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Sex-Specific Selection and the Evolution of Between-Sex Genetic Covariance

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Abstract

Because the sexes share a genome, traits expressed in males are usually genetically correlated with the same traits expressed in females. On short timescales, between-sex genetic correlations (r_{mf}) for shared traits may constrain the evolution of sexual dimorphism by preventing males and females from responding independently to sex-specific selection. However, over longer timescales, r_{mf} may evolve, thereby facilitating the evolution of dimorphism. Although it has been suggested that sexually antagonistic selection may reduce r_{mf}, we lack a general theory for the evolution of r_{mf} and its multivariate analog, the between-sex genetic covariance matrix (**B**). Here, we derive a simple analytical model for the within-generation change in **B** due to sex-specific directional selection. We present a single-trait example demonstrating that sex-specific directional selection may either increase or decrease between-sex genetic covariance, depending on the relative strength of selection in each sex and on the current value of r_{mf}. Although sexually antagonistic selection can reduce between-sex covariance, it will only do so when selection is much stronger in one sex than in the other. Counterintuitively, sexually antagonistic selection that is equal in strength in the 2 sexes will maintain positive between-sex covariance. Selection acting in the same direction on both sexes is predicted to reduce between-sex covariance in many cases. We illustrate our model numerically using empirical measures of sex-specific selection and between-sex genetic covariance from 2 populations of sexually dimorphic brown anole lizards (*Anolis sagrei*) and discuss its importance for understanding the resolution of intralocus sexual conflict.

Key words: constraint, genetic correlation, intralocus sexual conflict, sexual dimorphism, sexual selection

Males and females often differ dramatically in the expression of morphological, physiological, and behavioral phenotypes (Darwin 1871; Andersson 1994; Delph 2005; Fairbairn et al. 2007; Fairbairn 2013). The evolution of such sexual dimorphism is favored whenever selection is sex-specific, that is, when selection differs between the sexes. Sex-specific selection may occur when a trait is under selection in only one sex, when selection acts in the same direction in both sexes but differs in magnitude, or when selection acts in opposite directions in the 2 sexes. All these cases are common in nature (Cox and Calsbeek 2009). In addition to sex-specific selection, the evolution of sexual dimorphism requires that genetic variation in one sex is at least partially independent of genetic variation in the other (Lande 1980a, 1987; Chenoweth and McGuigan 2010). Such independence presents somewhat of a paradox because

in most species, males and females share an identical genome apart from the sex chromosomes (Lande 1980a, 1987; Bonduriansky 2007; Fairbairn 2007a; Bonduriansky and Chenoweth 2009). This relationship should cause genetic variation for most traits to be highly correlated across the sexes, which may lead to strong constraints on the evolution of sexual dimorphism. In the face of such constraints, the evolution of sexual dimorphism ultimately requires genetic variation in males and females to become decoupled to some extent. Such decoupling may involve a number of mechanisms, including evolutionary changes in sex-chromosome linkage and sex-biased expression of autosomal genes (Rice 1984; Kirkpatrick and Hall 2004; Rhen 2007; Mank et al. 2008; Bonduriansky and Chenoweth 2009; Connallon and Clark 2010; Parsch and Ellegren 2013; Ingleby et al. 2015; Cheng and Kirkpatrick 2016; Cox et al. 2017; Mank 2017).

In quantitative genetics, the extent to which the evolution of sexual dimorphism is constrained by shared genetic variation may be predicted using an equation derived by Lande (1980a):

$$\begin{bmatrix}\Delta\bar{\mathbf{z}}_\mathbf{m}\\ \Delta\bar{\mathbf{z}}_\mathbf{f}\end{bmatrix} = \frac{1}{2}\begin{bmatrix}\mathbf{G_m} & \mathbf{B}\\ \mathbf{B}^\mathrm{T} & \mathbf{G_f}\end{bmatrix}\begin{bmatrix}\boldsymbol{\beta}_\mathbf{m}\\ \boldsymbol{\beta}_\mathbf{f}\end{bmatrix}. \quad (1)$$

In Equation 1, $\Delta\bar{\mathbf{z}}_\mathbf{m}$ and $\Delta\bar{\mathbf{z}}_\mathbf{f}$ represent evolutionary change in vectors of phenotypic means for males and females, respectively. The effects of directional selection (represented by the vectors of sex-specific selection gradients $\boldsymbol{\beta}_\mathrm{m}$ and $\boldsymbol{\beta}_\mathrm{f}$) are modified by genetic variances and covariances, here represented by the sex-specific matrices $\mathbf{G}_\mathrm{m}$ and $\mathbf{G}_\mathrm{f}$. The response to selection is also influenced by the between-sex genetic covariance matrix **B**, which describes the extent to which male and female traits share genetic variation. The **B** matrix contains between-sex genetic covariances for analogous male and female traits on its diagonal and for heterologous trait pairs as off-diagonals. Because **B** is not necessarily symmetric (i.e., the covariance between trait z_1 in males and trait z_2 in females is not necessarily the same as the covariance between trait z_1 in females and trait z_2 in males), it is shown with the superscript T, which denotes matrix transposition, in the lower left corner. Equation 1 emphasizes that whenever **B** is nonzero, sex-specific selection has consequences for both male and female phenotypes. This occurs because males carry an unexpressed breeding value for female-specific traits and vice versa. As such, the **B** matrix sets limits on the rate at which sexual dimorphism can evolve in a population.

It is often useful to standardize the elements of **B** as between-sex genetic correlations (r_mf). For a single trait, r_mf is defined as the ratio of the between-sex covariance (B) to the square root of the sex-specific genetic variances (G_m and G_f), or

$$r_\mathrm{mf} = \frac{B}{\sqrt{G_\mathrm{m}G_\mathrm{f}}}. \quad (2)$$

When $r_\mathrm{mf} = 1$, selection on one sex will lead to a perfectly correlated response in the other sex, leading to severe constraints on the evolution of sexual dimorphism. A meta-analysis has shown that values of r_mf are often quite large and positive, suggesting that this type of genetic constraint may be quite common (Poissant et al. 2010). When considering the evolution of a set of traits, multivariate constraints come into play, and it is important to consider not only a single r_mf, but both diagonal and off-diagonal elements of **B** (cf. Blows and Hoffmann 2005; Fedorka et al. 2007; Steven et al. 2007; Gosden et al. 2012).

When coupled with sexually antagonistic selection (i.e., selection that acts in different directions in males and females), strong between-sex genetic correlations (or multivariate constraints) may lead to a scenario known as intralocus sexual conflict (Rice 1984; Rice and Chippindale 2001; Arnqvist and Rowe 2005; Bonduriansky and Rowe 2005; Bonduriansky and Chenoweth 2009; Cox and Calsbeek 2009; Connallon et al. 2010). In this scenario, alleles that are harmful to one sex but beneficial to the other may be maintained in the population as long as phenotypes are displaced from their sex-specific optima (Prasad et al. 2007; Bonduriansky and Chenoweth 2009; Connallon et al. 2010). Intralocus sexual conflict can be resolved by mechanisms that reduce the genetic constraints represented by **B**, which may in turn facilitate the evolution of optimal levels of sexual dimorphism (Bonduriansky and Chenoweth 2009).

Although **B** is expected to constrain males and females from responding independently to sex-specific selection in the short term, comparative evidence suggests that in the long term, the constraints may become relaxed. Both within and across studies, more strongly dimorphic traits tend to have lower between-sex genetic correlations than monomorphic or weakly dimorphic traits (Ashman 2003; Delph et al. 2004; Bonduriansky and Rowe 2005; McDaniel 2005; Fairbairn 2007b; Poissant et al. 2010). One interpretation of this pattern is that traits with ancestrally low between-sex genetic correlations are less constrained in their ability to respond to sex-specific selection, and thus achieve greater levels of dimorphism. Another explanation of this pattern, however, is that sex-specific selection itself leads to a reduction in between-sex genetic correlations (Lande 1980a; Bonduriansky and Chenoweth 2009). Experimental evidence indicates that **B** can evolve in response to selection in a small number of generations (Delph et al. 2011), suggesting that this explanation is indeed plausible. To understand whether such phenomena are likely to be important for the evolution of sexual dimorphism, we need not only more empirical studies explicitly addressing the evolution of **B**, but also the development of theory addressing the plausibility of such changes.

Unfortunately, quantitative geneticists currently lack a general model for the evolution of **B**. Quantitative genetic parameters such as **B** are statistical descriptions influenced by diverse phenomena, including allele frequencies, locus effect sizes, pleiotropy, and linkage disequilibrium (Falconer and MacKay 1996; Lynch and Walsh 1998). As such, deriving simple models with exact predictions is difficult, and depends heavily on genetic details (Bulmer 1971; Phillips and McGuigan 2006; Arnold et al. 2008; Walsh and Lynch 2018). However, some theoretical progress can be made via 2 avenues. First, simulations making reasonable genetic assumptions can be used to test how selection causes changes in quantitative genetic parameters over many generations (Jones et al. 2003, 2004; Revell 2007; Arnold et al. 2008). Such simulations are useful because they allow for the specification of genetic details and the simultaneous examination of multiple evolutionary phenomena. Second, analytical equations can be derived that predict the change in quantitative genetic parameters after selection but before reproduction (i.e., before segregation and recombination, Lande 1980b; Phillips and Arnold 1989; Phillips and McGuigan 2006). Although the latter approach cannot be used to make predictions across generations without making assumptions about the nature of genetic architecture, it may still provide insight into the potential effects of single generation of selection and may easily be parameterized with data from wild populations (e.g., McGlothlin et al. 2005).

Here, we derive an equation that predicts within-generation changes in between-sex genetic covariances and sex-specific genetic

variances arising from sex-specific directional and nonlinear selection. Our work expands upon that of Barker et al. (2010), who considered only the effects of sex-specific stabilizing selection. In addition to deriving a general model, we discuss a simplified version that leads to a useful approximation summarizing the effects of sex-specific selection on **B**. We illustrate our model using quantitative genetic parameters and sex-specific selection gradients collected from 2 populations of a highly dimorphic lizard, the brown anole (*Anolis sagrei*), that differ in the extent of sexual size dimorphism they exhibit.

Theory

In a model that did not consider sex-specific selection, Lande (1980b) developed an equation for the predicted within-generation change in **G** that can be expressed in the form

$$\Delta_s \mathbf{G} = \mathbf{G}\left(\boldsymbol{\gamma} - \boldsymbol{\beta}\boldsymbol{\beta}^{\mathrm{T}}\right)\mathbf{G} \tag{3}$$

where $\boldsymbol{\gamma}$ is nonlinear selection, $\boldsymbol{\beta}$ is directional selection, the superscript T indicates matrix transposition (Lande and Arnold 1983; Phillips and Arnold 1989; Phillips and McGuigan 2006). The subscript s indicates that change described by Equation 3 is the change predicted after selection but before segregation and recombination. Such within-generation changes may be translated across generations by making explicit assumptions about genetic architecture (Bulmer 1971; Lande 1980b; Tallis and Leppard 1988). Although Lande (1980a) elsewhere suggested that sex-specific selection may alter **B**, there is no analog to Equation 3 to predict how **B** and the sex-specific genetic (co)variance matrices $\mathbf{G}_{\mathrm{m}}$ and $\mathbf{G}_{\mathrm{f}}$ should be altered by selection (Bonduriansky and Chenoweth 2009). Barker et al. (2010) made some progress toward this goal, deriving an analog of Equation 3 that included only sex-specific nonlinear selection. However, an equation incorporating directional selection is desirable for 2 reasons. First, directional selection, and especially directional sexual selection, is prevalent in nature (Hoekstra et al. 2001; Kingsolver et al. 2001). Second, directional selection may act differently on each sex (Cox and Calsbeek 2009; Morrissey 2016), which should be taken into account when making predictions about the evolution of **B**.

Here, we derive a version of Equation 3 that incorporates sex-specific directional selection. To begin, we write the general equation for within-generation **G**-matrix change as

$$\Delta_s \mathbf{G} = \mathrm{Cov}\left[(\mathbf{a} - \bar{\mathbf{a}})(\mathbf{a} - \bar{\mathbf{a}})^{\mathrm{T}}, w\right] - \Delta_s \bar{\mathbf{a}}\,(\Delta_s \bar{\mathbf{a}})^{\mathrm{T}} \tag{4}$$

where **a** is a column vector of additive genetic values and w is relative fitness (after Lande 1980b). This formulation separates change in **G** into 2 components, the first of which represents the effects of covariance between squared deviations and fitness, that is, nonlinear selection, and the second of which represents the change in mean breeding value due to directional selection. Writing the second component in terms of covariance (Price 1970) and using the accent ~ to denote a deviation from the mean, Equation 4 becomes

$$\Delta_s \mathbf{G} = \mathrm{Cov}(\tilde{\mathbf{a}}\,\tilde{\mathbf{a}}^{\mathrm{T}}, w) - \mathrm{Cov}(\mathbf{a}, w)\,\mathrm{Cov}(w, \mathbf{a}^{\mathrm{T}}). \tag{5}$$

By substituting Lande and Arnold's (1983) regression expression for relative fitness in the absence of sex-specific selection, this equation can be used to recover Equation 3.

An analogous equation for the change in **G** caused by sex-specific selection can be derived in one of 2 ways. First, the covariances in Equation 5 can be solved for each sex and averaged across sexes. Equivalently, we can write an equation for an individual's expected relative fitness averaged across male and female contexts for substitution into Equation 5. We take the latter approach because it is slightly more straightforward. Assuming an equal sex ratio, expected relative fitness may be written as

$$w = \frac{1}{2}(w_{\mathrm{m}} + w_{\mathrm{f}}). \tag{6}$$

Substituting Equation 6 into Equation 5, the total change in the **G** matrix due to selection on both sexes becomes

$$\Delta_s \mathbf{G} = \frac{1}{2}\mathrm{Cov}(\tilde{\mathbf{a}}\,\tilde{\mathbf{a}}^{\mathrm{T}}, w_{\mathrm{m}} + w_{\mathrm{f}}) - \frac{1}{4}\mathrm{Cov}\,(\mathbf{a}, w_{\mathrm{m}} + w_{\mathrm{f}})\,\mathrm{Cov}(w_{\mathrm{m}} + w_{\mathrm{f}}, \mathbf{a}^{\mathrm{T}}). \tag{7}$$

To solve for the effects of sex-specific selection, we write male and female relative fitness as

$$w_{\mathrm{m}} = \alpha_{\mathrm{m}} + \mathbf{z}_{\mathrm{m}}^{\mathrm{T}}\boldsymbol{\beta}_{\mathrm{m}} + \frac{1}{2}\tilde{\mathbf{z}}_{\mathrm{m}}^{\mathrm{T}}\boldsymbol{\gamma}_{\mathrm{m}}\tilde{\mathbf{z}}_{\mathrm{m}} + \varepsilon_{\mathrm{m}} \tag{8a}$$

and

$$w_{\mathrm{f}} = \alpha_{\mathrm{f}} + \mathbf{z}_{\mathrm{f}}^{\mathrm{T}}\boldsymbol{\beta}_{\mathrm{f}} + \frac{1}{2}\tilde{\mathbf{z}}_{\mathrm{f}}^{\mathrm{T}}\boldsymbol{\gamma}_{\mathrm{f}}\tilde{\mathbf{z}}_{\mathrm{f}} + \varepsilon_{\mathrm{f}}. \tag{8b}$$

In Equation 8, between-sex correlational selection ($\boldsymbol{\gamma}_{\mathrm{mf}}$) is assumed to be absent because male and female traits are by definition never expressed in the same individual in dioecious or gonochoric species (Barker et al. 2010; Delph et al. 2011). Substituting Equation 8 into Equation 7 and assuming the genetic basis of all traits is autosomal, we find

$$\Delta_s \mathbf{G} = \begin{bmatrix}\mathbf{G}_{\mathrm{m}} & \mathbf{B}\\ \mathbf{B}^{\mathrm{T}} & \mathbf{G}_{\mathrm{f}}\end{bmatrix}\left(\frac{1}{2}\begin{bmatrix}\boldsymbol{\gamma}_{\mathrm{m}} & 0\\ 0 & \boldsymbol{\gamma}_{\mathrm{f}}\end{bmatrix} - \frac{1}{4}\begin{bmatrix}\boldsymbol{\beta}_{\mathrm{m}}\\ \boldsymbol{\beta}_{\mathrm{f}}\end{bmatrix}\begin{bmatrix}\boldsymbol{\beta}_{\mathrm{m}}\\ \boldsymbol{\beta}_{\mathrm{f}}\end{bmatrix}^{\mathrm{T}}\right)\begin{bmatrix}\mathbf{G}_{\mathrm{m}} & \mathbf{B}\\ \mathbf{B}^{\mathrm{T}} & \mathbf{G}_{\mathrm{f}}\end{bmatrix}. \tag{9}$$

It is apparent from Equation 9 that the effects of directional selection in males and females on change in **G** are not additive; rather, the product of male- and female-specific directional selection must be factored in to predict changes in the genetic (co)variances. We can rearrange Equation 9 to show that sex-specific directional selection creates a component of selection that is similar to correlational selection:

$$\Delta_s \mathbf{G} = \begin{bmatrix}\mathbf{G}_{\mathrm{m}} & \mathbf{B}\\ \mathbf{B}^{\mathrm{T}} & \mathbf{G}_{\mathrm{f}}\end{bmatrix}\begin{bmatrix}\frac{1}{2}\boldsymbol{\gamma}_{\mathrm{m}} - \frac{1}{4}\boldsymbol{\beta}_{\mathrm{m}}\boldsymbol{\beta}_{\mathrm{m}}^{\mathrm{T}} & -\frac{1}{4}\boldsymbol{\beta}_{\mathrm{m}}\boldsymbol{\beta}_{\mathrm{f}}^{\mathrm{T}}\\ -\frac{1}{4}\boldsymbol{\beta}_{\mathrm{f}}\boldsymbol{\beta}_{\mathrm{m}}^{\mathrm{T}} & \frac{1}{2}\boldsymbol{\gamma}_{\mathrm{f}} - \frac{1}{4}\boldsymbol{\beta}_{\mathrm{f}}\boldsymbol{\beta}_{\mathrm{f}}^{\mathrm{T}}\end{bmatrix}\begin{bmatrix}\mathbf{G}_{\mathrm{m}} & \mathbf{B}\\ \mathbf{B}^{\mathrm{T}} & \mathbf{G}_{\mathrm{f}}\end{bmatrix}. \tag{10}$$

The off-diagonal components of the center matrix will be positive when selection is antagonistic and negative when selection is concordant, that is, in the same direction in both sexes. These components are similar to correlational selection, as they involve the cross product of effects of male and female fitness. This result suggests,

somewhat counterintuitively, that sexually antagonistic selection will tend to maintain positive between-sex genetic covariances, while sexually concordant selection will tend to break them down.

To understand the importance of the effects of sex-specific selection on between-sex genetic covariance, it is instructive to examine the simplest case of a single trait expressed in both males and females. Using Equation 9, the change in each genetic variance (G_m and G_f) and the between-sex covariance (B) will equal

$$\Delta_s G_m = \frac{1}{2}\left(G_m^2\gamma_m + B^2\gamma_f\right) - \frac{1}{4}\left(G_m^2\beta_m^2 + B^2\beta_f^2\right) - \frac{1}{2}G_m B\beta_m\beta_f \tag{11a}$$

$$\Delta_s G_f = \frac{1}{2}\left(G_f^2\gamma_f + B^2\gamma_m\right) - \frac{1}{4}\left(G_f^2\beta_f^2 + B^2\beta_m^2\right) - \frac{1}{2}G_f B\beta_m\beta_f \tag{11b}$$

and

$$\Delta_s B = \frac{1}{2}B\left(G_m\gamma_m + G_f\gamma_f\right) - \frac{1}{4}B\left(G_m\beta_m^2 + G_f\beta_f^2\right) - \frac{1}{4}(G_m G_f + B^2)\beta_m\beta_f. \tag{11c}$$

Each of these equations has 3 terms, the first of which represents the effects of nonlinear selection in each sex (γ_m and γ_f), the second the independent effects of directional selection within each sex (β_m and β_f), and the third the interactive effects of selection in the 2 sexes. In these equations, G_m and G_f represent sex-specific genetic variance, while B represents between-sex genetic covariance.

In general, nonlinear selection will reduce variance when it is stabilizing ($\gamma < 0$) and increase it when it is disruptive ($\gamma > 0$). Stabilizing selection also leads to a decrease in the absolute value of B, with disruptive selection having the opposite effect. All selection components in the second term of each equation are squared, which means that the independent effects of directional selection in each sex will be similar to stabilizing selection, reducing both sex-specific variances, as well as B, regardless of the direction of selection. In the third equation, the second term will always have the opposite sign of B, showing that the independent effects of sex-specific directional selection will reduce the absolute magnitude of B.

The third term of each equation is potentially the most interesting, as it will differ in sign depending on whether selection is antagonistic or concordant between the sexes. Sexually antagonistic selection will tend to increase variance when B is positive and decrease it when B is negative, with the opposite holding true for sexually concordant selection. Conversely, sexually antagonistic selection will lead to an increase in B regardless of its sign, while sexually concordant selection will reduce B. Depending on the strength and direction of selection and the magnitude of the genetic (co)variances, the effects of this third term have the potential to outweigh the erosive effects of the second term.

We can make further simplifying assumptions to isolate the effects of sex-specific directional selection. First, we assume that genetic variance is initially equal for males and females ($G_m = G_f = G$) and nonlinear selection is absent. Equation 11a–c then simplify to

$$\Delta_s G_m = -\frac{1}{4}\left(G^2\beta_m^2 + B^2\beta_f^2\right) - \frac{1}{2}GB\beta_m\beta_f \tag{12a}$$

$$\Delta_s G_f = -\frac{1}{4}\left(G^2\beta_f^2 + B^2\beta_m^2\right) - \frac{1}{2}GB\beta_m\beta_f \tag{12b}$$

and

$$\Delta_s B = -\frac{1}{4}BG\left(\beta_m^2 + \beta_f^2\right) - \frac{1}{4}(G^2 + B^2)\beta_m\beta_f. \tag{12c}$$

To simplify even further, we can reduce the genetic component to 2 parameters, the between-sex genetic correlation (r_{mf}) and genetic variance (G). To do so, we replace B with $r_{mf}G$, which after some rearrangement gives

$$\Delta_s G_m = -\frac{1}{4}G^2\left(\beta_m^2 + r_{mf}^2\beta_f^2 + 2r_{mf}\beta_m\beta_f\right) \tag{13a}$$

$$\Delta_s G_f = -\frac{1}{4}G^2\left(\beta_f^2 + r_{mf}^2\beta_m^2 + 2r_{mf}\beta_m\beta_f\right), \tag{13b}$$

and

$$\Delta_s B = -\frac{1}{4}G^2\left(r_{mf}\beta_m^2 + r_{mf}\beta_f^2 + \beta_m\beta_f + r_{mf}^2\beta_m\beta_f\right). \tag{13c}$$

Setting each of these equal to zero, we can solve for the conditions under which genetic (co)variances will not change. Equation 13a,b each have one nontrivial solution:

$$\Delta_s G_m = 0 \quad \text{when} \quad r_{mf} = \frac{-\beta_m}{\beta_f} \quad \text{and} \quad |\beta_m| \le |\beta_f| \tag{14a}$$

and

$$\Delta_s G_f = 0 \quad \text{when} \quad r_{mf} = \frac{-\beta_f}{\beta_m} \quad \text{and} \quad |\beta_m| \ge |\beta_f| \tag{14b}$$

Equation 13c has 2 nontrivial solutions:

$$\begin{aligned}\Delta_s B = 0 \ \text{ when } \ r_{mf} &= \frac{-\beta_m}{\beta_f} \ \text{ and } \ |\beta_m| \le |\beta_f| \\ \text{or when } \ r_{mf} &= \frac{-\beta_f}{\beta_m} \ \text{ and } \ |\beta_m| \ge |\beta_f|.\end{aligned} \tag{14c}$$

This means that one or more (co)variances will change due to selection unless male and female traits are perfectly positively correlated and under equally strong antagonistic selection ($r_{mf} = 1, \beta_m = -\beta_f$) or perfectly negatively correlated and under equally strong concordant selection ($r_{mf} = -1, \beta_m = \beta_f$). Of course, a trivial solution of no genetic variance ($G_m = G_f = B = 0$) is also stable.

In other situations, (co)variance components will change in response to sex-specific selection. These conditions can be simplified even further by considering only the relative magnitudes of the selection gradients in each sex. Using the subscripts "smaller" and "larger" to refer to the relative absolute values of the selection gradients,

$$\Delta_s B > 0 \text{ when } r_{mf} < \frac{-\beta_{smaller}}{\beta_{larger}} \tag{15a}$$

and

$$\Delta_s B < 0 \text{ when } \quad r_{mf} > \frac{-\beta_{smaller}}{\beta_{larger}}. \tag{15b}$$

These relationships are depicted in Figure 1. When r_{mf} is initially strong and positive, selection that is sexually antagonistic and of the same magnitude in both sexes will tend to maintain a high value of B. Conversely, selection that is sexually antagonistic but stronger in one sex than in the other will tend to favor lower values of B. Sexually concordant selection will always reduce B when r_{mf} is positive. Inverse predictions hold for negative values of r_{mf}.

Although it is tempting to view the line in Figure 1 as a line of equilibria, this temptation should be resisted for 2 reasons. First, the evolution of r_{mf} will depend on changes in both B and the 2 genetic variances. Second, predictions across generations must make explicit assumptions about genetic architecture in order to consider processes such as segregation and recombination in addition to selection.

Because our analytical results are somewhat counterintuitive, we used a simulation to visualize the results of our model. We simulated populations of 50 000 individuals with a single trait expressed in both sexes with genetic parameters $G_m = 0.5$, $G_f = 0.5$, and $r_{mf} = 0.75$. These populations were exposed to 1 of 3 survival selection regimes: sexually antagonistic selection that was strong in both sexes, sexually antagonistic selection that was strong in one sex but and weak in the other, and sexually concordant selection that was strong in both sexes. The results of representative simulations are plotted in Figure 2. When selection is sexually antagonistic and strong in both sexes, individuals with extreme phenotypes in each sex are more likely to survive. In the survivors, the distribution of breeding values is elongated along the major axis of covariance, increasing genetic variance in each sex and strengthening the between-sex genetic correlation (Figure 2a). This effect is analogous to disruptive selection along the major axis of variation (cf. Brodie 1992). When selection is sexually antagonistic but strong in males only, the mean of the distribution shifts in the direction of selection on males (Figure 2b). Because extreme phenotypes are not favored as heavily in females, the distribution of survivors does not become elongated along the major axis of covariance, and sex-specific genetic variance does not change appreciably. Rather, the distribution after selection is slightly rounder than the distribution before selection, leading to a slight reduction in B and r_{mf}. These results hold true if selection is strong in females but weak in males (not shown). Finally, when selection is sexually concordant and strong in both sexes, the mean shifts substantially and the distribution becomes noticeably rounder, reducing both genetic variances as well as B and r_{mf} (Figure 2c). This occurs because, although extremes are favored in both sexes, selection in each sex occurs without regard to the breeding value of the opposite sex.

Parameterization

To demonstrate our model numerically, we used data from 2 populations of brown anole lizards (*A. sagrei*) in The Bahamas. Brown anoles are highly sexually dimorphic, but populations vary in the degree of dimorphism. One of our study populations, located on the island of Great Exuma, exhibits relatively high sexual size dimorphism, with males 32% larger than females in snout-vent length (SVL). Our other study population, located on the island of Eleuthera, exhibits substantially lower dimorphism, with males only 22% larger than females in SVL (Table 1, Cox and Calsbeek 2010). Females are similar in size on each island, but males from Great Exuma grow more quickly and reach significantly larger sizes than males from Eleuthera. This population difference in sexual size dimorphism persists in captive-bred individuals, showing that divergence in at least partially genetic (Table 2).

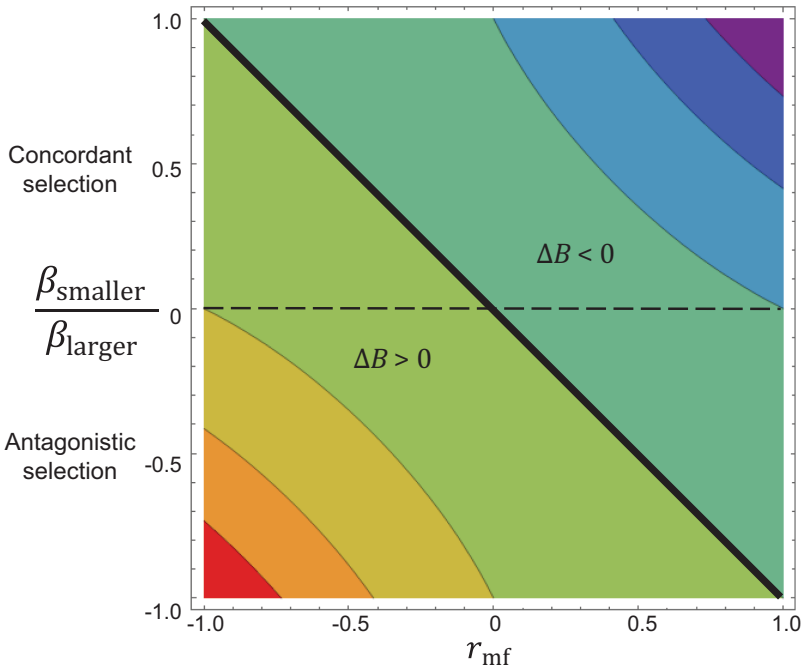

Figure 1. Contour plot depicting the predicted within-generation change in the between-sex genetic covariance (ΔB) as a function of the between-sex genetic correlation (r_{mf}) and the ratio of sex-specific selection gradients ($\beta_{smaller}/\beta_{larger}$) (Equation 15a,b). These results assume zero nonlinear selection and equal genetic variances in males and females ($G_m = G_f = G$). Above the dashed line, selection is in the same direction in each sex (concordant), and below the dashed line, selection is in opposite directions (antagonistic). The solid diagonal line represents parameter combinations where no change in B is expected, with a decrease in B predicted above the diagonal and an increase in B predicted below the diagonal. The absolute magnitude of ΔB is largest in the lower left and upper right corners, with warmer colors indicate larger increases in B and cooler colors indicating larger decreases. The magnitude of ΔB depends on both genetic variation and the strength of selection. Here, contour lines represent units of $\frac{1}{4}(G^2\beta^2_{larger})$.

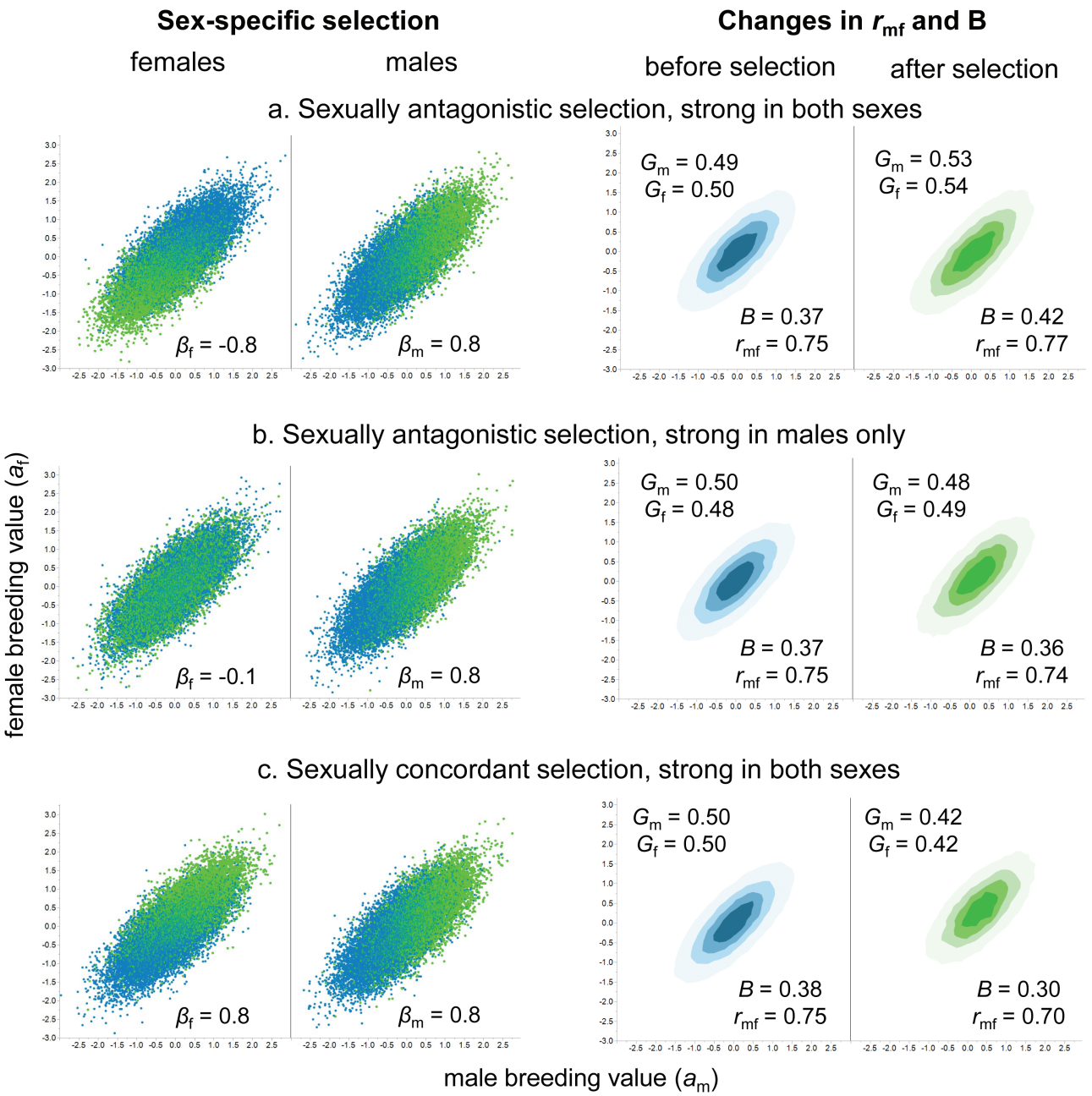

Figure 2. Simulation of sex-specific selection on a single trait. The panels on the left show the distribution of breeding values in both sexes before selection, with eventual survivors in green and nonsurvivors in blue. The panels on the right show density plots visualizing the distribution of breeding values in both sexes before (blue) and after one bout of selection (green). When sexually antagonistic selection is equally strong in both sexes (a), the between-sex genetic correlation is predicted to increase in strength. Antagonistic selection that is strong in only one sex (b) reduces the between-sex genetic correlation slightly, while strong sexually concordant selection (c) leads to a larger reduction in the between-sex genetic correlation.

To understand the potential selective causes of this population difference, Cox and Calsbeek (2010) estimated viability selection on SVL in each population (results reprinted in Table 1). Briefly, they found that selection on male size was directional and positive in both populations, and slightly stronger in the less dimorphic population of Eleuthera. Selection on female size was primarily stabilizing on both islands but contained a positive directional component in the Eleuthera population. Considering directional selection alone, selection appears to be sexually concordant in both populations. However, nonlinear selection was stabilizing in both populations, suggesting the potential for sexual conflict. In Great Exuma in particular, directional selection favored larger males and stabilizing selection favored no change in females, suggesting that selection may become antagonistic if females were displaced from their phenotypic mean. Although these results include only viability selection on adults, a subsequent study found similar results for selection via reproductive success in Great Exuma, suggesting that lifetime selection on SVL may resemble viability selection (Duryea et al. 2016).

For quantitative genetic parameters, we reanalyzed data from Cox et al. (2017), who estimated sex-specific genetic variances and between-sex genetic covariance for SVL in a laboratory study of brown anoles from Great Exuma. Cox et al. (2017) reported

Table 1. Characteristics of study populations and selection gradients for snout-vent length (SVL)

	Eleuthera	Great Exuma
Body size and dimorphism		
Mean SVL, males (mm)	52.24 ± 0.37	56.52 ± 0.39
Mean SVL, females (mm)	42.90 ± 0.14	42.94 ± 0.12
Standard deviation, males	5.30	6.34
Standard deviation, females	2.17	2.40
Sexual dimorphism index	0.22	0.32
Selection on SVL		
Directional (β_m), males	0.279 ± 0.081	0.183 ± 0.049
Directional (β_f), females	0.143 ± 0.080	0.011 ± 0.058
Nonlinear (γ_m), males	−0.064 ± 0.156	−0.006 ± 0.170
Nonlinear (γ_f), females	−0.202 ± 0.202	−0.241 ± 0.094
β_f/β_m	0.512	0.060

Phenotypic standard deviations were not shown in the original article. Sexual dimorphism index is calculated as the ratio of male to female size minus one. Selection gradients were calculated using traits standardized to zero mean and unit variance. Where given, estimates of uncertainty are standard errors. Reprinted from Cox and Calsbeek (2010).

Table 2. Phenotypic characteristics of 24-month-old brown anoles reared in a common garden, alongside genetic (co)variances and the between-sex genetic correlation for body size (Cox et al. 2017; Cox RM and McGlothlin JW, unpublished data)

	Eleuthera	Great Exuma
Body size and dimorphism		
Mean SVL, males (mm)	53.03 ± 0.18	54.56 ± 0.16
Mean SVL, females (mm)	42.02 ± 0.14	41.59 ± 0.09
Standard deviation, males	2.37	2.67
Standard deviation, females	1.84	1.60
Sexual dimorphism index	0.26	0.31
Genetic parameters		
G_m	0.681 ± 0.243	0.291 ± 0.142
G_f	0.107 ± 0.151	0.373 ± 0.136
B	0.052 ± 0.133	0.125 ± 0.099
r_{mf}	0.191 ± 0.484	0.380 ± 0.279

Where given, estimates of uncertainty are standard errors. Genetic parameters are estimated using variance-standardized traits, such that genetic variance components are equivalent to heritabilities. Standard errors for genetic parameters are asymptotic standard errors calculated in ASReml.

results based on natural-log transformed SVL at various ages. We reanalyzed these data and report results from untransformed SVL standardized to unit variance for compatibility with selection estimates from Cox and Calsbeek (2010). We also added analogous data collected from lizards from Eleuthera that were raised in the same laboratory environment following the same protocol (Cox RM, McGlothlin JW, unpublished data). Using a half-sib breeding design (40 sires, 69 dams), 161 male and 166 female offspring from the Eleuthera population were reared alongside the 429 male and 460 female offspring from the Great Exuma population (62 sires, 103 dams). All genetic parameters and their approximate standard errors were estimated in ASReml 4.1 (Gilmour et al. 2015; see Cox et al. 2017 for details). Results from both populations are summarized in Table 2. Briefly, male size was more strongly heritable in Eleuthera than in Great Exuma, female size was more strongly heritable in Great Exuma than in Eleuthera, and the between-sex genetic correlation was stronger in Great Exuma than in Eleuthera. We used Equation 11a–c to predict the change in observed genetic (co)variances due to selection estimated in these populations. We estimated standard errors by recalculating this predicted change from 10 000 random samples drawn from point estimates and their associated error structure and taking the standard error of the resulting distribution (Houle and Meyer 2015). For **G**, we used the approximate error (co)variance matrix estimated by ASReml, and for selection gradients, we used the standard errors reported in Table 1. The predicted change in r_{mf} was calculated by subtracting its initial value from its predicted value after selection. We did not estimate standard errors for the change in r_{mf} because some of the replicate **G** matrices were not positive definite, making calculation of r_{mf} impossible.

In Table 3, we present total predicted change, as well as components of change attributable to nonlinear and directional selection. All estimates of change are associated with large standard errors and should thus be interpreted with caution. Examining the point estimates, all (co)variance components, as well as the between-sex genetic correlations, were predicted to decrease in both populations. These predicted changes were largely attributable to nonlinear selection. Strong negative quadratic selection on female SVL in both populations led to a predicted decrease in both female genetic variance and the between-sex genetic covariance. In each population, the sex with more genetic variance was expected to experience a larger decrease in genetic variance. As expected given the stronger selection gradients, the effect of directional selection on male genetic variance and the between-sex genetic correlation was more intense in Eleuthera than in Great Exuma. In both populations, directional selection was of the same sign, which predicted a reduction in B (Table 1; Figure 1). However, the magnitudes of the male and female selection gradients were more similar in Eleuthera, resulting in

Table 3. Predicted change in genetic parameters using Equation 12 and data from Tables 1 and 2

	Eleuthera	Great Exuma
Change due to β		
$\Delta_s G_m$	−0.0097 ± 0.011	−0.0007 ± 0.001
$\Delta_s G_f$	−0.0002 ± 0.001	−0.0002 ± 0.001
$\Delta_s B$	−0.0015 ± 0.003	−0.0004 ± 0.001
Change due to γ		
$\Delta_s G_m$	−0.0151 ± 0.048	−0.0021 ± 0.012
$\Delta_s G_f$	−0.0012 ± 0.008	−0.0168 ± 0.016
$\Delta_s B$	−0.0017 ± 0.010	−0.0057 ± 0.008
Total change		
$\Delta_s G_m$	−0.0248 ± 0.051	−0.0029 ± 0.012
$\Delta_s G_f$	−0.0015 ± 0.008	−0.0170 ± 0.016
$\Delta_s B$	−0.0032 ± 0.012	−0.0061 ± 0.008
$\Delta_s r_{mf}$	−0.0070	−0.0082

For (co)variances, change is partitioned into components due to directional selection (β) and nonlinear selection (γ).

a larger negative effect on B. This effect arose from the product of the 2 selection gradients in Equation 11c.

When we compared the estimated values of B and r_{mf} in each population (Table 2) to the ratio of directional selection gradients, we found results consistent with our theoretical expectations. Eleuthera showed strong concordant directional selection, which predicts a larger negative change in B than in Exuma, where selection is still concordant in direction, but stronger in magnitude in males. Consistent with predictions, Eleuthera displayed lower values of both B and r_{mf}, which suggests that these values may have been reduced by selection in Eleuthera relative to Exuma.

Discussion

We derived a general model for the effects of sex-specific selection on male and female genetic variances and the between-sex genetic covariance (B). In an illustrative special case, our model predicts that the effects of sex-specific directional selection on B will depend on both the direction and relative magnitude of selection in each sex. When selection is antagonistic but of similar magnitude in males and females, strong positive values of B should be maintained. When selection is antagonistic but much stronger in one sex than in the other, B should often decrease. Selection that is in the same direction in both sexes will lead to a larger decrease in B.

Selection may differ between the sexes in direction, magnitude, or both (Cox and Calsbeek 2009; Morrissey 2016). When selection is antagonistic in direction and equally strong in both sexes, our model suggests that directional selection alone will be unable to resolve intralocus sexual conflict by reducing between-sex genetic covariance. Instead, equally strong antagonistic selection is expected to maintain strong positive between-sex genetic covariance, ensuring that conflict persists. Consistent with this prediction, Stewart and Rice (2018) found that 250 generations of sexually antagonistic artificial selection on size in a laboratory population of *Drosophila melanogaster* led to no change in males and only a small change in females. Although that study did not measure changes in the between-sex genetic correlation, these results suggest that r_{mf} remained likely large and positive in spite of strong sexually antagonistic selection.

In natural populations, selection is often much stronger in one sex than in the other. In particular, sexual selection, which tends to be the strongest type of directional selection, is commonly much stronger in males (Hoekstra et al. 2001; Shuster and Wade 2003; Cox and Calsbeek 2009). For example, a sexually selected ornamental trait may be greatly beneficial to males but only moderately costly when expressed in females, which would lead to a small negative value of β_f/β_m. Our model predicts that under such conditions, selection should act to reduce between-sex genetic covariances, helping to resolve sexual conflict and to facilitate the elaboration of sexual dimorphism.

Although our theoretical treatment focused primarily on directional selection, the potential influence of nonlinear selection should not be ignored. Stabilizing selection, even in a single sex, may lead to a predicted decline in the between-sex genetic covariance as well as sex-specific genetic variances. Equation 9 suggests that the influence of nonlinear selection will tend to be twice as strong as that of directional selection. Parameterizing this equation with data from brown anoles shows that the influence of stabilizing selection in a single sex may indeed overwhelm the effects of sex-specific directional selection. This result suggests that differences in both directional and nonlinear selection should be considered to understand the resolution of sexual conflict.

Comparison to Population Genetic Models

Although our results may seem counterintuitive at first glance, they are largely in accordance with population genetic models of sex-specific selection. Single-locus models (Kidwell et al. 1977; Connallon and Clark 2012) do not directly model changes in B or r_{mf}, but instead tend to focus on the conditions under which sexually antagonistic selection will favor the maintenance of genetic polymorphism. Such polymorphism is directly related to the between-sex genetic covariance; segregating variation at loci that affect male and female phenotypes similarly will maintain both overall genetic variance and between-sex genetic covariance. Single-locus models predict that sexually antagonistic selection will maintain polymorphism only when an allele is nearly as detrimental to one sex as it is beneficial to the other (Kidwell et al. 1977; Connallon and Clark 2012). In phenotypic selection terminology, this is analogous to the case when β_f/β_m is approximately equal to negative one. Such segregating polymorphism at a sexually antagonistic locus should contribute to high between-sex genetic covariance; in general, quantitative genetic (co) variance is higher when allele frequencies are intermediate (Falconer and MacKay 1996). This conclusion agrees with our prediction that high negative values of β_f/β_m should maintain high positive values of r_{mf}. As selection becomes much stronger in one sex, single-locus models predict that sexually antagonistic alleles that are beneficial to one sex and deleterious to the other will begin to fix, reducing both genetic variance and covariance. This reduction in polymorphism should lead to lower values of r_{mf}, at least temporarily.

Two-locus population genetic models show that sexually antagonistic selection can generate linkage disequilibrium as well (Patten et al. 2010; Ubeda et al. 2011). As in single-locus models, when sexually antagonistic selection is of similar strengths in males and females, more polymorphism is maintained, in this case because more linkage disequilibrium is generated. Such linkage disequilibrium builds up under sexually antagonistic selection because haplotypes that consist of 2 male-beneficial or 2 female-beneficial alleles at 2 loci are more common in gametes after selection (Ubeda et al. 2011). This maintenance of polymorphism via linkage disequilibrium should also lead to higher positive between-sex genetic covariances when directional selection is of similar strength in each sex.

When selection acts in the same direction in both sexes, standard population genetic models would predict that alleles that either increase or decrease the trait in both sexes (depending on the direction

of selection) would become fixed. From a quantitative genetic perspective, both sex-specific genetic variances and *B* should decrease as alleles fix. Therefore, the apparent prediction made by Figure 1 that concordant selection should lead to negative values of r_{mf} is probably unrealistic. Instead, the more likely scenario is that concordant selection will tend to deplete genetic variances and covariances at about the same rate (Equation 11). Because the between-sex genetic correlation has genetic variances in its denominator (Equation 2), r_{mf} will become undefined when either genetic variance is equal to zero.

Across-Generation Changes

Unlike population-genetic models, the model we develop here applies only to change in **B** within a generation (i.e., after selection but before mutation, segregation, and recombination, Lande and Arnold 1983). Although our model may help to illuminate the effects of selection *per se*, it does not include specific assumptions about the genetic system (e.g., number of loci, number of alleles, and recombination rate), and thus should not be used to make predictions about changes across generations. This caveat applies equally to earlier quantitative genetic models that did not consider sex-specific selection (Phillips and McGuigan 2006). Despite this fact, however, observed patterns of genetic (co)variance often fit what would be predicted from a within-generation model alone (Brodie 1989, 1992; McGlothlin et al. 2005; Roff and Fairbairn 2012), which suggests that our model may have some utility for studying the evolution of sex-specific genetic architecture in the wild.

Analytical models of cross-generation change may be developed by applying the infinitesimal model, which assumes that genetic variance is caused by a very large number of loci with very small effects (Bulmer 1971; Walsh and Lynch 2018). Under these conditions, response to selection in both the mean and variance derive from changes in gametic phase disequilibrium rather than changes in allele frequencies. Analyzing the infinitesimal model, Bulmer (1971) showed that only half of the within-generation change in the genetic variance is carried to the next generation. At the same time, half of the existing disequilibrium generated by selection is lost each generation through recombination. The result of these opposing processes is that the cumulative change in the genetic variance is small, with selection and recombination rapidly reaching an equilibrium (Bulmer 1971; Walsh and Lynch 2018). Bulmer's logic applies equally well to genetic covariances. Therefore, our within-generation model should not be used naively to make predictions about long-term changes in **B**, especially when the infinitesimal model applies.

When allele frequencies do change appreciably, the relationship between within- and cross-generation change is much less clear. Future work should build upon our within-generation results and develop of explicit multilocus models of **B** evolution that enable predictions that extend across generations. Multilocus models are notoriously difficult to treat analytically (Barton and Turelli 1991; Kirkpatrick et al. 2002), and more insight is likely to be derived from simulation models (Jones et al. 2003, 2004, 2007, 2012, 2014; Arnold et al. 2008). Results from one such simulation model that was not explicitly designed to examine the evolution of **B** tentatively suggest that the predictions of our model may carry over to cross-generation change in some cases. Reeve and Fairbairn (2001) modeled the evolution of sexual dimorphism using a stochastic genetic simulation with 50 autosomal loci, 3 of which had sex-specific expression. In that model, the between-sex genetic correlation decreased rapidly due to allele frequency change, doing so to a greater extent in a model that included sexual selection (i.e., a larger difference in selection between the sexes). However, the predicted change may be transient; when both sexes reached their equilibrium (i.e., when sex-specific directional selection was absent), genetic correlations returned to their starting points.

The simulation framework introduced by Jones et al. (2003, 2004, 2007, 2012, 2014; Arnold et al. 2008) to model the evolution of **G**, which explicitly simulates change at a relatively small number of polymorphic loci, could be modified to include sex-specific optima to model the long-term evolution of **B**. Although the within-generation effects modeled here are likely to be transient in such a simulation, sex-specific selection may have long-term consequences for the evolution of genetic architecture, especially in the mutational architecture of traits is allowed to evolve, as in Jones et al. (2007, 2014). Changes in sex-specific gene expression due to epistatic interactions with other genes or physiological mediators (such as sex hormones, Cox et al. 2016, 2017) may allow mutations to have sex-independent effects, leading to long-term changes in between-sex genetic covariance.

Conclusion

We developed a model for predicting the within-generation effects of sex-specific selection on both within-sex genetic variances and between-sex genetic covariances. Our model extends previous work by explicitly incorporating the effects of sexually antagonistic directional selection, which is common in nature (Cox and Calsbeek 2009). Although such sexually antagonistic selection is often intuitively assumed to erode between-sex genetic covariance, our model shows that sexually antagonistic selection will favor a reduction in between-sex genetic covariance only when the strength of selection is highly asymmetric between the sexes. Contrary to what is often assumed, sexually antagonistic selection will tend to maintain strong between-sex genetic covariance when the strength of selection is similar in each sex. Moreover, sexually concordant selection will often be more effective than sexually antagonistic selection at reducing between-sex genetic covariance over short evolutionary timescales. These results suggest that, in the short term, sexually antagonistic selection may paradoxically maintain sexual conflict when selection is of equal magnitude in each sex, though it may help to resolve conflict when selection is much stronger in one sex than in the other. Future work should attempt to extend our conclusions, which apply strictly to within-generation changes, to the long-term evolution of sex-specific genetic architecture.

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