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SEXUAL SELECTION AND SEX LINKAGE

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Abstract.—Some animal groups, such as birds, seem prone to extreme forms of sexual selection. One contributing factor may be sex linkage of genes affecting male displays and female preferences. Here we show that sex linkage can have substantial effects on the genetic correlation between these traits and consequently for Fisher's runaway and the good-genes mechanisms of sexual selection. Under some kinds of sex linkage (e.g. Z-linked preferences), a runaway is more likely than under autosomal inheritance, while under others (e.g., X-linked preferences and autosomal displays), the good-genes mechanism is particularly powerful. These theoretical results suggest empirical tests based on the comparative method.

Key words.—Good genes, runaway process, sex linkage, sexual selection.

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Why do some groups of animals, such as the birds of paradise and cichlid fishes, seem particularly prone to extreme sexual selection? Several factors have been proposed, including the form of parental care (Snow 1962), diet (Snow 1962), neuroanatomy (Ryan 1986), and sex linkage (Hastings 1994). A role for the sex linkage of genes affecting mating preferences and male displays is suggested by the casual observation that birds and butterflies, which have Z-W sex chromosomes, often seem to have more conspicuous male secondary sexual traits than groups with X-Y sex determination, such as mammals and flies (Hastings 1994).

How sex linkage affects sexual selection depends on the evolutionary mechanism that causes exaggeration of male displays and female preferences. One possibility is indirect selection, in which a mating preference becomes exaggerated as the result of a genetic correlation that it naturally develops with the male display (reviewed in Kirkpatrick and Ryan 1991). Under Fisher's (1930) runaway process, the display is initially at an evolutionary equilibrium that balances selection favoring increased survival against selection for increased mating success. The genetic correlation does not affect the location of this equilibrium. If the genetic correlation exceeds a critical value, however, the equilibrium becomes unstable (Lande 1981). Selection on the display in males exaggerates the preference through the correlation so rapidly that the display cannot catch up with the new regime of sexual selection. Consequently, the preference and display evolve at an explosive rate and in an unpredictable direction.

A second form of indirect selection is the good-genes process. It operates when a male display is genetically correlated with other traits that are under directional selection (the good genes). For example, a male's ability to display vigorously to females may be related to his ability to fight off pathogens (Hamilton and Zuk 1982). As preference genes become genetically associated with male display genes, they will also become correlated with the good genes. Consequently, more extreme preferences are indirectly selected as the good genes spread.

Thus, Fisher's runaway and the good-genes processes depend critically on a genetic correlation between the preference and other genes that are directly selected, either those for the display (in the runaway) or those for other traits that enhance survival (with good genes). This correlation has been calculated under the assumption that the preference and display are autosomally inherited (Lande 1981; Kirkpatrick 1982; Kirkpatrick and Barton 1997; Kirkpatrick et al. 2002), but general results for sex-linked genes have not yet been developed.

What evidence is there that preference and display genes might be sex linked? Remarkably, recent data suggest it may be quite common. Reinhold (1998) reviewed data on X-linkage of genes affecting sexually selected traits in 13 genera of insects and mammals and concluded that they typically contribute as much as one-third of all phenotypic variation. Prowell's (1998) review of data from the lepidoptera found that 60% of traits with sex-limited expression are Z-linked. On the other hand, Ritchie and Phillips (1998) did not find overrepresentation of sex-linked genes for traits involved in

premating isolation of insects other than the Lepidoptera and perhaps the Orthoptera. Y-linked genes affecting sexually dimorphic traits have been found in a variety of animals, including humans, and are particularly common in the bright and highly dimorphic poeciliid fishes (guppies and their relatives; Lindholm and Breden 2002). In birds, which have ZZ males and ZW females, there are Z-linked genes that control male plumage characters that are important to species recognition (Saetre et al. 2002; G.A. Parker, pers. comm.). While data on preferences are less numerous, they also suggest sex linkage is common. Most variation in a female mating preference in *Colias* butterflies (Grula and Taylor 1980) and *Utetheisa* moths (Iyengar et al. 2002) is carried on the Z chromosome. This is particularly striking in the case of *Colias*, where the Z comprises only 1.6% of a female's genome. X-linked preferences are found in flies (Heisler 1984). In groups like the Hymenoptera that have haploid males and diploid females, the entire genome—and hence all preference and display genes—behave as if X-linked.

Motivated by these data, we determine here how sex linkage affects the evolution of female mating preferences by indirect selection. We also consider maternal transmission, which occurs with cytoplasmic inheritance and some forms of cultural inheritance. The paper begins by extending earlier theory for autosomal genes to calculate the genetic correlation between a preference and display under all possible forms of sex linkage. Those results are then used to determine the implications for Fisher's runaway and the good-genes processes. We find that sex linkage can have a substantial effect on the outcome of sexual selection.

The Genetic Correlation between Display and Preference

In this section we calculate the additive genetic correlation between a female preference and male display trait that naturally develops as the result of nonrandom mating (Fisher 1930). We developed a genetic model that allows for any mode of inheritance of the preference and display. Here we consider autosomal (A), X-linked, Y-linked, Z-linked, W-linked inheritance, and maternal (M) modes.

Our model is based on a quasi-linkage equilibrium (QLE) approximation whose genetic assumptions are quite general (Kirkpatrick and Barton 1997). Variation in the preference and display can be contributed by any number of loci with any distribution of genetic effects. The loci can be linked or not; at equilibrium, the genetic correlation between a preference and a display is unaffected by the recombination rates (Lande 1981; Kirkpatrick 1982; Kirkpatrick et al. 2002). A key parameter in the model is ρ, the phenotypic correlation between the female's preference and male's display among mated pairs. The genetic correlation between preference and display can be calculated from ρ, and the results apply regardless of the ecological and behavioral details of how mates are chosen (Kirkpatrick and Barton 1997).

Their are four main restrictions of our model. First, we assume all genes have additive effects, that is, dominance and epistasis are negligible. (Although a QLE approximation can be derived when there is nonadditive gene action, the results are complicated and difficult to interpret.) Second, we assume that ρ is not too large (less than 0.4). This assumption allows us to find expressions that depend in a simple linear way on ρ. Third, we assume that the strength of selection acting on individual genes is weak. Fourth, we assume that allele frequencies are changing slowly, if at all. This assumption will be met when the population is at a stable equilibrium, in the initial stage of an unstable runaway, or when the effects of a good-genes process on individual preference loci are weak. The third and fourth assumptions imply that the linkage disequilibria evolve more rapidly than allele frequencies and have values that are much smaller than their maximum possible values. The QLE approximation uses those consequences to find simple expressions for the disequilibria and the genetic correlations they produce.

To make the effects of sex linkage clear, we assume that all genes affecting the preference have the same mode of inheritance (e.g., X-linkage), and likewise for the male display (e.g., autosomal). Kirkpatrick et al. (2002) present detailed calculations for the genetic correlation between the preference and display in two cases, when both traits are autosomal and when the preference is X-linked and the display is autosomal. Following the same analytic method, we calculated the genetic correlation for preference and trait under 18 additional cases. Although the calculations are tedious and rather involved, it was possible to largely automate them using Mathematica (Wolfram 1999). Appendix 1 shows the calculations for one case, and a Mathematica notebook showing details for all 20 cases is provided in the Supplementary Materials, available at: http://dx.doi.org/10.1554/03-332.1.s1 and http://dx.doi.org/10.1554/03-332.1.s2.

The concept of the preference-display correlation may be unclear because both traits typically have sex-limited expression and so are not expressed within an individual. In these cases, we can adopt any definition for an individual's value of the unexpressed trait that is the equivalent of additive gene action. For example, it is convenient to think of the value for the male display trait in a female as the number of alleles she carries that would increase the trait's value if carried in a male. Appendix 1 shows that the following results are insensitive to the specific definition used.

Table 1 shows the results for r_m, the genetic correlation in males, and r_f, the genetic correlation in females. The mode of inheritance can have substantial effects, causing the correlations to vary from 0.4 to more than twice the value generated under autosomal inheritance. The largest values occur when the display is Y linked or when the preference is W linked or maternally inherited. The explanation is simple: with Y-linkage of the display, for example, the sons of a chosen male inherit their father's display genotype intact, undiluted by the mixing with the mother's genes that occurs with autosomal inheritance. At the other extreme, maternal inheritance of the display prevents establishment of a genetic correlation because offspring inherit none of their father's display genes.

The results shown in Table 1 are given in terms of expressions that depend on the genetic variances for the male trait and female mating preference (see the table caption). Given values for those variances, either from data or a model, we can calculate the genetic correlations that are expected to develop. Although data and theory suggest genetic variances

TABLE 1. Genetic correlations and propensities for the runaway and good-genes processes under different modes of inheritance. The first two columns indicate the mode of inheritance for the preference and display. The third and fourth columns give the relative sizes of the genetic correlations in males and females produced by sexual selection. The correlations are obtained by multiplying each entry by $h_P\ h_T\ \rho/2$, where h_P is the square root of the heritability of the preference in females, h_T is the square root of the heritability of the display in males, and ρ is the phenotypic correlation between the preference in females and the display in males among mated pairs. The fifth column gives a relative measure of the critical size of ρ needed to initiate a runaway process. The critical value is found by multiplying the entry by $2\sqrt{2G_T^m}k_T/(k_{PT}\sqrt{G_P^f})$, where G_T^m and G_P^f are the additive genetic variances for the display in males and the preference in females, k_T is the strength of stabilizing natural selection on the display, and k_{PT} is the rate at which the directional selection gradient on the display increases as a function of the mean preference (see Hall et al. 2000). The last column gives the relative (per generation) rate of preference evolution by the good-genes mechanism, measured in phenotypic standard deviations of the preference. Actual rates are found by multiplying the entry by $\rho r_{TW}^m h_P^2 h_T\sqrt{G_w}/2$, where r_{TW}^m is the genetic correlation between the display and lifetime fitness in males, and G_w is the additive genetic variance for relative fitness. ''NP'' means a runaway is not possible, bold entries show cases more favorable to exaggeration of preferences than when the display and preference are autosomal, and dashes indicate modes of inheritance that do not exist. ''Undet'' indicates that the value is undetermined by the model: when there is no recombination between the display and preference genes, the correlation between them is determined by forces other than sexual selection.

Preference	Display	r_m	r_f	ρ^*	$\Delta\bar{P}$
A	A	1	1	1	1
	X	0.4	$\sqrt{32/25} \approx 1.13$	$5/3 \approx 1.67$	$(1 + \sqrt{8})/5 \approx 0.766$
	Y	2	—	1	1
	Z	1.2	$\sqrt{32/25} \approx 1.13$	$10/9 \approx 1.11$	$\mathbf{(3 + \sqrt{8})/5 \approx 1.17}$
	M	0	0	NP	0
X	A	$\sqrt{32/25} \approx 1.13$	1.2	1.875	$\mathbf{4/3 \approx 1.33}$
	X	$1/\sqrt{2} \approx 0.707$	$\sqrt{2} \approx 1.41$	2	$\mathbf{(1 + \sqrt{8})/3 \approx 1.28}$
	Y	$\sqrt{2} \approx 1.41$	—	3	$2/3 \approx 0.667$
	M	0	0	NP	0
Z	A	$\sqrt{32/25} \approx 1.13$	0.4	$\mathbf{15/32 \approx 0.469}$	$2/3 \approx 0.667$
	Z	$\sqrt{2} \approx 1.41$	$1/\sqrt{2} \approx 0.707$	**0.5**	$(4 + \sqrt{2})/6 \approx 0.902$
	M	0	0	NP	0
W	A	—	2	NP	**2**
	Z	—	$\sqrt{2} \approx 1.41$	NP	$\mathbf{\sqrt{2} \approx 1.41}$
	M	—	Undet	NP	Undet
M	A	2	2	NP	**2**
	X	2	$\sqrt{8} \approx 2.83$	NP	$\mathbf{2\sqrt{2} \approx 2.83}$
	Y	2	—	NP	0
	Z	2	$\sqrt{2} \approx 1.41$	NP	$\mathbf{\sqrt{2} \approx 1.41}$
	M	Undet	Undet	Undet	Undet

can be affected by sex linkage (Charlesworth et al. 1987), currently there are no robust generalizations about those effects because they are numerous and can interact in complex ways. To make further progress, we will therefore make the simplest assumption, that genetic variances do not change systematically with the mode of inheritance. The verbal conclusions below are therefore tentative and can be revisited if new data or theory about the relation between modes of inheritance and levels of genetic variation becomes available. The expression shown in Table 1 are valid in any event if appropriate values for the genetic variances are used.

THE RUNAWAY PROCESS

Although the runaway process depends critically on the genetic correlation in males, the size of this correlation by itself is a poor indicator of the propensity for a runaway process. That is because the runaway is also affected by other factors, such as the relative numbers of genes for display and preference carried by males and females.

To determine the conditions that lead to a runaway, we assumed the male display is under stabilizing natural selection but that the female mating preference has no direct effects on fitness. The calculations follow the method of Hall et al. (2000), which we applied here to the 20 combinations of modes of inheritance for the display and preference shown in Table 1. It was again possible to automate these calculations using Mathematica (Wolfram 1999). The details for each case are given in the Supplementary Materials (available online), which also include calculations for the more general case in which there is natural selection on the preference as well as the male display. The conditions leading to a runaway involve (among other things) the additive genetic correlation r_m between the preference and the display in males. That correlation can, in turn, be expressed in terms of the phenotypic correlation ρ between mated pairs using the results from the last section.

A convenient measure for the propensity of a runaway is ρ^*, which is defined as the minimum value of the phenotypic correlation between the display in males and the preference in females among mated pairs that will cause the genetic correlation to become large enough to trigger a runaway. Thus, larger values of ρ^* imply that runaway is more difficult.

Results are shown in Table 1. Sex linkage can have a substantial effect. Z-linkage of preference genes is particularly conducive to a runaway. With Z-linked preferences and a Z-linked or autosomal display, for example, the phenotypic correlation between mated pairs needed to trigger a runaway is only about half as large as that needed when the preference is autosomal. The explanation lies in the mixing of preference genes inherited from males and females. Whenever selection acts only on males, evolutionary change is retarded each generation by the dilution of genes from successful males

with genes from females, which are not under selection. With an autosomal locus, half of the copies of a gene are inherited from females. With Z-linkage, however, only one-third of the genes are inherited from females, so the dilution effect is weaker and evolution is more rapid. The runaway is triggered when selection for extreme displays in males also selects indirectly for the extreme preference genes that those males tend to carry. Evolutionary exaggeration of the preference is more rapid with Z-linkage because a greater fraction of the preference genes in each generation are inherited from the selected males. As a result, runaway is easier to achieve.

Sex linkage of the display also influences the runaway. When Z-linked, the display evolves more rapidly as a result of the dilution effect. The display is therefore more able to catch up with the changing preference, making runaway less likely than when the display is autosomally inherited. (The contrast between Z-linked and autosomal inheritance, however, is smaller for the display than for the preference.) When the display is maternally inherited, runaway is impossible altogether, despite a high genetic correlation between preference and display. The explanation here is that directional selection on males cannot cause exaggeration of the preference because extreme males favored by selection cannot transmit extreme preference genes to their offspring.

The Good-Genes Process

We determined the impact of modes of inheritance on the good-genes process using the quasi-linkage equilibrium approach outlined above. Our measure for the efficacy of good genes is $\Delta\bar{P}$, defined as the per generation change in the average strength of the female preference, measured in units of preference phenotypic standard deviations. Again we assume that the preference does not experience direct selection. If it does, then our measure $\Delta\bar{P}$ is proportional to how costly a preference will evolve as the result of indirect selection from good genes. The calculations are a direct extension of the calculations developed above and in Kirkpatrick and Barton (1997); details are given in Appendix 2.

The standardized rate of change in a mean female preference evolving under the good-genes process is

$$\Delta\bar{P} = h_P[Fr^f_{PW}\sqrt{G^f_W} + \sqrt{F(1-F)}r^m_{PW}\sqrt{G^m_W}], \quad (1)$$

where h_P is the square root of the heritability of the preference, F is the fraction of preference genes carried by females (1/2 for autosomal, 2/3 for X linked, 1/3 for Z linked, 1 for W linked and maternally inherited), G^f_W is the additive genetic variances for fitness in females, and r^f_{PW} is the genetic correlation between the preference and fitness in females. Parameters with a superscript m are the corresponding quantities in males. The genetic variances G^f_W and G^f_W refer to lifetime fitness including the effects of both natural and sexual selection.

This result is quite general. The first of the two terms that appear inside the parentheses of equation (1) represents the contribution from selection on females, and the second from selection on males. The fraction of preference genes carried by females, F, contributes to these terms in an intuitively reasonable way. If $F = 0$, then the preference cannot evolve because females carry no genes for it, and both terms in (1) vanish. At the other extreme, if $F = 1$ then selection for good genes carried by males has no effect on the preference because they carry no preference genes, and consequently the second term in (1) vanishes.

We can make further progress in describing the good-genes process in terms of measurable quantities. The genetic correlations between the preference and lifetime fitness, r^m_{PW} and r^f_{PW}, can be written in terms of ρ, the phenotypic correlation between the female preference and the male display in mated pairs. Standard regression theory shows that $r^m_{PW} = r^m_{TW}r_m$, where r^m_{TW} is the genetic correlation between the display and lifetime fitness in males, and r_m is again the genetic correlation in males between the display and preference (given in Table 1). An analogous argument gives $r^f_{PW} = r^m_{TW}r^{mf}_W r_f$, where r^{mf}_W is the correlation between males and females in the additive genetic component of lifetime fitness and r_f is again the genetic correlation between the display and preference in females.

When these quantities are substituted into equation (1), we get a result that is quite general for the impact of good genes:

$$\Delta\bar{P} = h_P\rho r^m_{TW}[Fr_f r^{mf}_W\sqrt{G^f_W} + \sqrt{F(1-F)}r_m\sqrt{G^m_W}]. \quad (2)$$

To simplify the following discussion, we will assume that the additive genetic variances in fitness are equal in males and females ($G^f_W = G^m_W$) and that the genetic correlation between fitness in males and females is perfect ($r^{mf}_W = 1$). Results from laboratory populations of *Drosophila* (Chippindale et al. 2001) show these last two assumptions are not general, but there is not yet sufficient data to draw robust conclusions about what parameter values might be typical. Equation (2) is valid in any event.

Table 1 shows the rate of preference evolution under good genes. Sex linkage has a large effect: all else equal, a preference can evolve from 0.67 to 2.8 times faster than it does when both display and preference are autosomal. Situations particularly favorable to good genes are those where the preference is autosomal and the display is Z linked, the preference is X linked and the display genes are either autosomal or X linked, and the preference is W linked or maternally inherited. The outcomes result from interactions between how the mode of inheritance affects the display-preference genetic correlations and the fraction of preference genes carried by females (reflected by r_f, r_m, and F in eq. 2).

Discussion

Female mating preferences for extreme male displays can be established in many ways, including pleiotropic effects of preference genes, mutation, and random genetic drift (Kirkpatrick and Ryan 1991). In the absence of a runaway or good genes, the equilibrium for the display does not depend on the size of the genetic correlation between it and the preference.

This correlation comes into play, however, when the preference evolves by indirect selection, and sex linkage can have large effects on its size (Table 1). With an autosomal preference and Y-linked display, for example, the correlation in females is twice the value expected when both preference and display are autosomal. At the other extreme, with an

autosomal preference and maternally transmitted display, the display-preference correlation is zero. Furthermore, the correlations can be very different in males and females: with an autosomal preference and X-linked display, for example, the correlation is almost three times larger in females than males.

Sex linkage also has a strong impact on the outcome of sexual selection under indirect selection (Table 1). The runaway process is enhanced under some kinds of sex linkage (e.g., with a Z-linked preference), while good genes is accelerated by others (e.g., an X-linked preference and an autosomal display). These comparative conclusions are tentative, however, because they depend on the assumption that other factors, such as levels of genetic variation, do not depend in a systematic way on the mode of inheritance. A conservative conclusion is that mode of inheritance can be expected to have an effect on indirect selection.

One strong conclusion is that maternal inheritance or W-linkage of either the preference or display makes a runaway impossible (Table 1). This implies that a runaway can be ruled out when females transmit the preference to their daughters culturally, for example, by some form of behavioral imprinting.

For both the runaway and good-genes process, Y-linkage of the display is not conducive to the evolution of extreme mating preferences. Y chromosomes are largely inert in groups such as mammals and flies, but they carry many of the genes for male color in poeciliid fishes (Lindholm and Breden 2002). This suggests that something other than the runaway or good-genes processes are responsible for extreme displays and preferences in guppies and their relatives, or that this group has unusual values for the other parameters appearing in Table 1. This last possibility could, in principle, be tested as all of the parameters are empirically measurable.

Two previous papers suggested that sex linkage could influence the outcome of sexual selection. Hastings (1994) used a heuristic mathematical argument to suggest that a W-linked preference would be particularly prone to exaggeration via good genes. Our analysis corroborates that conclusion. It seems, however, that the W chromosome in birds is almost devoid of genes (Fridolfsson et al. 1998), so it is not clear how important this finding is.

More recently, Reeve and Pfennig (2003) and their coworkers (Iyengar et al. 2002) suggested that sex linkage could play an important role in sexual selection. They used simulation results to argue that Z-linkage of a preference and trait increases the potential for their exaggeration by sexual selection and X-linkage decreases the potential, relative to the autosomal case (Reeve and Pfennig 2003). Rather than assuming there is standing genetic variation for a male display, as we have here, they considered evolution of the display when the availability of new, selectively favored mutations is limiting. Their simulations show that sex linkage influences the likelihood that a new display mutation will escape loss when rare.

Reeve and Pfennig argued verbally that these results can be explained by the effects of sex linkage on the genetic correlation between the preference and display. As a favorable display mutation spreads, the correlation causes the preference for that mutation to increase also. Reeve and Pfennig (2003) suggested that some kinds of sex linkage increase the genetic correlation, causing the preference for the display mutation to increase faster and so making it less likely that the mutation is lost by chance when it is still rare. In fact, the probability that a display mutant is lost in a large population is unaffected by this correlation. When it is rare, the display mutation can generate only negligible indirect selection on the preference. That is because its statistical associations with other alleles must be small: the maximum possible value of the linkage disequilibrium of a rare allele is equal to its frequency. By the time that the frequency of the display mutation is large enough to affect the preference, the mutation is no longer at risk of being lost. Thus, the survival or loss of mutation in a large population is governed only by its direct effects on fitness.

What then explains Reeve and Pfennig's (2003) simulation results? They implicitly assume that new male display mutations are completely dominant with respect to how they are perceived by females. When this dominance effect is accounted for, their simulation results show exactly the pattern predicted by the classic theory for the fixation probability of a rare mutation that is sex-linked and under direct positive selection (Charlesworth et al. 1987). Contrary to their conclusions, however, this probability does not translate directly into differences in evolutionary rates for male displays under different modes of inheritance. That is because the mode of inheritance also affects the number of genes available to mutate. For example, there are only three-fourths as many X-linked genes as autosomal genes. After accounting for these two effects of sex linkage, if fitness effects are additive and if males and females have equal mutation rates, then evolutionary rates for male displays are unaffected by the mode of inheritance (Charlesworth et al. 1987). In short, Reeve and Pfennig's (2003) results are not caused by the effects of sex linkage, but rather their implicit assumptions about the dominance of new mutations and failure to account for differences in the rate at which new mutations appear under different modes of inheritance.

While we have focused on indirect selection, extreme mating preferences can be established in other ways that do not involve genetic correlations with a male display. Direct selection acting on sensory systems can establish perceptual biases that result in a female mating preference as a pleiotropic side effect (Kirkpatrick 1987; Kirkpatrick and Ryan 1991). If there is standing genetic variation for the display that the preference acts on, then the display will typically evolve to an equilibrium that balances the forces of natural and sexual selection. In that event, the mode of inheritance will not affect the equilibrium, regardless of how preference and trait genes are inherited.

On the other hand, if evolution of a male display is limited by the availability of favored mutations, then sex-linkage can influence its rate of evolution in two ways. First, if the mutations are either partly dominant or recessive, then the mode of inheritance for the display affects the substitution rate (Charlesworth et al. 1987). Current evidence suggests, however, that selectively favored substitutions may often involve mutations with additive fitness effects (Betancourt et al. 2002). Second, sex linkage has an effect when mutation rates in males and females are not equal. Recent data from mammals and birds suggest that mutation rates in males can be

2.0 to 8.5 times higher in males than in females (Hurst and Ellegren 1998). Male-biased mutation rates will cause Z-linked genes to evolve faster than autosomal genes, which in turn evolve faster than X-linked genes (Kirkpatrick and Hall 2004). These differences can be substantial; for example, Z-linked display genes will evolve 60% faster than X-linked genes when mutation rates are five times higher in males than in females.

Whether the outcome of sexual selection is influenced by these last two effects depends on whether displays are constrained by the availability of favorable mutations. Mutations do appear to be limiting in some cases, for example, in species where displays have not evolved despite preexisting female preferences for them (Ryan 1998). In other cases, however, displays show abundant standing genetic variation (Pomiankowski and Møller 1995).

The importance of the runaway and good-genes processes has long been a contentious and refractory question in behavioral ecology. A new opportunity to assess different mechanisms of preference evolution is provided by the diversity of ways that male displays and female mating preferences are inherited. Together with the theoretical results presented here, it may ultimately be possible to exploit this diversity using the comparative method (Reeve and Pfennig 2003). Because the runaway and good-genes processes are favored by different modes of inheritance, we might hope to test their relative contributions to mating preference evolution. Regardless of whether one of these or some other mechanism is at work, it would be most valuable to determine if the mode of inheritance is a factor that contributes to evolution of the remarkable sexual displays found in some animal taxa.

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Appendix 1

This appendix shows how the genetic correlation between a female preference and a male display are calculated for arbitrary modes of inheritance. We sketch the general approach, then give details for the case in which the preference is Z linked and the display is autosomal. The calculations follow the methodology developed in Kirkpatrick et al. (2002), who showed the calculations for the cases where the preference and display are both autosomally inherited and for that where the preference is X linked and the display is autosomal (p. 1741). The calculations for all the cases

shown in Table 1 are presented as a Mathematica notebook in the Supplemental Materials, available online at: http://dx.doi.org/10.1554/03-332.1.s1 and at http://dx.doi.org/10.1554/03-332.1.s2. Readers who wish further details are referred to the Supplemental Materials and Kirkpatrick et al. (2002).

General Results

The value of the genetic correlation between a preference and a display can be different in males and females because they can carry different number of genes for those traits (when one or both are sex linked) and because these genes may be expressed differently in the sexes. In many cases, the preference is not expressed at all in males, nor the display in females. In such cases we can still determine a genetic correlation between these traits by adopting an arbitrary definition for their expression. In the following we will assume that two alleles segregate at each locus, which we refer to as allele 0 and allele 1. A convenient option then is to define the preference phenotype for a male to be the number of 1 alleles he carries for the preference (even if the preference is not actually expressed). Likewise, we can define the display phenotype for a female to be the number of 1 alleles she carries for the display. We will see shortly that we can calculate general expressions for the genetic correlation that are insensitive to the definitions that are used.

We assume that genetic variation in the preference and display is caused by genes with additive effects. The preference phenotype for a female can then be written

$$P = \bar{P} + \sum_{\mathbf{i}\in\mathbb{P}_f} b_{\mathbf{i}}^{P,f}\zeta_{\mathbf{i}} + e_P, \tag{A1}$$

where $\bar{P}$ is the mean preference in the population, the summation is over the set $\mathbb{P}_f$ of all genes carried by females that affect the preference, $b_{\mathbf{i}}^{P,f}$ is the difference in a female's preference caused by carrying allele 1 rather than allele 0 at position $\mathbf{i}$, $\zeta_{\mathbf{i}}$ is an indicator variable that takes the value $1 - p_{\mathbf{i}}$ if the female carries allele 1 at position $\mathbf{i}$ and is $-p_{\mathbf{i}}$ otherwise, and e_P is a random environmental contribution to the female's preference phenotype. A position is a genetic location defined by the locus and parent from whom the gene was inherited; thus, a diploid autosomal locus has two positions in an individual, an X-linked locus has two positions in a female but only one in a male, etc. (Kirkpatrick et al. 2002). Similarly to (A1), the display trait phenotype for a male can be written

$$T = \bar{T} + \sum_{\mathbf{i}\in\mathbb{T}_m} b_{\mathbf{i}}^{T,m}\zeta_{\mathbf{i}} + e_T, \tag{A2}$$

where $\mathbb{T}_m$ is the set of all positions in males that affect the display trait.

From the basic definition of a correlation, the genetic correlations between the preference and display are

$$r_{PT}^f = G_{PT}^f/\sqrt{G_P^f G_T^f} \quad \text{and} \tag{A3a}$$

$$r_{PT}^m = G_{PT}^m/\sqrt{G_P^m G_T^m} \tag{A3b}$$

in males and females, respectively. In the denominator of (A3a) are additive genetic variances for the preference and display in females, respectively, and in the denominator of (A3b) are the corresponding variances in males. Under our assumption of additive gene action, when both traits are expressed in females, these variances are given by

$$G_P^f \approx \sum_{\mathbf{i}\in\mathbb{P}_f} (b_{\mathbf{i}}^{P,f})^2 pq_{\mathbf{i}} \quad \text{and} \tag{A4a}$$

$$G_T^f \approx \sum_{\mathbf{i}\in\mathbb{T}_f} (b_{\mathbf{i}}^{T,f})^2 pq_{\mathbf{i}}, \tag{A4b}$$

where the summations are over the sets $\mathbb{P}_f$ and $\mathbb{T}_f$ of all genes carried by females that affect the preference and display, respectively, and $pq_{\mathbf{i}} = p_{\mathbf{i}}(1 - p_{\mathbf{i}})$, where $p_{\mathbf{i}}$ is the frequency of allele 1 at locus i. These expressions are approximate because we are neglecting the contribution from associations (linkage disequilibria) between loci. Under the quasi-linkage equilibrium conditions described below, however, those contributions are small relative to the terms that have been retained. As discussed above, when the display is not expressed in females, we can adopt an arbitrary definition for the $b_{\mathbf{i}}^{T,f}$, for example $b_{\mathbf{i}}^{T,f} = 1$. The corresponding genetic variances when the preference and display are expressed in males are given by

$$G_P^m \approx \sum_{\mathbf{i}\in\mathbb{P}_m} (b_{\mathbf{i}}^{P,m})^2 pq_{\mathbf{i}} \quad \text{and} \tag{A5a}$$

$$G_T^m \approx \sum_{\mathbf{i}\in\mathbb{T}_m} (b_{\mathbf{i}}^{T,m})^2 pq_{\mathbf{i}}. \tag{A5b}$$

The numerators of equations (A3a,b) have the additive genetic covariances between the preference and display, G_{PT}^f and G_{PT}^m for females and males, respectively. When genetic variation for the preference and display is caused by different sets of genes (e.g., there is no pleiotropy between these two traits), these covariances are entirely caused by associations (linkage disequilibria) between alleles for the preference and those for the display:

$$G_{PT}^f = \sum_{\mathbf{i}\in\mathbb{P}_f}\sum_{\mathbf{j}\in\mathbb{T}_f} b_{\mathbf{i}}^{P,f} b_{\mathbf{i}}^{T,f}(D_{i_{ff}j_{ff}} + D_{i_{ff}j_{fm}} + D_{i_{fm}j_{ff}} + D_{i_{fm}j_{fm}}) \tag{A6}$$

and

$$G_{PT}^m = \sum_{\mathbf{i}\in\mathbb{P}_m}\sum_{\mathbf{j}\in\mathbb{T}_m} b_{\mathbf{i}}^{P,m} b_{\mathbf{i}}^{T,m}(D_{i_{mf}j_{mf}} + D_{i_{mf}j_{mm}} + D_{i_{mm}j_{mf}} + D_{i_{mm}j_{mm}}), \tag{A7}$$

where $D_{i_{ff}\,j_{fm}}$ is, for example, the association (disequilibrium) in females between the allele at locus i inherited from a female (the mother) and the allele at locus j inherited from a male (the father). See Kirkpatrick et al. (2002) for further explanation of the notation.

The main technical challenge is to calculate the D values that appear in equations (A6) and (A7). When the strength of selection acting on individual loci and sets of loci is relatively weak, the population rapidly attains a state of quasi-linkage equilibrium (QLE), at which the disequilibria or associations between alleles at different loci change slowly. At this point, the dynamics for the associations among preference and display genes are given by a set of recursion equations:

$$\begin{aligned} D'_{i_{ff}j_{ff}} &= t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{ff},j_{ff}\}} D_{i_{ff}j_{ff}} + t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{ff},j_{fm}\}} D_{i_{ff}j_{fm}} \\ &\quad + t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{fm},j_{ff}\}} D_{i_{fm}j_{ff}} \\ &\quad + t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{fm},j_{fm}\}} D_{i_{fm}j_{fm}} + O(a^2), \end{aligned} \tag{A8a}$$

$$\begin{aligned} D'_{i_{fm}j_{fm}} &= t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mf},j_{mf}\}} D_{i_{mf}j_{mf}} + t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mf},j_{mm}\}} D_{i_{mf}j_{mm}} \\ &\quad + t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mm},j_{mf}\}} D_{i_{mm}j_{mf}} \\ &\quad + t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mm},j_{mm}\}} D_{i_{mm}j_{mm}} + O(a^2), \end{aligned} \tag{A8b}$$

$$\begin{aligned} D'_{i_{mf}j_{mf}} &= t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{ff},j_{ff}\}} D_{i_{ff}j_{ff}} + t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{ff},j_{fm}\}} D_{i_{ff}j_{fm}} \\ &\quad + t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{fm},j_{ff}\}} D_{i_{fm}j_{ff}} \\ &\quad + t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{fm},j_{fm}\}} D_{i_{fm}j_{fm}} + O(a^2), \end{aligned} \tag{A8c}$$

$$\begin{aligned} D'_{i_{mm}j_{mm}} &= t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mf},j_{mf}\}} D_{i_{mf}j_{mf}} + t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mf},j_{mm}\}} D_{i_{mf}j_{mm}} \\ &\quad + t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mm},j_{mf}\}} D_{i_{mm}j_{mf}} \\ &\quad + t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mm},j_{mm}\}} D_{i_{mm}j_{mm}} + O(a^2), \end{aligned} \tag{A8d}$$

$$\begin{aligned} D'_{i_{ff}j_{fm}} &= t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{ff},j_{mf}\}} a_{i_f j_m} pq_{ij} + t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{ff},j_{mm}\}} a_{i_f j_m} pq_{ij} \\ &\quad + t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{fm},j_{mf}\}} a_{i_f j_m} pq_{ij} \\ &\quad + t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{fm},j_{mm}\}} a_{i_f j_m} pq_{ij} + O(a^2), \end{aligned} \tag{A8e}$$

$$D'_{i_{fm}j_{ff}} = O(a^2), \tag{A8f}$$

$$\begin{aligned} D'_{i_{mf}j_{mm}} &= t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{ff},j_{mf}\}} a_{i_f j_m} pq_{ij} + t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{ff},j_{mm}\}} a_{i_f j_m} pq_{ij} \\ &\quad + t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{fm},j_{mf}\}} a_{i_f j_m} pq_{ij} \\ &\quad + t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{fm},j_{mm}\}} a_{i_f j_m} pq_{ij} + O(a^2), \quad \text{and} \end{aligned} \tag{A8g}$$

$$D'_{i_{mm}j_{mf}} = O(a^2), \tag{A8h}$$

where primes denote the associations in zygotes in the next generation and $a_{i_f j_m}$ is the selection coefficient representing the force of sexual selection that unites in zygotes allele 1 at preference locus i in females with display trait locus j in males. Kirkpatrick et al. (2002) found that regardless of the mode of inheritance, this selection coefficient is

$$a_{i_f j_m} = \rho \frac{b_i^{P,f} b_j^{T,m}}{\sigma_P \sigma_T}, \tag{A9}$$

where ρ is the phenotypic correlation between the preference of a female and the display trait of a male among mated pairs, σ_P is the phenotypic standard deviation of the preference in females, and σ_T is the phenotypic standard deviation of the display trait in males.

The t values in equations (A8a–h) are transmission coefficients (see Kirkpatrick et al. 2002). For example, $t_{\{i_{mm},j_{mf}\}\leftarrow\{i_{mf},j_{ff}\}}$ is the joint probability that the allele at preference locus i in a male that was inherited from another male (his father) was in turn inherited from his father's mother, and that the allele at display locus j that male inherited from a female (his mother) was in turn inherited from his mother's mother. The transmission coefficients depend on the mode of inheritance for the preference and male display. When both characters are autosomal, for example, $t_{\{i_{mm},j_{mf}\}\leftarrow\{i_{mf},j_{ff}\}} = 1/4$.

We now have everything needed to calculate the values for the associations. At QLE, the associations in the next generation are approximately equal to those in the current generation: $D'_{\mathbf{ij}} = D_{\mathbf{ij}} + O(a^2)$. By setting these two values equal to each other, the linear system of equations (A8a–h) can be solved for the QLE values of these associations using, for example, Mathematica (Wolfram 1999). The following section shows how this is done for the case in which the preference genes are Z linked and the display trait genes are autosomal. The Supplemental Materials (available online) give the analogous calculations for all other cases shown in Table 1.

Z-linked Preferences, Autosomal Display Trait

When the preference is Z linked and the display trait is autosomal, the transmission coefficients in equations (A8a–h) for genes inherited by females are:

$$\begin{aligned}
t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{ff},j_{ff}\}} &= 0, & t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{ff},j_{fm}\}} &= 0,\\
t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{fm},j_{ff}\}} &= 0, & t_{\{i_{ff},j_{ff}\}\leftarrow\{i_{fm},j_{fm}\}} &= 0,\\
t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{ff},j_{mf}\}} &= 0, & t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{ff},j_{mm}\}} &= 0,\\
t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{fm},j_{mf}\}} &= 0, & t_{\{i_{ff},j_{fm}\}\leftarrow\{i_{fm},j_{mm}\}} &= 0,\\
t_{\{i_{fm},j_{ff}\}\leftarrow\{i_{mf},j_{ff}\}} &= 1/4, & t_{\{i_{fm},j_{ff}\}\leftarrow\{i_{mf},j_{fm}\}} &= 1/4,\\
t_{\{i_{fm},j_{ff}\}\leftarrow\{i_{mm},j_{ff}\}} &= 1/4, & t_{\{i_{fm},j_{ff}\}\leftarrow\{i_{mm},j_{fm}\}} &= 1/4,\\
t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mf},j_{mf}\}} &= 1/4, & t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mf},j_{mm}\}} &= 1/4,\\
t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mm},j_{mf}\}} &= 1/4, & t_{\{i_{fm},j_{fm}\}\leftarrow\{i_{mm},j_{mm}\}} &= 1/4.
\end{aligned}$$

The corresponding transmission coefficients for genes inherited by males are:

$$\begin{aligned}
t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{ff},j_{ff}\}} &= 0, & t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{ff},j_{fm}\}} &= 0,\\
t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{fm},j_{ff}\}} &= 1/2, & t_{\{i_{mf},j_{mf}\}\leftarrow\{i_{fm},j_{fm}\}} &= 1/2,\\
t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{ff},j_{mf}\}} &= 0, & t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{ff},j_{mm}\}} &= 0,\\
t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{fm},j_{mf}\}} &= 1/2, & t_{\{i_{mf},j_{mm}\}\leftarrow\{i_{fm},j_{mm}\}} &= 1/2,\\
t_{\{i_{mm},j_{mf}\}\leftarrow\{i_{mf},j_{ff}\}} &= 1/4, & t_{\{i_{mm},j_{mf}\}\leftarrow\{i_{mf},j_{fm}\}} &= 1/4,\\
t_{\{i_{mm},j_{mf}\}\leftarrow\{i_{mm},j_{ff}\}} &= 1/4, & t_{\{i_{mm},j_{mf}\}\leftarrow\{i_{mm},j_{fm}\}} &= 1/4,\\
t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mf},j_{mf}\}} &= 1/4, & t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mf},j_{mm}\}} &= 1/4,\\
t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mm},j_{mf}\}} &= 1/4, & t_{\{i_{mm},j_{mm}\}\leftarrow\{i_{mm},j_{mm}\}} &= 1/4.
\end{aligned}$$

Substituting those values into equations (A8a–h), setting $D'_{\mathbf{ij}} = D_{\mathbf{ij}} \equiv \tilde{D}_{\mathbf{ij}}$, and solving for the QLE value of the associations gives $\tilde{D}_{i_{ff}j_{ff}} = 0$, $\tilde{D}_{i_{fm}j_{fm}} = [(2b_P^f b_T^m)/(5\sigma_P\sigma_T)]\,pq_{ij}\rho$, $\tilde{D}_{i_{mf}j_{mf}} = [(b_P^f b_T^m)/(5\sigma_P\sigma_T)]\,pq_{ij}\rho$, $\tilde{D}_{i_{mm}j_{mm}} = [(2b_P^f b_T^m)/(5\sigma_P\sigma_T)]\,pq_{ij}\rho$, $\tilde{D}_{i_{ff}j_{fm}} = 0$, $\tilde{D}_{i_{fm}j_{ff}} = 0$, $\tilde{D}_{i_{mf}j_{mm}} = [(b_P^f b_T^m)/(\sigma_P\sigma_T)]\,pq_{ij}\rho$, $\tilde{D}_{i_{mm}j_{mf}} = 0$.

On substituting these results into equations (A6) and (A7), with the help of equations (A3–5), we find that the genetic correlation between the preference and display trait in female is

$$r_f = \left(\frac{2}{5}\right)\left(\frac{1}{2}h_P h_T \rho\right), \tag{A10}$$

and in males it is

$$r_m = \left(\sqrt{\frac{32}{25}}\right)\left(\frac{1}{2}h_P h_T \rho\right). \tag{A11}$$

These results are shown in Table 1, where the factor of $(\tfrac{1}{2}h_P h_T \rho)$ is suppressed from each entry in the interests of space (see the table caption). The analogous calculations for the other cases shown in Table 1 are given in the Supplemental Materials, available online. Note that the allelic effects (the b values) do not affect the correlations; thus, their definition when either the preference or display is not expressed has no impact on the final result.

Appendix 2

This appendix derives the general expressions for the per generation change in the mean of a female mating preference evolving under the good-genes process. The notation follows Appendix 1 and Kirkpatrick et al. (2002); please see that paper for details.

We assume that the genes that affect the preference have additive effects and that there are two alleles at each locus, which we denote 0 and 1. Thus, the preference phenotype of a particular female can be written

$$P = \sum_{\mathbf{i}\in\mathbb{P}_f} b_i X_{\mathbf{i}} + e_P, \tag{A12}$$

where $b_{\mathbf{i}}$ is the difference between the effects of alleles 1 and 0 at locus i, $X_{\mathbf{i}}$ is an indicator variable that takes the value one if the individual carries allele 1 at position $\mathbf{i}$ and is zero otherwise, and e_P is a random environmental effect. The sum is over the positions in set $\mathbb{P}_f$, which is the set of all positions that affect preferences in females.

We standardize the rate of change of the preference mean by dividing by the phenotypic standard deviation of the preference, σ_P. This standardized evolutionary rate is therefore

$$\Delta\bar{P} = \frac{1}{\sigma_P}\sum_{\mathbf{i}\in\mathbb{P}_f} b_{\mathbf{i}}\Delta p_{\mathbf{i}}, \tag{A13}$$

where $\Delta p_{\mathbf{i}}$ is the change in frequency of allele 1 at position $\mathbf{i}$. When the population reaches QLE, it can be shown that the change in allele frequencies is equal for all positions at a given locus. This rate is equal to the average change caused by selection within a generation at all the positions for that locus:

$$\begin{aligned}
\Delta p_i &= \frac{1}{n_i}\sum_{\mathbf{i}}\sum_{\mathbf{j}\in\mathbb{W}} a_{\mathbf{j}} D_{\mathbf{ij}}\\
&= \frac{1}{n_i}\left(\sum_{\mathbf{j}\in\mathbf{i}_f}\sum_{\mathbf{k}\in\mathbb{W}_f} a_{\mathbf{k}} D_{\mathbf{jk}} + \sum_{\mathbf{j}\in\mathbf{i}_m}\sum_{\mathbf{k}\in\mathbb{W}_m} a_{\mathbf{k}} D_{\mathbf{jk}}\right),
\end{aligned} \tag{A14}$$

where n_i is the total number of positions at locus i (e.g., four for autosomal diploid loci, three for X-linked loci, one for W-linked). The a values are the selection coefficients and the D values the associations of alleles at different positions as defined in Kirkpatrick et al. (2003). By $\mathbf{i}_f$ we mean the positions at locus i that are carried in females; $\mathbb{W}_f$ is the set of all positions in females that affect lifetime fitness; $\mathbf{i}_m$ and $\mathbb{W}_m$ are the corresponding quantities for males. Thus,

$$\begin{aligned}
\Delta\bar{P} &= \frac{1}{\sigma_P}\sum_{\mathbf{i}\in\mathbb{P}_f} b_{\mathbf{i}}\frac{1}{n_i}\left(\sum_{\mathbf{j}\in\mathbf{i}_f}\sum_{\mathbf{k}\in\mathbb{W}_f} a_{\mathbf{k}} D_{\mathbf{jk}} + \sum_{\mathbf{j}\in\mathbf{i}_m}\sum_{\mathbf{k}\in\mathbb{W}_m} a_{\mathbf{k}} D_{\mathbf{jk}}\right)\\
&= \frac{1}{\sigma_P}\sum_{\mathbf{i}\in\mathbb{P}_f}\frac{1}{n_i}\left(\sum_{\mathbf{j}\in\mathbf{i}_f}\sum_{\mathbf{k}\in\mathbb{W}_f} a_{\mathbf{k}} b_{\mathbf{j}} D_{\mathbf{jk}} + \sum_{\mathbf{j}\in\mathbf{i}_m}\sum_{\mathbf{k}\in\mathbb{W}_m} a_{\mathbf{k}} b_{\mathbf{j}} D_{\mathbf{jk}}\right).
\end{aligned} \tag{A15}$$

Assume that all preference loci have the same mode of inheritance, and write F for the fraction of all positions at a preference locus that are carried by a female (e.g., 1/2 for autosomal, 1 for W-linked and maternally-inherited, 2/3 for X-linked). Then,

$$\begin{aligned}
\Delta\bar{P} &= \frac{1}{\sigma_P}F\left(\sum_{\mathbf{i}\in\mathbb{P}_f}\sum_{\mathbf{j}\in\mathbb{W}_f} a_{\mathbf{j}} b_{\mathbf{i}} D_{\mathbf{ij}} + \sum_{\mathbf{i}\in\mathbb{P}_m}\sum_{\mathbf{j}\in\mathbb{W}_m} a_{\mathbf{j}} b_{\mathbf{i}} D_{\mathbf{ij}}\right)\\
&= \frac{1}{\sigma_P}F(G_{PW}^f + G_{PW}^m),
\end{aligned} \tag{A16}$$

where G^{f}_{PW} and G^{m}_{PW} are the genetic covariances between the preference and lifetime fitness in females and males, respectively. The covariance in males is defined to be the covariance that would be obtained if preference alleles in males were expressed as they are in females. This expression is valid even when fitness alleles are expressed and selected differently in males and females.

Rewriting the genetic covariances in terms of the corresponding genetic correlations gives

$$\Delta\bar{P} = \frac{1}{\sigma_P}F(\sqrt{G^{f}_{P}}\sqrt{G^{f}_{W}}r^{f}_{PW} + \sqrt{G^{m}_{P}}\sqrt{G^{m}_{W}}r^{m}_{PW}), \qquad \text{(A17)}$$

where G_P and G_W are the additive genetic variances for the preference and fitness (with superscripts indicating the sex). The genetic variance of the preference in males, G^{m}_{P}, is defined as what would be measured if preference alleles had the same individual phenotypic effect in males as females, and so $G^{m}_{P} = [(1 - F)/F]G^{f}_{P}$. Thus,

$$\Delta\bar{P} = h_P[Fr^{f}_{PW}\sqrt{G^{f}_{W}} + \sqrt{F(1 - F)}r^{m}_{PW}\sqrt{G^{m}_{W}}], \qquad \text{(A18)}$$

where h_P is the square root of the heritability of the preference in females.

We can express that result in terms of the phenotypic correlation between a female mating preference and a male display trait among mated pairs. The genetic correlation between preference and fitness in males can be written $r^{m}_{PW} = k^{m}\rho_{PW} = k^{m}r^{m}_{TW}\rho$, where k^m is a factor determined by the mode of inheritance shown in column 3 of Table 1 (e.g., with autosomal inheritance of display, $k^{m} = h_P h_T/2$), ρ_{PW} is the phenotypic correlation between the female preference and male lifetime fitness among mated pairs, r^{m}_{TW} is the correlation between the trait and lifetime fitness, and ρ is the phenotypic correlation between the female preference and male display trait among mated pairs. By the same logic, we can write $r^{f}_{PW} = k^{f}r^{m}_{TW}r^{mf}_{W}\rho$, where r^{mf}_{W} is the genetic correlation between lifetime fitness in males and females, and k^f is the factor corresponding to column 4 of Table 1. (For example, when the preference genes are autosomal, $k^{f} = (\frac{1}{2}h_P h_T$.) Putting these facts together gives

$$\Delta\bar{P} = h_P\rho r^{m}_{TW}\{[Fk^{f}]r^{mf}_{W}\sqrt{G^{f}_{W}} + [\sqrt{F(1 - F)}k^{m}]\sqrt{G^{m}_{W}}\}. \qquad \text{(A19)}$$

Again the contributions caused by selection on females and males appear (respectively) as the two terms within the curly brackets. Factors that are affected by how the preference and fitness genes are inherited are grouped within square brackets. When a genetic correlation between the preference and fitness genes does not exist in one sex (e.g., in females when the fitness genes are Y linked), then the corresponding factor k in that equation takes the value zero.