxin distribution. Thus, AVP1 facilitates the auxin fluxes that regulate organogenesis.

The phytohormone auxin [principally indole acetic acid (IAA)] plays a fundamental role in the formation of all plant organs, and gradients of auxin have been shown to be essential to polarity of development (1, 2). Because IAA is a weak acid (pK<sub>a</sub> 4.75, where K<sub>a</sub> is the acid dissociation constant), a chemiosmotic model describes polar auxin uptake and efflux driven by a plasma membrane H<sup>+</sup> gradient (3). In the acidic apoplast, an enrichment of the lipophilic protonated species of IAA facilitates its entry into the cell. The same gradient motivates efflux of anionic (non-lipid soluble) IAA<sup>-</sup>

retained in the neutral cytoplasm by means of polarly localized efflux complexes (4). Thus, the transporters responsible for setting cytoplasmic and apoplastic pH are likely to have key roles in driving this polar flux of auxin.

Plants have three distinct membrane H<sup>+</sup>-pumps capable of generating pH gradients (5). The P-type H<sup>+</sup>-adenosine triphosphatase (P-ATPase) is a single-subunit protein that energizes transport across the plasma membrane (PM) by extruding H<sup>+</sup> from the cell (6). The vacuolar H<sup>+</sup>-ATPase (V-ATPase) complex, encoded by at least 26 genes, acidifies