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A multivariate view of the evolution of sexual dimorphism

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Abstract

Sexual differences are often dramatic and widespread across taxa. Their extravagance and ubiquity can be puzzling because the common underlying genome of males and females is expected to impede rather than foster phenotypic divergence. Widespread dimorphism, despite a shared genome, may be more readily explained by considering the multivariate, rather than univariate, framework governing the evolution of sexual dimorphism. In the univariate formulation, differences in genetic variances and a low intersexual genetic correlation (r_{MF}) can facilitate the evolution of sexual dimorphism. However, studies that have analysed sex-specific differences in heritabilities or genetic variances do not always find significant differences. Furthermore, many of the reported estimates of r_{MF} are very high and positive. When monomorphic heritabilities and a high r_{MF} are present together, the evolution of sexual dimorphism on a trait-by-trait basis is severely constrained. By contrast, the multivariate formulation has greater generality and more flexibility. Although the number of multivariate sexual dimorphism studies is low, almost all support sex-specific differences in the **G** (variance-covariance) matrix; **G** matrices can differ with respect to size and/or orientation, affecting the response to selection differently between the sexes. Second, whereas positive values of the univariate quantity r_{MF} only hinder positive changes in sexual dimorphism, positive covariances in the intersexual covariance **B** matrix can either help or hinder. Similarly, the handful of studies reporting **B** matrices indicate that it is often asymmetric, so that **B** can affect the evolution of single traits differently between the sexes. Multivariate approaches typically demonstrate that genetic covariances among traits can strongly constrain trait evolution when compared with univariate approaches. By contrast, in the evolution of sexual dimorphism, a multivariate view potentially reveals more opportunities for sexual dimorphism to evolve by considering the effect sex-specific selection has on sex-specific **G** matrices and an asymmetric **B** matrix.

Introduction

The evolution of sexual dimorphism has long fascinated biologists. Males and females obviously differ with regard to reproduction, but sometimes the sexes can diverge sufficiently in other aspects of the phenotype so as to be mistaken as different species. Although sexual dimorphism is widespread, its evolution is not necessarily easy to explain. Shared traits can have the same amount of genetic variance in each sex and a high degree of genetic correlation that together will impede phenotypic sexual divergence (Lande, 1980). How the sexes circumvent a common genetic architecture to evolve sex-specific differences remains an important question.

Evolutionary biologists employ two basic approaches to study trait evolution in response to selection. The univariate approach models evolution one trait at a time. By contrast, the multivariate approach includes additional traits that may or may not be correlated with the focal trait so that all traits are considered simultaneously.

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The multivariate approach can yield a predicted evolutionary response that differs from the univariate approach if genetic correlations between traits are substantial. In fact, multivariate studies typically reveal trait combinations that have little or no genetic variation and cannot respond to strong selection, even though the individual traits may have abundant genetic variation (Walsh & Blows, 2009). Thus, including more traits in the multivariate approach not only greatly complicates the study of their evolution, but typically suggests limited capacity to respond to selection overall. However, studying the evolution of multivariate sexual dimorphism suggests fewer potential constraints than the evolution of sexual dimorphism in single traits, as we elaborate further below.

Predicting the evolution of sexual dimorphism extends both the univariate and multivariate equations by considering the response to selection in two distinct populations – males and females. The univariate formulation includes sex-specific genetic variances and a measure of how genetically correlated males and females are for the shared focal trait. The multivariate formulation includes sex-specific genetic variances of all traits and between-sex genetic covariances for shared traits and across different traits – further complicating the quantitative genetic equations that predict the evolution of sexual dimorphism. Despite this increased complexity, we suggest that keeping track of these additional sex-specific variances and between-sex covariances actually reveals the potential for greater ease in evolving sexual dimorphism. As a result, multivariate methods – which typically expose constraints not seen in the univariate formulation – actually provide more potential for phenotypic divergence between the sexes and may partially explain the widespread nature of sexual dimorphism.

Here, we review and compare the univariate and multivariate equations that describe the evolution of sexual dimorphism. In particular, we examine aspects of the genetic architecture that can affect sex-specific responses to selection. Under the univariate view, the sex-specific genetic variances and the intersexual genetic covariance of a single trait affect how the sexes respond to sex-specific selection. Under the multivariate view, sexual dimorphism in the **G** (genetic variance–covariance) matrix and additional intersexual genetic covariances in the **B** (intersexual genetic covariance) matrix affect the response to sex-specific selection. Keeping track of these new high-dimensional quantities can provide additional avenues for sexual divergence. First, sex-specific **G** matrices can have an orientation and magnitude. The sexes may differ with respect to one or both features so that equivalent selection can lead to sexual dimorphism. Second, the **B** matrix can be asymmetric so that equivalent selection will affect one sex more and can lead to sexual dimorphism in shared single traits. We explain sex-specific **G** matrices and the asymmetric **B** matrix and discuss their implications. Finally, we emphasize that the increased potential for the evolution of sexual dimorphism under the multivariate view is not due to changes in the nature of genetic variation itself, but rather in how completely we view it.

Constraints in the evolution of sexual dimorphism

Univariate changes in sexual dimorphism

The univariate breeder's equation, $R = h^2 s$, describes the phenotypic response to selection in a single trait. Heritability, h^2, is the additive genetic variance of the trait, σ_A^2 (V_A), divided by the total phenotypic variance, σ_P^2 (V_P). The additive genetic variance is the variance due to the average effects of alleles, and phenotypic variance is the square of the phenotypic standard deviation. The selection differential, s, is defined as the difference in the phenotypes between the mean of the entire population and the mean after selection (Falconer & Mackay, 1996). Thus, the response to selection, R, is the difference in the mean of the entire population before selection and the mean of the offspring of the selected parents (Falconer & Mackay, 1996; Lynch & Walsh, 1998).

The univariate breeder's equation can be extended to predict trait changes in males ($\Delta\bar{Z}_m$) and females ($\Delta\bar{Z}_f$) separately:

$$\begin{aligned}\Delta\bar{Z}_m &= \frac{1}{2}(h_m^2\sigma_{P_m} i_m + h_m h_f r_{MF}\sigma_{P_m} i_f)\\ \Delta\bar{Z}_f &= \frac{1}{2}(h_f^2\sigma_{P_f} i_f + h_f h_m r_{MF}\sigma_{P_f} i_m).\end{aligned} \quad (1)$$

The coefficient 1/2 is introduced to take into account the autosomal contribution from each parent. h_m^2 and h_f^2 are the sex-specific heritabilities, which are the sex-specific genetic variances ($\sigma_{A_m}^2 = V_{A_m}$; $\sigma_{A_f}^2 = V_{A_f}$) divided by the sex-specific phenotypic variances ($\sigma_{P_m}^2 = V_{P_m}$; $\sigma_{P_f}^2 = V_{P_f}$). i is the intensity, which is the selection differential divided by the phenotypic standard deviation (s/σ_P).

At first, it may seem mysterious that males and females could have different additive genetic variances ($\sigma_{A_m}^2 \neq \sigma_{A_f}^2$) because they share almost the entire genome. However, as genetic variances can be imagined as the sum of locus-specific allelic variances, sexual dimorphism in allele frequencies, additive effects and dominance effects can all contribute. Empirically, alleles can have different additive effects or different dominance coefficients in males versus females (Fry, 2010). New mutations may have sex-specific effects that lead to allele frequency differences between the sexes. Such sex-specific effects may arise due to sex-linkage; although the breeder's equation (3) assumes autosomal

linkage of genes, sex chromosomes can often affect the expression of autosomal genes. Several studies have found statistically significant sex-specific differences in heritabilities or additive genetic variances (e.g. Mousseau & Roff, 1989; Wilcockson *et al.*, 1995; Ashman, 1999, 2003; Mignon-Grasteau, 1999; Jensen *et al.*, 2003; Rolff *et al.*, 2005; Fedorka *et al.*, 2007; Zillikens *et al.*, 2008; Gershman *et al.*, 2010; Stillwell & Davidowitz, 2010).

The new quantity, r_{MF}, is the intersexual genetic correlation, and it describes the degree of correlation between the sexes. It is the covariance of the additive effects between males and females ($\mathrm{Cov}_{A_{MF}}$) divided by the square root of the product of the male-specific additive variance (V_{A_m}) and female-specific additive variance (V_{A_f}):

$$r_{MF} = \frac{\mathrm{Cov}_{A_{MF}}}{\sqrt{V_{A_M} V_{A_F}}}. \quad (2)$$

When r_{MF} is positive, selection in one sex will produce a similar response in the opposite sex, according to the strength of the correlation. When r_{MF} is zero, selection on one sex will produce no response in the opposite sex; when it is negative, it will produce an opposite response in the opposite sex. r_{MF} is simply a specific case of the genetic correlation measured between traits, r_g.

The mean change in a male character ($\Delta\bar{Z}_m$) or female character ($\Delta\bar{Z}_f$) is governed by the sex-specific genetic variances and the degree of correlation between the sexes for the shared trait. The change in the sexual dimorphism (ΔSD) may be described as the difference in the response to selection for males and females, measured as changes in trait means:

$$\begin{aligned}\Delta SD &= \Delta\bar{Z}_m - \Delta\bar{Z}_f \\ &= \frac{1}{2}[h_m^2\sigma_{P_m} i_m - h_f^2\sigma_{P_f} i_f + h_m h_f r_{MF}(\sigma_{P_m} i_f - \sigma_{P_f} i_m)].\end{aligned} \quad (3)$$

As equation 3 is complicated, it is easier to see the effect of r_{MF} by making a few simplifications. To illustrate, we follow the Cheverud *et al.* (1985) decomposition of the univariate breeder's equation for sexual dimorphism, which assumed that heritabilities and phenotypic variances are the same in each sex ($h_m^2 = h_f^2 = h^2$ and $\sigma_{P_m}^2 = \sigma_{P_f}^2 = \sigma_P^2$):

$$\Delta SD = \frac{1}{2} h^2 \sigma_P (i_m - i_f)(1 - r_{MF}). \quad (4)$$

Because of the negative sign in front of r_{MF}, large positive correlations always have a constraining effect on positive, male-biased changes in sexual dimorphism, that is, $i_m - i_f > 0$ such that $\Delta SD > 0$ (NB: for simplicity in this paper we only consider positive changes in sexual dimorphism due to stronger selection in males; however, female-biased changes may be easily substituted). The absolute constraining effect of $r_{MF} = 1$ rests upon the assumption of identical genetic variances (Leutenegger & Cheverud, 1982; Slatkin, 1984; Cheverud *et al.*, 1985; Leutenegger & Cheverud, 1985; Reeve & Fairbairn, 1996; Lynch & Walsh, 1998; Reeve & Fairbairn, 2001; Bonduriansky & Chenoweth, 2009; Poissant *et al.*, 2009).

The negative correlation between r_{MF} and the extent of sexual dimorphism predicted by equation 4 has received empirical support. A study of antler flies revealed an overall negative relationship between r_{MF} and morphological sexual dimorphism (Bonduriansky & Rowe, 2005). When the intersexual correlation for a shared trait was negative or low, the trait was more dimorphic. By contrast, when the intersexual correlation was positive and substantial, the trait was less dimorphic. A recent survey confirms that this negative relationship extends to a variety of taxa and trait types (Poissant *et al.*, 2009). Yet, there is a great deal of scatter in this relationship, particularly at low sexual dimorphism values (e.g. Bonduriansky & Rowe, 2005; Poissant *et al.*, 2009). Low phenotypic dimorphism could be associated with high or low r_{MF}. The intersexual genetic correlation may be small when the intersexual covariance is small, or when the sex-specific genetic variances are large, or both - suggesting why the correlation between r_{MF} and phenotypic sexual dimorphism can sometimes be weak (Poissant *et al.*, 2009). Simulations confirm that high positive r_{MF} can be associated with both low and high sexual dimorphism, as measured by means and phenotypic standard deviations (Reeve & Fairbairn, 2001). Furthermore, although temporary decreases in r_{MF} are necessary for the evolution of sexual dimorphism, the equilibrium magnitude of r_{MF} ultimately depends upon differences in selection, mutation and the number of concordantly and discordantly selected alleles between the sexes (Bonduriansky & Chenoweth, 2009). For instance, sex-specific selection may make some loci sex-limited in expression to accommodate greater phenotypic sexual dimorphism. Sex-limited expression can potentially increase r_{MF} because the intersexual covariance in the numerator will depend only upon the remaining concordantly selected alleles. However, if the new sex-limited loci are still polymorphic, male variance increases in the denominator may instead decrease the overall value of r_{MF}. The interplay between concordantly and discordantly selected alleles may further explain why r_{MF} and sexual dimorphism need not be negatively correlated.

The potentially confounding standardization introduced by r_{MF} demonstrates that examining the unstandardized intersexual covariance directly can also be helpful (see also Walsh & Blows, 2009; Conner, 2012). It is possible to recast the univariate equation 3 for sexual dimorphism by making the components of r_{MF} explicit:

$$\Delta SD = \Delta\bar{Z}_m - \Delta\bar{Z}_f = \frac{1}{2}\left[\left(\frac{V_{A_m}}{V_{P_m}}s_m - \frac{V_{A_f}}{V_{P_f}}s_f\right) - \mathrm{Cov}_{A_{MF}}\left(\frac{s_m}{V_{P_m}} - \frac{s_f}{V_{P_f}}\right)\right]. \quad (5)$$

Because the sign before the covariance term is negative, positive covariance values will always inhibit positive, male-biased changes in sexual dimorphism (i.e. selection is stronger in males than in females, $\frac{s_m}{V_{P_m}} - \frac{s_f}{V_{P_f}} > 0$), whereas negative covariance values will accentuate it.

Few studies have explicitly examined sex-specific differences in additive genetic variances and shown significant differences on a trait-by-trait basis. A recent analysis (Wyman and Rowe, *unpublished data*) showed that the overall mean difference in male- and female-specific heritabilities was not statistically different from zero (although extreme differences did occur for certain traits). If the same traits with monomorphic heritabilities also have high r_{MF}, genetic constraints may indeed be widespread in single traits that covary little with other traits.

Multivariate changes in sexual dimorphism

Here, we move from a single trait view to the multivariate framework proposed by Lande and Arnold (Lande, 1979; Lande & Arnold, 1983). Because of correlations among traits, selection on one trait can cause an indirect response to selection in other traits. The multivariable response to selection is modelled as:

$$\Delta\bar{\mathbf{Z}} = \mathbf{G}\beta, \quad (6)$$

where **G** is the additive genetic variance-covariance matrix for the traits under consideration whereas $\boldsymbol{\beta}$ is the vector of the selection gradients for each trait. $\boldsymbol{\beta}$ results from multiplying the inverse of the phenotypic variance-covariance matrix, $\mathbf{P^{-1}}$, and the vector of selection differentials, **s**.

Lande (1980) repurposed this multivariate approach to consider the evolution of sexual dimorphism because trait expression can be correlated between the sexes. Between- and within-sex evolution of traits is modeled as:

$$\begin{pmatrix}\Delta\bar{\mathbf{Z}}_\mathbf{m}\\ \Delta\bar{\mathbf{Z}}_\mathbf{f}\end{pmatrix} = \frac{1}{2}\begin{pmatrix}\mathbf{G_m} & \mathbf{B}\\ \mathbf{B^T} & \mathbf{G_f}\end{pmatrix}\begin{pmatrix}\beta_\mathbf{m}\\ \beta_\mathbf{f}\end{pmatrix}. \quad (7)$$

$\Delta\bar{\mathbf{Z}}_\mathbf{m}$ and $\Delta\bar{\mathbf{Z}}_\mathbf{f}$ are vectors representing mean changes in traits for males and females, respectively. $\mathbf{G_m}$ and $\mathbf{G_f}$ represent the sex-specific additive genetic variance–covariance matrices for autosomal traits. $\beta_\mathbf{m}$ and $\beta_\mathbf{f}$ are the vectors containing the sex-specific selection gradients. **B** is the matrix of the intersexual covariances; the *ij*th element of the **B** matrix is the additive genetic covariance for character *i* expressed in males and character *j* expressed in females. Unlike **G** or **P** matrices, **B** is not necessarily symmetrical: the off-diagonal elements are not equivalent on either side of the diagonal, for example Cov(*Trait* i^f, *Trait* j^m) $\neq$ Cov(*Trait* i^m, *Trait* j^f), where the superscripts *m* and *f* indicate which sex expresses traits *i* and *j*. Alternatively, **B**'s asymmetry may be expressed as $\mathbf{B} \neq \mathbf{B^T}$ where $\mathbf{B^T}$ is the transpose matrix of **B**.

The change in sexual dimorphism is defined as the difference in the mean changes in males and females, $\mathbf{\Delta SD} = \Delta\bar{\mathbf{Z}}_\mathbf{m} - \Delta\bar{\mathbf{Z}}_\mathbf{f}$. **ΔSD** expanded yields:

$$\mathbf{\Delta SD} = \frac{1}{2}[(\mathbf{G_m}\beta_\mathbf{m} + \mathbf{B}\beta_\mathbf{f}) - (\mathbf{G_f}\beta_\mathbf{f} + \mathbf{B^T}\beta_\mathbf{m})]. \quad (8)$$

Because of the complexity of this equation, Lande (1980) made a few simplifying assumptions to elucidate its meaning. First, he assumed monomorphic **G** matrices. Second, he assumed that $\mathbf{B} = \mathbf{B^T}$, resulting in:

$$\mathbf{\Delta SD} = \frac{1}{2}[(\mathbf{G} - \mathbf{B})(\beta_\mathbf{m} - \beta_\mathbf{f})]. \quad (9)$$

In other words, the extent of change in sexual dimorphism is governed by the similarity of the **G** and **B** matrices, in addition to differences in selection gradients. As a result, if male and female **G** matrices are the same and if the intersexual covariance is complete, the **B** matrix is simply the **G** matrix; under these circumstances, no divergence is possible. However, what kind of support exists for monomorphic **G** matrices and a symmetrical **B** matrix?

Sexual dimorphism in G matrices

In the univariate formulation, a single variance value only has a magnitude, whereas in the multivariate formulation, the **G** matrix has both a magnitude (i.e. eigenvalues) and an orientation (i.e. eigenvectors). Eigenvectors describe a direction in multi-trait space, whereas eigenvalues describe the variation in that direction. Eigenvectors reveal the direction where the genetic variance is oriented, whereas eigenvalues reveal how fast the predicted response to selection along that direction will be (Walsh & Blows, 2009).

Because of this distinction, comparing **G** matrices between the sexes is more involved than statistically comparing a pair of heritabilities or variances. It is possible to do an element-by-element comparison of the heritabilities and genetic correlations in the male and female **G** matrices (e.g. Steven *et al.*, 2007; Leinonen *et al.*, 2010), but such one-to-one comparisons between matrices can be misleading (Walsh, 2007). So, better yet is to take advantage of the multivariate framework and compare eigenvectors and eigenvalues between the sexes. For instance, the Flury hierarchy analysis compares a set of eigenvectors and eigenvalues for their shapes and relative sizes (Phillips & Arnold, 1999). The Flury analysis proceeds by testing a nested set of hypotheses in an ascending manner, or by comparing the various hypotheses with the hypothesis of no relationship. **G** matrices may be identical, sharing the same

eigenvectors and same eigenvalues. Or, they may be proportional, sharing the same eigenvectors but having eigenvalues that differ by a constant factor. Alternatively, **G** matrices may share the same eigenvectors but have nonproportional eigenvalues (i.e. full common principal components (CPC)) or share a subset of eigenvectors (i.e. partial CPC). Finally, **G** matrices may not share any eigenvectors and be unrelated (Phillips & Arnold, 1999). However, although intuitive, the Flury hierarchy tends to overestimate matrix differences and may miss underlying similarities (e.g. Houle *et al.*, 2002; Mezey & Houle, 2003). For instance, two matrices might share a common eigenvector but for different associated eigenvalues (e.g. the largest eigenvalue in one matrix and the second largest eigenvalue in the other matrix).

The difference between genetic variance in univariate and multivariate space can be seen by comparing two vectors in one- and two-dimensional space (Fig. 1). In one-dimensional space, male and female heritabilties lie along a number line. Their difference is one factor that contributes to the change in the extent of sexual dimorphism. When $h_m^2 = h_f^2$, the heritabilities cancel out (Fig. 1a); the evolution of sexual dimorphism depends only upon differences in the strength of sex-specific selection and the intersexual genetic covariance of the shared single trait (equation 4). In two-dimensional space (i.e. two different traits under consideration), sexual dimorphism in genetic variances may be measured by the direction of the greatest genetic variance ($\mathbf{g_{max}}$, or principal component (PC) 1 of the **G** matrix). $\mathbf{g_{max}}$ for the male and female **G** matrices may have the same magnitudes but point in different directions (Fig. 1b). By considering a second trait, the multivariate view illustrates the potential for sexual dimorphism to evolve through orientation differences even though the total amount of genetic variation is the same. Similarly, even if the male $\mathbf{g_{max}}$ and female $\mathbf{g_{max}}$ point in the same direction, differences in their magnitude will provide genetic variance for the evolution of sexual dimorphism (Fig. 1c). Thus, sexual dimorphism in magnitude and/or direction of the genetic variances – along with the covariance structure and sex-specific selection – contributes to phenotypic divergence. The potential for a multivariate view to accommodate more avenues for evolving sexual dimorphism contrasts with the typical view of multivariate quantitative genetics, so that including additional traits typically leads to more constraints (Walsh & Blows, 2009).

We emphasize that the fundamental genetic architecture does not alter when moving from a univariate to a multivariate view. Neither does the process of evolving sexual dimorphism change. In fact, differences in eigenvalues and eigenvectors indicate that sex-specific genetic variances in single traits differ. However, increasing the number of traits alters our perspective on how the evolution of dimorphism proceeds. For

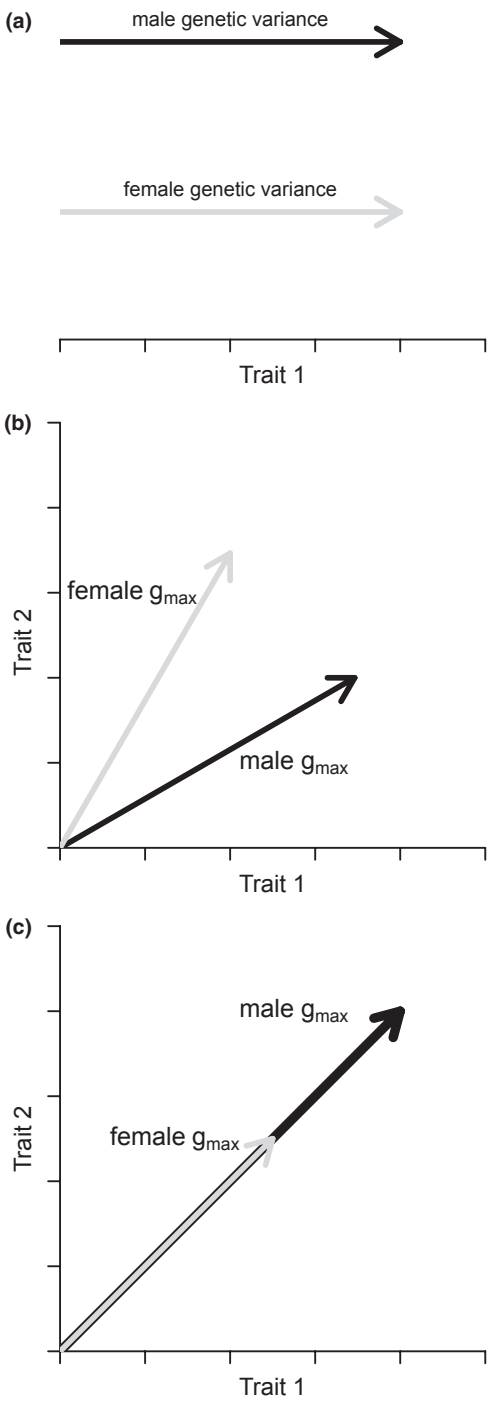

Fig. 1 Male and female variances in one dimension (a) and two dimensions (b and c). The difference in genetic variances between the sexes is one factor that contributes to the total change in sexual dimorphism. In single traits, differences only occur through differences in the magnitude of sex-specific genetic variances. In multivariate space, genetic variances can have the same magnitude (equal lengths of male and female $\mathbf{g_{max}}$) but still differ in orientation (b). Similarly, genetic variances can have the same orientation, but still differ in magnitude (c).

instance, if three traits are under consideration but the observer considers only pairwise combinations, each combination can potentially give a different view of how the genetic variation is oriented. By simultaneously considering all three, one arrives at a fuller understanding of $\mathbf{g_{max}}$. Likewise, by studying sexual dimorphism in multiple dimensions, one might conclude that sexual dimorphism might not evolve in one particular trait but may evolve when considering the entire phenotype. Increasing the number of traits will likely increase the apparent number of differences between males and females, but prudent trait selection aimed at sensible hypothesis testing remains the standard. Comparing the selection gradients $\beta_{\mathbf{m}}$ and $\beta_{\mathbf{f}}$ should suggest which traits are among the more interesting to examine.

Sexual dimorphism in **G** occurs through sex-specific differences in genetic variances and covariances. As with sex-specific heritabilities, sexual dimorphism in dominance, allele frequencies and additive effects may contribute to sex-limited patterns of gene expression and pleiotropy to alter eigenstructures between the sexes.

Several studies have shown that sexual dimorphism is common in sex-specific **G** matrices (e.g. Holloway *et al.*, 1993; Guntrip *et al.*, 1997; Ashman, 2003; Jensen *et al.*, 2003; Rolff *et al.*, 2005; McGuigan & Blows, 2007; Sakai *et al.*, 2007; Steven *et al.*, 2007; Campbell *et al.*, 2010; Dmitriew *et al.*, 2010; Lewis *et al.*, 2011) (see also Steven *et al.*, 2007; Barker *et al.*, 2010 for further discussion). When the Flury hierarchical testing procedure is applied, by and large, **G** matrices across populations, experimental treatments, species and sexes share some subset of eigenvectors (Arnold *et al.*, 2008) suggesting conservation. In particular, the sexes appear to share all or some principle components in 78% of comparisons (Arnold *et al.*, 2008). For instance, **G** matrices between females and hermaphrodites of the gynodioecious plant species, *Fragaria virginiana* (Ashman, 2003) and *Schiedea salicaria* (Campbell *et al.*, 2010), shared all eigenvectors but not eigenvalues so that they differed in shape but not orientation.

Conservation of eigenvectors means that the direction along which males and females may respond to selection is the same. In either the full or partial CPC case, if eigenvalues differ for shared eigenvectors, the total response to identical selection will differ in males versus females along that shared direction – as in the univariate case. If the eigenvectors are completely shared, then comparing univariate variances would be sufficient. However, even in the full CPC case, a multivariate approach will provide new insights through the **B** matrix (see more below). If the eigenvectors are partially shared, then the multivariate approach has revealed potentially interesting multidimensional trait axes, so that responses to selection will be different between the sexes because of the distinct eigenvectors.

Despite the widespread conservation of eigenvectors, comparisons of **G** between the sexes also revealed that they are never equal (same eigenvectors and same eigenvalues) or proportional (same eigenvectors but with proportional eigenvalues) (Arnold *et al.*, 2008) – in contrast to comparisons of **G** between experimental treatments, populations, or species (Arnold *et al.*, 2008). Moreover, distinct, unrelated eigenstructures are by no means uncommon, representing 22% of between-sex comparisons (Arnold *et al.*, 2008). For instance, male and female **G** matrices were completely unrelated in the plant *Silene latifolia* (Steven *et al.*, 2007) and the house sparrow *Passer domesticus* (Jensen *et al.*, 2003). Sex-specific **G** matrices that do not share any eigenvectors mean that the response to selection may proceed along completely different axes in each sex so that changes in sexual dimorphism will occur, but due to the evolution of different trait sets in each sex (depending on the strength and direction of $\boldsymbol{\beta}$). And although the Flury hierarchical approach tends to overestimate matrix differences (e.g. Houle *et al.*, 2002; Mezey & Houle, 2003), there is no reason why this issue should afflict sex-based comparisons of the **G** matrix more than comparisons across environments or experimental treatments.

Because full rank **G** matrices have as many eigenvalues as there are number of traits measured, it is important to characterize the eigenvalues – for example, how many of the eigenvalues describe a large or statistically significant proportion of the total variance? The number of statistically supported eigenvalues will indicate the number of directions that evolution may proceed along in multivariate space. Recent studies suggest that many **G** matrices seem to be ill-conditioned, such that most of the genetic variance is explained by the first 1 or 2 eigenvalues (Kirkpatrick, 2009; Walsh & Blows, 2009; Simonsen & Stinchcombe, 2010) – although determining the contribution of sampling biases to these results (e.g. Hill & Thompson, 1978; Hayes & Hill, 1981) is an ongoing challenge. Thus, although many traits may be measured, the response to selection may only proceed along 1 or 2 dimensions. However, in terms of the evolution of sexual dimorphism, it may not be necessary for sex-specific differences to proceed along all potential directions. Sexual dimorphism can evolve because males and females can differ with regard to the effective number of dimensions, the amount of genetic variance explained, the orientations and/or the magnitudes. For example, McGuigan & Blows (2007) compared male and female differences in genetic dimensions and found that females possessed a greater number of effective dimensions than males in *Drosophila bunnanda*. In *Drosophila serrata*, males and females diverged in different sets of multivariate axes with respect to population divergence (Chenoweth & Blows, 2008). Although empirical studies may underestimate the true dimensionality of **G**

matrices, there is no *a priori* reason why dimensionality should be consistently over- or under-estimated in one sex relative to the other.

B matrix and r_{MF}

In addition to the sex-specific **G** matrices, the intersexual covariance **B** matrix is important to understanding the evolution of sexual dimorphism. Because **B** keeps track of additional genetic covariances across different traits in each of the sexes, it will typically modify the predicted response to selection provided by the univariate quantity r_{MF} alone.

The multivariate **B** matrix is furthermore interesting because positive intersexual covariances in the matrix may either help or hinder positive, male-biased changes in sexual dimorphism (i.e. $\Delta SD > 0$ because $\beta_m - \beta_f$). By contrast, in the univariate breeder's equation, positive intersexual covariances are always a constraint on positive changes in sexual dimorphism. It is possible to see this by applying equation 8 to a simple two trait example. Consider here the homologous characters K and L, each measured in males (m) and females (f). The response to selection for sexual dimorphism in the shared homologous trait K evolves as:

$$\Delta SD_K = \frac{1}{2}\{V_{A(K^m)}\beta_{K^m} + \mathrm{Cov}_{A(K^m,L^m)}\beta_{L^m} \quad (10a)$$

$$-V_{A(K^f)}\beta_{K^f} - \mathrm{Cov}_{(K^f,L^f)}\beta_{L^f} \quad (10b)$$

$$-\mathrm{Cov}_{A(K^m,K^f)}[\beta_{K^m} - \beta_{K^f}] \quad (10c)$$

$$-\mathrm{Cov}_{A(K^f,L^m)}\beta_{L^m} + \mathrm{Cov}_{A(K^m,L^f)}\beta_{L^f}\}. \quad (10d)$$

The response in sexual dimorphism for trait K (ΔSD_K) is very complicated even in this two trait example. For the moment setting aside the $\beta_{trait^{sex}}$ terms, the equation consists of the following: the sex-specific genetic variances ($V_{A(K^m)}$, $V_{A(K^f)}$); the cross-trait, within-sex genetic covariances ($\mathrm{Cov}_{A(K^m,L^m)}$, $\mathrm{Cov}_{A(K^f,L^f)}$); the within-trait cross-sex genetic covariances ($\mathrm{Cov}_{A(K^m,K^f)}$); and the cross-trait cross-sex genetic covariances ($\mathrm{Cov}_{A(K^m,L^f)}$, $\mathrm{Cov}_{A(K^f,L^m)}$). The negative sign of term 10c means that when $\mathrm{Cov}_{A(K^m,K^f)}$ is strongly positive, it will decrease positive changes in sexual dimorphism predicted by the within-sex variances and within-sex covariances (10a–10b) – as in the univariate breeder's equation. When $\mathrm{Cov}_{A(K^m,K^f)}$ is strongly negative, it will increase the positive changes in sexual dimorphism.

Because equation 10 describes two traits, it has additional intersexual covariance terms (10d) that are not present in the univariate breeder's equation. Interestingly, these additional intersexual covariances can have a negative or positive sign, which will, respectively, hinder or facilitate positive, male-biased changes in sexual dimorphism for trait K (i.e. $\Delta SD_K > 0$ because $\beta_{K^m} - \beta_{K^f} > 0$). By contrast, positive covariances may only hinder positive changes in sexual dimorphism in the breeder's equation. Finally, the difference between the last two intersexual covariance terms (10d) is a measure of how asymmetric the **B** matrix is. If these two terms are very different, **B** can affect one sex more strongly than the other.

Equation 10 demonstrates the implications of an asymmetrical **B** on single traits. However, **B**'s multivariable impact may also be considered by analysing its orientation with respect to the sex-specific selection vectors, $\beta_{\mathbf{m}}$ and $\beta_{\mathbf{f}}$. Both Lewis et al. (2011) and Gosden *et al.* (2012) show that including **B** reorients the response to selection away from the direction of selection more for females than males. Also, including **B** tends to decrease the degree of sexual dimorphism expected by r_{MF} alone. It remains to be seen whether these are general patterns. Furthermore, it is as yet unclear whether sex-specific selection is properly aligned with **B**'s asymmetry to increase changes in total male-biased sexual dimorphism (i.e. $\mathbf{B}\beta_{\mathbf{f}} - \mathbf{B}^{\mathbf{T}}\beta_{\mathbf{m}} > 0$ in equation 8).

Several studies have reported the complete **B** intersexual covariance matrix (Meagher, 1999; Steven *et al.*, 2007; Campbell *et al.*, 2010; Lewis *et al.*, 2011; Gosden *et al.*, 2012). In general, it appears that $\mathbf{B} \neq \mathbf{B}^{\mathbf{T}}$ according to point estimates from the matrix. For example, in Lewis *et al.* (2011), male longevity has a positive covariance with female size, but female longevity has a negative covariance with male size in *Plodia interpunctella*. The asymmetry of **B** was apparent in a study of seven cuticular hydrocarbons in *Drosophila serrata* by demonstrating that the above- and below- diagonal elements of **B** had a smaller correlation than a similar comparison between the $\mathbf{G_m}$ and $\mathbf{G_f}$ matrices (Gosden *et al.*, 2012). The remaining studies of **B** in plants – *Silene latifolia* (Meagher, 1999; Steven *et al.*, 2007) and *Schiedea adamantis* (Campbell *et al.*, 2010) – also support differences in the male-to-female versus female-to-male patterns of covariation among traits. In *Silene latifolia*, male leaf length and female calyx width had a positive covariance, but female leaf length and male calyx width had a negative covariance (Steven *et al.*, 2007). In *Schiedea adamantis*, the off-diagonal covariances are all positive, but with the size of the covariances differing greatly below and above **B**'s diagonal; this difference caused the genetic correlation between female terminal capsule weight and hermaphrodite terminal carpel weight to be twice the genetic correlation in the converse direction. Formal hypothesis tests for determining asymmetry between individual elements of **B** or for the entire matrix await further development.

Factors such as genomic imprinting and sex-limited expression may play a role in creating asymmetries in **B**. It may also be that asymmetries actually point to the prior efficacy of sex-specific selection in producing sexual dimorphism. Correlational selection can cause the **G** matrix to point in the same direction of the selection

as it accumulates mutations oriented in this same direction (Roff & Fairbairn, 2012), making the direction of $\mathbf{g_{max}}$ nonrandom with respect to the direction of selection (Schluter, 1996). In a similar manner, the pattern of intersexual covariances may have been altered to accommodate sex-specific selection (Barker *et al.*, 2010; Delph *et al.*, 2011).

Putting G, B, and β together

Sex-specific selection is typically invoked as the cause of sex-specific differences (Darwin, 1874; Andersson, 1994). We have pointed out two key aspects of sex-specific genetic architectures that further deserve attention when studying the evolution of sexual dimorphism – sex-specific **G** matrices and the intersexual genetic covariance **B** matrix. Taking seriously Lande's equation (7) for the evolution of sexual dimorphism means not just focusing upon selection. Strong differences between $\mathbf{G_m}$ and $\mathbf{G_f}$, or between the upper and lower elements of **B**, or between $\beta_\mathbf{m}$ and $\beta_\mathbf{f}$ can all contribute to large changes in the extent of sexual dimorphism.

For example, although marked differences between $\mathbf{G_m}$ and $\mathbf{G_f}$ can predict a large degree of phenotypic sexual dimorphism, it is only one part of the whole picture. Two closely related plants, *Schiedea adamantis* and *Schiedea salicaria*, have been studied for $\mathbf{G_m}$ and $\mathbf{G_f}$ differences. Both species are gynodioecious (possessing hermaphrodite and female individuals), but *S. adamantis* has a higher proportion of females than *S. salicaria*. Interestingly, sexes of the less sexually dimorphic species, *S. salicaria*, shared no principal components (Campbell *et al.*, 2010), whereas sexes of the more dimorphic species, *S. adamantis*, shared all principal components (Sakai *et al.*, 2007). Furthermore, Campbell *et al.* (2010) found no evidence that the intersexual genetic correlations for homologous traits were lower in the more sexually dimorphic species compared with the less dimorphic species (Campbell *et al.*, 2010), suggesting that the between-sex imposed constraints were not fundamentally different between the two species. As a result, the greater sexual dimorphism in *S. salicaria* may have occurred through eigenvalue differences between $\mathbf{G_m}$ and $\mathbf{G_f}$, or through the greater asymmetry of **B**. Alternatively, sex-specific selection may have been altogether absent or not aligned with the sex-specific **G** and/or **B** matrices in *S. salicaria* resulting in less sexual dimorphism. The work in *Schiedea* illustrates how understanding the components of equation 7 can elucidate the factors that permitted sexual dimorphism to evolve.

Discussion

Biologists have long wondered how sexual dimorphism might arise given that males and females share a common genetic architecture that ought to impede phenotypic divergence. Examining the univariate and multivariate equations suggests that sexual divergence may not be so difficult to understand. This facility is provided, in part, by considering the change in perspective provided by the multivariate approach.

Sex-specific genetic variances

Multivariate differences in genetic variances between the sexes can be more apparent than univariate differences due to the summary nature of multivariate data. Single trait studies indicate that mean male and mean female heritabilities are not sexually dimorphic overall (Wyman and Rowe, *unpublished results*). By contrast, most studies that have measured sex-specific **G** matrices demonstrate that they are dimorphic (Holloway *et al.*, 1993; Guntrip *et al.*, 1997; Ashman, 2003; Jensen *et al.*, 2003; Rolff *et al.*, 2005; McGuigan & Blows, 2007; Sakai *et al.*, 2007; Steven *et al.*, 2007; Dmitriew *et al.*, 2010; Campbell. Such et al. 2010; Lewis *et al.*, 2011). Such differences are due to the fact that the multivariate formulation can take into account both the size of the genetic variance and how it is oriented between the sexes (Fig. 1). Previous authors have pointed out that $\mathbf{g_{max}}$ will always explain more variance than any single univariate genetic variance by default (except when all covariances of **G** are zero) (Mercer & Mercer, 2000; Kruuk & Garant, 2007; Chenoweth & Blows, 2008). So, the multivariate perspective necessarily provides more potential for change in sexual dimorphism than the univariate perspective. However, contrasting the magnitude and orientation of $\mathbf{G_m}$ versus $\mathbf{G_f}$ and their effective number of dimensions will further accentuate the differences observed between males and females under a multivariate view compared with a univariate one (beyond the default expectation pointed out by Mercer & Mercer (2000)). Consequently, studying the evolution of sexual dimorphism in multiple traits can potentially yield a picture of fewer constraints when compared to studying the evolution of multiple traits without regard for sex.

Finally, although sex-specific genetic variances are important, a particular univariate V_A or multivariate **G** matrix may also evolve. Selection may alter genetic and phenotypic variances, thereby changing the course of the evolution of sexual dimorphism. For instance, male sexually selected traits oftentimes experience strong directional selection, whereas homologous female traits are more likely to experience stabilizing selection (Pomiankowski & Moller, 1995; Rowe & Houle, 1996). Strong directional selection can briefly increase the variance if the frequencies of rare alleles rise (Barton & Turelli, 1987; Blows & Higgie, 2003). Yet, sustained selection (directional or stabilizing) is expected to deplete genetic variation. Both scenarios may alter the genetic variance in males. Conversely, if stabilizing selection is stronger in females relative to males, female

variances should decrease (e.g. Rundle & Chenoweth, 2011). The stability of the **G** matrix is an open question and the factors supporting the long-term permanence of **G** are not well understood (Arnold *et al.*, 2008). In addition, theoretical work suggests that whereas variance–covariance structures can determine the short-term responses to selection, long-term responses rely only upon the type of selection (e.g. stabilizing versus directional) (Zeng, 1998). Understanding the relative stability and importance of $\mathbf{G_m}$, $\mathbf{G_f}$, $\beta_\mathbf{m}$, and $\beta_\mathbf{f}$ will be important to the study of multivariate sexual dimorphism.

Intersexual covariances

Considering only the univariate quantity r_{MF} can give a misleading picture of trait evolution between the sexes for two reasons. First, r_{MF} is subject to issues of standardization. The correlation may be small when the intersexual covariance is small, or when the sex-specific genetic variances are large, or both. Second, r_{MF} misses all of the additional cross-trait, intersexual covariance terms. As a result, in the univariate formulation, positive intersexual covariances may only decrease the response to selection for positive changes in sexual dimorphism. By contrast, the multivariate formulation is more general, and the intersexual covariance between one trait in males and a different trait in females is included. These extra intersexual covariance terms can either constrain or facilitate positive changes in sexual dimorphism – beyond those predicted by r_{MF} alone. Again, the multivariate formulation provides an additional avenue for male–female differences to evolve that is absent in the univariate formulation. So far, empirical inclusion of **B** substantially decreases the predicted phenotypic divergence between males and females (Lewis *et al.*, 2011; Gosden *et al.*, 2012). More studies will be required to understand whether the effects captured by **B** can actually increase phenotypic divergence between the sexes.

The current evidence suggests that **B** is often asymmetric. Recent experiments demonstrate that the intersexual covariances themselves can change due to selection (Delph *et al.*, 2011). Furthermore, theory and data suggest that the **B** matrix may be intrinsically more pliable than the **G** matrix. **B** matrices among populations are more variable in eigenstructure compared with **G** matrices (Barker *et al.*, 2010). The **B** matrix can change more easily than the **G** matrix because the values in **B** are not realized in any particular individual and thus are less strongly affected by stabilizing and/or correlational selection (Barker *et al.*, 2010). Among population variation in **B** is intriguing and may suggest that **B** matrices can potentially evolve to reverse its stronger impact on one sex compared with the other. We require further empirical work on **B**'s alignment with respect to sex-specific selection to demonstrate these possibilities.

Diversity in sexual dimorphism

In addition to differences in sex-specific selection, the presence of dimorphism in **G** and asymmetry in **B** may account for widespread dimorphism among animal and plant taxa in a multivariate context. No matter how long it took to evolve dimorphic **G** matrices and an asymmetric **B** matrix, once in place, it may be easy to introduce further sexual dimorphism afterwards. Moreover, the data from Arnold *et al.* (2008) suggest that eigenvalue differences may be more important than eigenvector differences (because eigenvectors are relatively conserved between the sexes). Such a genetic architecture may account for how homologous ornaments across taxa can vary so widely – for example dung beetle horns, cervid antlers, butterfly colours, bird plumage, etc. It may also explain why dimorphism exhibits so much phylogenetic lability (e.g. Price & Birch, 1996; Burns, 1998; Amundsen, 2000; Coyne *et al.*, 2008; Oliver & Monteiro, 2011) over relatively short phylogenetic timescales. Sexual selection may be constantly reshaping characters that already differ between males and females with respect to eigenvalues because those are precisely the traits that represent the path of least genetic resistance.

The apparent paradox of widespread sexual dimorphism in the face of a constraining genetic architecture is partly resolved by considering multivariate trait combinations. However, it will ultimately be difficult to assess how much sexual dimorphism failed to evolve within lineages, even though selection was favoring it. Consequently, it will be challenging to detect cases where the evolution of sexual dimorphism was thwarted due to the relative orientations of $\mathbf{G_m}$, $\mathbf{G_f}$, **B**, $\beta_\mathbf{m}$ and $\beta_\mathbf{f}$. Moreover, the quantitative genetics equations are best for short-term predictions, as it is unclear how stable variances and covariances may be over longer time periods or how their relative importances shift. Developing hypotheses about phylogenetic or macroevolutionary trends in sexual dimorphism based on current estimates of quantitative genetic variation will likely be challenging.

Conclusions

Although it is reasonable to assume that sexual dimorphism in traits is hard to evolve, this is not an underlying feature of the equations describing their evolution. Multivariate sexual dimorphism is potentially easier to evolve than univariate sexual dimorphism, perhaps reconciling the fact that sexual differences are nearly universal despite the widespread prevalence of very high intersexual genetic correlations. Additional work on sex-specific selection, sex-specific **G** matrices and the **B** matrix will enable us to dissect the forces underlying the evolution of sexual dimorphism and arrive at a fuller understanding of how present differences have come to be.

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