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# Male Bias in Distributions of Additive Genetic, Residual, and Phenotypic Variances of Shared Traits

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**ABSTRACT:** Despite a shared genetic architecture between males and females, sexual differences are widespread. The extent of this shared genetic architecture, reflected in the intersexual genetic correlation, has previously been correlated with the extent of phenotypic sexual dimorphism in shared traits. However, the magnitude of the difference in sex-specific additive genetic variances may also fuel sexual dimorphism. To explore the correlation between additive genetic variance dimorphism and phenotypic dimorphism, we conducted a literature search. We targeted traits expressed in both sexes and excluded sex-limited traits. The mean difference between the sexes in additive genetic variance was not significantly different from 0. However, the distribution of the sexual difference in additive genetic variance had a significant male-biased skew. This pattern persists even after removing traits explicitly related to reproduction. Furthermore, male traits had more residual and phenotypic variance than homologous female traits (as measured by both the mean and the skew), and this difference was not necessarily due to the difference between sexual traits and nonsexual traits. We found no evidence that sex chromosome system could explain sex differences in additive genetic, nonadditive genetic, or phenotypic variances. Finally, we found a significant correlation between the extent of sexual dimorphism in additive genetic variances and the extent of phenotypic sexual dimorphism. Understanding why traits have sex-specific patterns of variation awaits further investigation.

**Keywords:** sexual dimorphism, intersexual genetic correlation, sexual conflict, sexual antagonism.

## Introduction

Sexual phenotypic differences are often attributed to sex-specific differences in selection (Darwin 1871; Andersson 1994). However, because males and females share most of the same genes, selection on one sex may produce a correlated response in the opposite sex, inhibiting phenotypic divergence (Lande 1980). Adaptive sexual dimorphism evolves in the presence of an accommodating genetic architecture that provides free additive genetic variance ( $V_A$ )

for sex-specific selection to act upon. Additive genetic variance for sexual dimorphism exists through a low intersexual genetic covariance between the sexes, through sexual dimorphism in male- and female-specific additive genetic variances, or both (Lande 1980; Lynch and Walsh 1998).

The importance of a low intersexual genetic covariance to the extent of phenotypic sexual dimorphism has been demonstrated previously using a standardized quantity, the intersexual genetic correlation ( $r_{MF}$ ; e.g., Bonduriansky and Rowe 2005; Poissant et al. 2009). The term  $r_{MF}$  is the intersexual genetic covariance divided by the square root of the product of the male  $V_A$  and the female  $V_A$ . Empirical data have demonstrated that  $r_{MF}$  is negatively correlated with the extent of sexual dimorphism, so that high values are associated with low phenotypic divergence and low values are associated with high phenotypic divergence across a variety of traits and taxa (Poissant et al. 2009).

By contrast, the importance of sexual dimorphism in additive genetic variance to the evolution of phenotypic sexual dimorphism has not been analyzed in detail. Heritabilities can differ between males and females (e.g., Holloway et al. 1993; Jensen et al. 2003; Rolff et al. 2005). The **G** matrix (multivariate variance-covariance matrix) can also differ between males and females—either in individual values or in the geometry of the matrix (e.g., Holloway et al. 1993; Guntrip et al. 1997; Ashman 2003; Jensen et al. 2003; Rolff et al. 2005; McGuigan and Blows 2007; Sakai et al. 2007; Steven et al. 2007; Dmitriew et al. 2010; Campbell et al. 2011; Lewis et al. 2011). It is unclear whether such differences in  $V_A$  are common and how they relate to the degree of phenotypic sexual dimorphism.

Sexual dimorphism in additive genetic variances can arise through sex-specific differences in long- and short-term factors. In the long term, sexually dimorphic additive genetic variances may arise from sex-specific differences in selection. Females are expected to experience stabilizing natural selection on most traits while males are expected to experience directional selection on mating-related traits

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(Rowe and Houle 1996). Stabilizing selection can reduce the additive genetic variance while directional selection can increase or decrease the additive genetic variance (Barton and Turelli 1987; Blows and Higgie 2003). Mating traits under strong directional selection may also adopt the genetic variance of condition, ultimately increasing their additive genetic variance (Rowe and Houle 1996). Concordant with this process, male sexual traits have more additive genetic variance than nonsexual traits (Pomiankowski and Møller 1995; Rowe and Houle 1996). However, it is unclear whether shared homologous traits with a role in reproduction have more sexual dimorphism in  $V_A$  than shared traits that do not.

For a one-locus, two-allele model,  $V_A$  depends on the allele frequencies, the genotypic value  $a$  of the homozygotes, and the dominance deviation  $d$  of the heterozygote from the mean of the two homozygotes (Falconer and Mackay 1996). In the short term, sex-specific  $V_A$  can arise from differences in these factors. For example, sex differences in allele frequencies can occur, most obviously through sex linkage. At a sex-linked locus, the homogametic sex can be homozygous or heterozygous, whereas the heterogametic sex is hemizygous. Under certain assumptions, the heterogametic sex will have a more additive genetic variance than the homogametic sex (Lynch and Walsh 1998; Reinhold and Engqvist 2013). Alternatively, homologous traits between males and females may express distinct alleles or genes through sexual antagonism (Chipindale et al. 2001) or by experiencing distinct mutational effects (Mallet et al. 2011; Sharp and Agrawal 2012), which will affect  $a$ . Finally, alleles may have sex-specific dominance effects (Fry 2010), leading to a difference in  $d$  and, thus, in sex-specific  $V_A$  (Falconer and Mackay 1996).

The sexes may also differ with regard to nonadditive sources of variance due to sexual differences in dominance, epistatic, and/or environmental variance. Male sexual traits have elevated amounts of phenotypic variance compared to nonsexual traits that are expressed in either females or males (Darwin 1871; Pomiankowski and Møller 1995). However, it is unknown whether homologous shared traits differ with regard to residual and total phenotypic variance. It is also unclear whether this difference relates to the difference between traits related to and not related to reproduction.

In this article, we asked four questions about sex differences in variability. First, are male- and female-specific additive genetic variances systematically different? We addressed this question by comparing levels of additive genetic variance in homologous male and female traits. We also compared differences in additive genetic variance that could be due to differences between reproductive and nonreproductive traits. Second, are male and female phenotypic and nonadditive genetic variances systematically dif-

ferent, and could such differences also be associated with the difference between reproductive and nonreproductive traits? Third, to what extent is phenotypic sexual dimorphism across traits correlated with sexual dimorphism in additive genetic variances? Finally, is sex chromosome system associated with differences in additive genetic variance? We discuss how the sexes might have evolved differences in additive, residual, and phenotypic variance.

## Methods

We conducted a literature search (in September 2011) to compile estimates of male and female additive genetic variances. Our search consisted of three parts. First, we searched for “(male\* OR female\* OR dimorph\* OR monomorph\*)” in ISI Web of Science and targeted journals of interest to evolutionary biologists: *Evolution*, *Journal of Evolutionary Biology*, *Proceedings of the Royal Society B: Biological Sciences*, *Heredity*, *Nature*, *Science*, *American Naturalist*, *Evolutionary Ecology*, *Evolutionary Ecology Research*, *Biology Letters*, *PLoS Biology*, and *Current Biology*. Second, we searched for “(heritability\* AND sex\*)” to retrieve heritability estimates from a wider variety of journals. We ignored any etiological hits in the animal breeding or human medicine literature. Last, we searched the literature cited sections of review articles that have compiled additive genetic variance estimates (Ashman 2003; Ashman and Majetic 2006; Poissant et al. 2009; Hansen et al. 2011).

We collected additive genetic variances (or sire-related variance), narrow-sense heritabilities ( $h^2$ ), and/or coefficients of additive genetic variance ( $CV_A$ ) for traits expressed in both males and females. Narrow-sense heritability was calculated as  $V_A/V_p$ , where  $V_p$  is phenotypic variance, and  $CV_A$  was calculated as  $100 \times (V_A^{1/2}/\mu)$ , where  $\mu$  is the trait mean (Houle 1992). If an article reported only  $h^2$  and  $V_p$ , we could calculate  $V_A$ . If the article did not report  $V_p$  but did include the observed phenotypic standard deviation (SD; or the standard error and sample size) of the raw data, we could calculate  $V_A$ . Because we were interested in sex-specific differences in variance, we did not include any sex-limited traits such as male-limited sexually selected traits. However, we did include a male sexually selected trait if a clear homologue existed in the female, for example, male tail streamers and female tail feathers in barn swallows. We did not include measures of fecundity (i.e., egg counts, offspring number). We did include traits such as age of first reproduction or copulation duration. For additional considerations concerning the collection of variance data, see the appendix, available online.

We also collected data to calculate the coefficients of phenotypic and residual variance. The coefficient of phenotypic variance is  $CV_p = 100 \times (V_p^{1/2}/\mu)$ . The coefficient of residual (or nonadditive genetic) variance is  $CV_R =$

$100 \times (V_R^{1/2}/\mu)$ , where the residual genetic variance is  $V_R = V_P - V_A$ .

We compiled male and female trait means to determine the degree of phenotypic sexual dimorphism. Some studies provided only a ratio of size as measured by the Lovich-Gibbons index (Lovich and Gibbons 1992). To include these values in our study we ensured that the male measurement was always in the numerator and the female measurement in the denominator and that the value 1 was not subtracted from the ratio. We did not use the Lovich-Gibbons index per se since we log transformed the ratio in our analyses. The data for all analyses have been deposited in the Dryad Digital Repository: <http://dx.doi.org/10.5061/dryad.43j02> (Wyman and Rowe 2014).

### *Metrics, Hypotheses, and Statistical Analyses*

Sexual dimorphism in additive genetic variances can be measured as the male – female difference in  $CV_A$  and  $h^2$ . We analyzed the distribution of  $CV_A$  and  $h^2$  differences between the sexes by studying means, skews, and kurtoses. If male variances are systematically larger, we predicted that there should be a significant positive (male-biased) mean in the distribution of the male  $CV_A$  – female  $CV_A$  and male  $h^2$  – female  $h^2$  differences. By contrast, if female variances are systematically larger, there should be a significant negative (female-biased) mean. Sex bias in the distribution of male  $CV_A$  – female  $CV_A$  or male  $h^2$  – female  $h^2$  might also be indicated through a significant skew. A skew indicates whether a distribution has a longer or fatter tail (due to the presence of more extreme values) on one side of the mean. Biologically speaking, a positive skew indicates that extreme values are male biased and a negative skew that extreme values are female biased. Finally, we also examined kurtosis to further characterize departures from normality. Under a normal distribution, kurtosis is expected to equal 0 so that the distribution is not too pointy or too broad in the middle. Significance tests for skew and kurtosis were conducted as recommended in Crawley (2007). All estimates are reported  $\pm 1$  SE unless otherwise noted.

For differences between or among means, we used two basic approaches. First, we asked whether the mean of the male – female distribution was significantly different from 0. We conducted a one-sample *t*-test on the male – female difference (in  $CV_A$  or  $h^2$ ) to test for a mean difference from 0. We also took a nonparametric approach and randomized the original data to generate a distribution of new mean male – female differences in  $CV_A$  and  $h^2$ . Both the  $CV_A$  and  $h^2$  data sets have a great deal of structure (e.g., each estimate is with respect to a specific trait measured in a specific organism). We preserved this structure by taking the original paired male and female data and ran-

domly making the pair's difference positive or negative, thereby randomizing which value is "male" and which is "female." We could then create a new data set over which to calculate a new mean of the male – female difference. We repeated this process 9,999 times and included the original mean to generate a distribution of new means. We noted where the absolute value of the observed mean fell in the randomized distribution and considered it to be significant if it exceeded 95% of the absolute values of the randomized means (two-tailed test). Because the parametric and nonparametric tests largely agreed, we report only the results from the randomization tests (except when the tests disagreed). We repeated the same sets of analyses on the male  $CV_P$  – female  $CV_P$  and on the male  $CV_R$  – female  $CV_R$  to examine sexual dimorphism in phenotypic and residual variances.

Second, we asked whether means between or among categories of traits or organisms differed in additive genetic variance (see appendix). We used both parametric and nonparametric one-way ANOVAs. When the assumption of normality was violated, we used the R package *lmPerm* (Wheeler 2010) to conduct permutation tests to compare groups with the command *aovp()*. The permutation test implements a one-way ANOVA by randomly reassigning the observed data values to each of the groups being compared and calculating the *F* statistic for each iteration. The actual *F* is then compared to the distribution of permuted *F* statistics to see what percentage of permuted *F* values exceed the observed *F* value. Because of their general concordance, we report only the results of the nonparametric tests, except when their results differed from the parametric tests.

Next, we predicted that the amount of dimorphism in  $V_A$  should correlate positively with the amount of dimorphism in sex-specific phenotypic trait means. We measured the correlation between the log(base 2) of the male : female ratio in phenotypic means and the log(base 2) of the male : female ratio in additive genetic variances. The ratio of means and the ratio of variances are both dimensionless numbers, making it possible to compare a wide variety of trait types. We also used a mixed model to account for the significant structure of the data set and the phylogenetic nonindependence of the measurements. We entered log(male  $V_A$ /female  $V_A$ ) as the dependent variable, log(male size/female size) as a covariate, and trait type nested within species as a random factor.

It is possible to weight measurements with the sample size or the variance of the reported estimates. When the sample size (*n*) was available, we performed a bias correction by multiplying the factor  $[1 + (1/4n)]$  and the coefficients of variance (i.e.,  $CV_A$ ,  $CV_P$ , or  $CV_R$ ; Sokal and Rohlf 1995; Reinhold and Engqvist 2013). However, we did not weight our CV (additive, residual, or phenotypic)

or  $h^2$  data by the variance or the standard error for practical reasons. Our main metric was the sexual difference in the CV. Calculating its error would require the standard error of the sex-specific CVs and the covariance between the male CV and the female CV, neither of which was reported in any study. The standard error of heritability was reported in 88 out of 113 studies, and the standard error of additive genetic variance was reported in 19 out of 57 studies.

Finally, we tested for differences in sexual dimorphism in additive genetic variance, residual variance, and phenotypic variance due to sex linkage. In a two-allele sex-linked locus model, the homogametic sex can be homozygous or heterozygous at that locus. By contrast, the heterogametic sex can express only one of the two alleles. As a result, the heterogametic sex will have a greater additive genetic variance on average compared to the homogametic sex under a certain set of assumptions (Lynch and Walsh 1998, p. 715; Engqvist and Reinhold 2013). Thus, in general, XY systems should have greater male-specific additive genetic variance, whereas ZW systems should have greater female-specific additive genetic variance. Since sex chromosome information was not available for all organisms, we proceeded under the justified assumption that certain organismal groups are male heterogametic (e.g., mammals, flies, beetles) while others are female heterogametic (e.g., butterflies, moths, birds). We ignored fish and plants since their sex chromosomes are not as well characterized and show sex chromosome reversals.

## Results

### *Characteristics of the Data*

We found 149 articles that provided male and female estimates of coefficients of additive genetic variance and/or heritabilities. We removed duplicated traits within the same species (see appendix for more details). We had 279 pairs of male and female  $CV_A$  estimates and 508 pairs of male and female  $h^2$  estimates from 122 studies representing 75 species. Table A3 (tables A1–A3 available online) details the references and the types of information that were available for each organism. The vast majority of the paired estimates (241  $CV_A$  and 430  $h^2$ ) came from controlled laboratory studies using wild-caught founders or long-term laboratory lines. There were fewer  $CV_A$  than  $h^2$  pairs because many studies did not report enough information to calculate  $CV_A$  in both males and females. In addition, there were many studies for which it is possible to calculate  $h^2$  on residual, PC, binary, or nominal data but for which calculating  $CV_A$  would be invalid (appendix).

Among the studies that we found, only a handful ex-

PLICITLY analyzed differences in additive genetic variance between the sexes. Of the 21 studies that statistically tested for male and female differences in heritabilities, 12 found significant differences in a subset of the traits examined (Mousseau and Roff 1989; Wilcockson et al. 1995; Ashman 1999, 2003; Mignon-Grasteau 1999; Jensen et al. 2003; Rolff et al. 2005; Ng et al. 2006; Fedorka et al. 2007; Zillikens et al. 2008; Gershman et al. 2010; Stillwell and Davidowitz 2010). Of the seven studies that tested for differences in unstandardized additive genetic variances, three found statistical difference between the sexes (Towne et al. 1992; Rolff et al. 2005; Zillikens et al. 2008). To increase our sample size and take advantage of all of the data, we decided to use all of the heritabilities and coefficients of variance reported in the 122 studies, whether the estimates were reported as significantly different from 0 or whether the sexes were significantly sexually dimorphic.

Most of the data came from insects and vertebrates. Among the four plant species, one species was dioecious, and three species were gynodioecious (female and hermaphrodite flowers). We designated the hermaphrodite measurements as “male” (55  $CV_A$  pairs, 60  $h^2$  pairs). Dropping the hermaphrodite measurements did not qualitatively alter our results for the entire data set. Gynodioecious species might have less sexual dimorphism in  $V_A$  because hermaphrodite flowers are less “male” and experience more similar selective pressures as female flowers compared to dioecious species. Thus, including hermaphrodites is conservative with respect to our hypotheses.

### *Sexual Dimorphism in Additive Genetic Variance*

*Entire Data Set.* The mean of the male  $CV_A$  – female  $CV_A$  was positive (male biased) across the entire data set; however, the mean was not significantly different from 0 (table 1). The mean of the male  $h^2$  – female  $h^2$  was negative (female biased) and not significantly different from 0.

Normal distributions have a skew of 0. We tested for departures from a skew of 0 in the male – female distributions of  $CV_A$  and  $h^2$ . There was a significant right skew in the entire  $CV_A$  data set, indicating that extreme  $CV_A$  was more common in males than in females. Upon closer inspection, we removed three outliers with extreme  $CV_A$  differences (e.g., two male – female differences less than –40 and one difference greater than 100). We felt justified in this removal because the outliers were 8 and 33 SD away from the mean for the culled data set. After outlier removal, all of the data fell within equally sized ranges on either side of 0 (e.g., –25 to 25; fig. 1). The resulting distribution of both the  $CV_A$  and  $h^2$  difference remained significantly right skewed (table 2) so outlier removal did not alter the direction of the skew. These

**Table 1:** Mean of the sexual difference in additive genetic variances

	Male $CV_A$ – female $CV_A$		Male $h^2$ – female $h^2$	
	Skew $\pm$ 1 SE (N)	P value	Skew $\pm$ 1 SE (N)	P value
Combined data	.410 $\pm$ .320 (279)	.206	–.007 $\pm$ .0089 (508)	.406
Reproduction	1.539 $\pm$ 1.324 (43)	.263	.086 $\pm$ .035 (68)	.001*
Not reproduction	.204 $\pm$ .292 (236)	.480	–.022 $\pm$ .009 (440)	.014*

Note: The mean was calculated on the male coefficient of additive genetic variance ( $CV_A$ ) – female  $CV_A$  and male narrow-sense heritability ( $h^2$ ) – female  $h^2$  frequency distribution for different trait groupings. Positive values indicate that the additive genetic variance is male biased, and negative values indicate that it is female biased. We removed three outliers in the  $CV_A$  data set (see text for details). “Reproduction” included traits known or indicated to be under sexual selection; “not reproduction” included all other traits. Asterisks denote means significantly different from 0 at  $P < .05$  by a randomization test.

results demonstrate that extreme values of  $CV_A$  and  $h^2$  are more common among males than among females.

We looked at data points  $\sim 2$  SD away from the mean to determine the source of the right male-biased skew. For male  $CV_A$  – female  $CV_A$ , the right tail (male-biased variance) had 8 (out of 188) morphological, 1 (of 10) physiological, 1 (of 14) behavioral, and 2 (of 26) survival and developmental traits. The proportion of these groups did not differ (Fisher’s exact test,  $P = .349$ ). For male – female  $h^2$ , the right tail had 10 (out of 338) morphological, 1 (out of 64) mass, 2 (out of 32) behavioral, and 4 (out of 41) survival and developmental traits. The proportions did not differ (Fisher’s exact test,  $P = .104$ ). We analyzed the trait- and organism-specific distributions further in the appendix. Subsets of the data did not generally demonstrate significant sex-biased means and skews after multiple testing correction (tables A1, A2).

Normal distributions have a kurtosis of 0. We tested for departures from normality. We find that the distribution of the  $h^2$  differences has a kurtosis of  $2.490 \pm 0.217$  ( $P < .0001$ ) and that the distribution of the  $CV_A$  differences has a kurtosis of  $4.352 \pm 0.293$  ( $P < .0001$ ). Both distributions are significantly leptokurtic, or pointy; many traits had a very small male – female difference, as confirmed by the nonsignificant mean difference.

*Reproduction-Related Traits.* Several studies specified whether traits were under sexual selection or related to sexual reproduction. In animals, these were traits such as copulation duration, sexually selected trait size, age at first reproduction, mating frequency, and body size (when explicitly stated as under sexual selection). In plants, these were traits such as flower number, flower size, calyx size, and seed mass (only in gynodioecious species).

To see whether the patterns in figure 1 were attributable to the reproduction-related traits, we analyzed the “reproduction–” and “not reproduction–” related traits separately. The  $CV_A$  mean difference among the not reproduction–related traits was not significantly different from

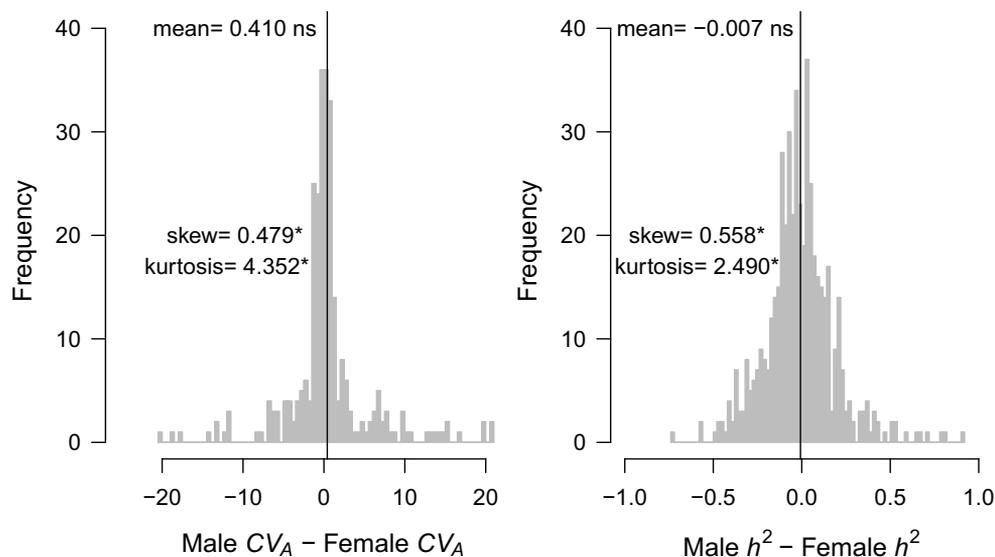
0 (table 1) although there was a significant male-biased skew (table 2). By contrast, the  $h^2$  mean difference was significantly female biased and the skew was only marginally male biased. Extreme female-biased values were not the cause of the significant female-biased mean  $h^2$  among the traits not related to reproduction.

We also separately analyzed the distribution characteristics for the reproduction-related traits by themselves. For the male  $CV_A$  – female  $CV_A$  distribution, the mean difference was also not significantly different from 0 by a two-tailed randomization test (table 1); the skew was also not significant (table 2). By contrast, for the male  $h^2$  – female  $h^2$  distribution, the mean difference was male biased and significantly different from 0. Furthermore, the skew was significantly positive and male biased (table 2). So, heritabilities but not coefficients of additive genetic variance suggest that male variances on average are greater than female variances among reproduction-related traits. We looked more closely at the means and skews for animal versus plant reproduction-related traits and find no obvious differences between the two groups in the male – female difference for either  $h^2$  or  $CV_A$  (see appendix for more details).

Finally, we directly compared the data set of the reproduction-related traits to the data set without the reproduction-related traits. The mean  $CV_A$  difference was not statistically different between the two groups (parametric ANOVA,  $F_{1,277} = 2.276$ ; permutation test,  $P = .0624$ , 1,507 iterations). However, the mean  $h^2$  difference was significantly different between the two groups (parametric ANOVA,  $F_{1,506} = 17.74$ ; permutation test,  $P < .0001$ , 5,000 iterations), with the traits related to reproduction having a male-biased mean and the traits not related to reproduction having a female-biased mean (table 1).

#### *Sexual Dimorphism in Phenotypic and Residual Variances*

Males had significantly larger coefficients of phenotypic variance than females across the entire data set (table 3).



**Figure 1:** Variance dimorphism frequency distributions. The frequency distributions of the male coefficient of additive genetic variance ( $CV_A$ ) – female  $CV_A$  difference (*left*) and the male narrow-sense heritability ( $h^2$ ) – female  $h^2$  difference (*right*). Positive values indicate that the additive genetic variance is male biased, and negative values indicate that it is female biased. The black line represents the mean difference, which is not significantly (ns) different from 0 for either distribution.

The mean male  $CV_p$  – female  $CV_p$  was significantly male biased among the not reproduction–related traits by a randomization test; however, it was not significantly different by a parametric  $t$ -test ( $t_{188} = 1.796$ ,  $P = .074$ ; table 3). The mean male  $CV_p$  – female  $CV_p$  was not significantly different from 0 among the reproduction-related traits (table 3). The coefficients of phenotypic variance of the entire data set, the reproduction-related traits, and the not reproduction–related traits had significant male-biased skews (table 3). Finally, reproduction-related traits showed more male-biased phenotypic variance than not reproduction–related traits in the entire data set (parametric ANOVA,  $F_{1,215} = 6.281$ ; permutation test,  $P = .030$ , 3,183 iterations).

Males also had significantly larger coefficients of residual variance than females in the entire data set and in the traits not related to reproduction (table 4). The male  $CV_R$  – female  $CV_R$  was not statistically different from 0 among the reproduction-related traits. The coefficients of residual variance of the entire data set, the reproduction-related traits, and the not reproduction–related traits had significant male-biased skews (table 4). Finally, a direct comparison between the reproduction-related and not reproduction–related traits did not show any significant differences in male – female means (parametric ANOVA,  $F_{1,205} = 1.379$ ; permutation test,  $P = .083$ , 1,104 iterations).

#### Sexual Dimorphism in $V_A$ and the Mean

We found a positive correlation between sexual dimorphism in variances and sexual dimorphism in means (fig. 2). A nonparametric Spearman rank correlation test between the log(base 2) of the male : female variance ratios and mean ratios was significant ( $N = 256$ ;  $\rho = 0.243$ ;  $P < .0001$ ) for the entire data set. After removing four outliers with extremely high sexual dimorphism in genetic variances or in means (over 16-fold difference between the sexes), this correlation remained significant ( $N = 252$ ;  $\rho = 0.194$ ;  $P = .007$ ). When the data are divided into traits with male-biased means versus female-biased means, the correlation between variance dimorphism and mean dimorphism is significant in both the female-biased traits ( $N = 120$ ;  $\rho = 0.183$ ;  $P = .045$ ) and the male-biased traits ( $N = 132$ ;  $\rho = 0.405$ ;  $P < .0001$ ) considered separately. When the correlations between dimorphism in means and in variances are performed on a trait-by-trait basis, they are significant for morphological ( $N = 150$ ;  $\rho = 0.231$ ;  $P = .004$ ) and mass ( $N = 49$ ;  $\rho = 0.591$ ;  $P < .0001$ ) traits. We also used a mixed model (see “Methods”) to take into account the nested structure of the data. Sexual dimorphism in additive genetic variance had a significant effect on phenotypic size dimorphism ( $F_{1,149} = 9.67$ ,  $P = .0022$ ).

**Table 2:** Skew of the sexual difference in additive genetic variances

	Male $CV_A$ – female $CV_A$		Male $h^2$ – female $h^2$	
	Skew $\pm$ 1 SE (N)	P value	Skew $\pm$ 1 SE (N)	P value
Combined data	.479 $\pm$ .147 (279)	.001*	.558 $\pm$ .109 (508)	<.0001*
Reproduction	.212 $\pm$ .374 (43)	.287	.526 $\pm$ .297 (68)	.041*
Not reproduction	.312 $\pm$ .159 (236)	.026*	.192 $\pm$ .117 (440)	.051

Note: The skew was calculated on the male coefficient of additive genetic variance ( $CV_A$ ) – female  $CV_A$  and male narrow-sense heritability ( $h^2$ ) – female  $h^2$  frequency distribution for different trait groupings. We removed three outliers in the  $CV_A$  data set (see text for details). “Reproduction” included traits known or indicated to be under sexual selection; “not reproduction” included all other traits. Asterisks denote skews significantly different from 0 at  $P < .05$ .

### Sex Chromosomes

We find no evidence consistent with the hypothesis that sex linkage alone is enough to explain sex-specific variance differences. For example, when measured by  $CV_A$ , butterflies have a nonsignificant male-biased mean (table A1), whereas a significant female-biased mean would be predicted by female heterogamety. Similarly, birds have a female-biased mean, but it is not significantly different from 0. By contrast, mammals have a nonsignificant female-biased mean whereas male heterogamety would predict a significant male-biased mean. However, we do observe that flies and beetles have a significant male-biased mean and skew as measured by  $CV_A$  (tables A1, A2). These patterns are not observed when the additive genetic variance is measured by  $h^2$ : flies have a significant male-biased skew but a nonsignificant male-biased mean. Beetles have a significant female-biased skew and nonsignificant male-biased mean.

Because of the complicated nature of the data set, we tested for a sex chromosome effect in a mixed model. We entered heterogamety type (i.e., male or female) as a fixed effect and entered the sexual dimorphism index ( $\log(\text{male size}/\text{female size})$ ) as a covariate. We entered the trait type within species as a random factor to partially adjust for the nonindependence of trait measurements. We entered species within organismal group as a random factor to partially adjust for phylogenetic nonindependence. Organismal groups (i.e., plant, butterfly, beetle, fly, bird, grasshopper, mammal, fish) were based on the most frequent taxonomic types in the data (see appendix). How-

ever, the model fit was not improved by including organismal group, so we removed it. We tried three different indexes of additive genetic variance dimorphism as the dependent variable in our mixed model: male  $h^2$  – female  $h^2$ , male  $CV_A$  – female  $CV_A$ , and  $\log(\text{male } V_A/\text{female } V_A)$ . We found that heterogamety was not significant in any of the mixed models. We also looked at male  $CV_P$  – female  $CV_P$  and male  $CV_R$  – female  $CV_R$  and found no effect of heterogamety.

Although many traits in our study were phenotypically sexually dimorphic, suggesting potential sex linkage, very few studies employed methods capable of detecting sex-linked additive genetic variance. If a quantitative genetic design does not take into account heterogamety, the sire variance measured in females will be greater than the sire variance measured in males for XY species (Cowley et al. 1986; Cowley and Atchley 1988). Interestingly, despite this expected female bias, XY beetles and flies actually showed significant male-biased  $CV_A$  in means (table A1) and significant male-biased skews in variances (table A2).

### Discussion

Variability between the sexes has long fascinated biologists. Darwin (1871) hypothesized that much of this variation could be traced to differences in the intensity of sexual selection in males compared to females. This insight has been affirmed by the observation that male sexually selected traits often have higher amounts of phenotypic and additive genetic variance than nonsexually selected traits

**Table 3:** Sexual dimorphism in the coefficients of phenotypic variance (male  $CV_P$  – female  $CV_P$ )

	Mean $\pm$ 1 SE (N)	P value	Skew $\pm$ 1 SE (N)	P value
Combined data	4.212 $\pm$ 1.663 (217)	.004*	6.278 $\pm$ .166 (217)	<.0001*
Reproduction	14.91 $\pm$ 8.132 (28)	.065	2.181 $\pm$ .463 (28)	<.0001*
Not reproduction	2.627 $\pm$ 1.462 (189)	.040*	9.048 $\pm$ .178 (189)	<.0001*

Note: Tests for the significance of the mean and skew of the male  $CV_P$  – female  $CV_P$  frequency distribution for different trait groupings. “Reproduction” included traits known or indicated to be under sexual selection; “not reproduction” included all other traits. Asterisks denote significance for the mean at  $P < .05$  by a randomization test or for the skew at  $P < .05$ .

**Table 4:** Sexual dimorphism in the coefficients of residual variance (male  $CV_R$  – female  $CV_R$ )

	Mean $\pm$ 1 SE (N)	P value	Skew $\pm$ 1 SE (N)	P value
Combined data	2.004 $\pm$ .800 (207)	.009*	3.831 $\pm$ .170 (207)	<.0001*
Reproduction	4.429 $\pm$ 4.237 (27)	.319	1.274 $\pm$ .471 (27)	.006*
Not reproduction	1.641 $\pm$ .672 (180)	.006*	5.829 $\pm$ .183 (180)	<.0001*

Note: Tests for the significance of the mean and skew of the male  $CV_R$  – female  $CV_R$  frequency distribution for different trait groupings. “Reproduction” included traits known or indicated to be under sexual selection; “not reproduction” included all other traits. Asterisks denote significance for the mean at  $P < .05$  by a randomization test or for the skew at  $P < .05$ .

(Pomiankowski and Møller 1995; Rowe and Houle 1996). We asked whether male-biased patterns of variation extended more generally to homologous male and female traits.

We demonstrate that homologous male and female traits do not differ in their mean amount of additive genetic variance. Yet homologous traits did have greater mean male residual variance and greater mean male phenotypic variance. Furthermore, we find consistently significant male-biased skews in additive genetic, residual, and phenotypic variance across the entire data set and in some subsets of the data (after multiple testing correction). However, we also observed a female-biased mean  $CV_A$  in mass traits and a female-biased mean  $h^2$  in traits not related to reproduction. Dimorphism in additive genetic variances enables the evolution of trait dimorphism (Lynch and Walsh 1998), and in support, we show that  $V_A$  sexual dimorphism is positively associated with phenotypic sexual dimorphism in this large data set. While differences in sex chromosome system may account for sex-biased patterns of additive genetic variance, we found little support for this association in our data set.

#### *Male-Biased Skew but No Sexual Dimorphism in Mean $V_A$*

The mean male – female difference in both  $CV_A$  and  $h^2$  was not significantly different from 0 in the entire data set (fig. 1). Both the distributions were extremely leptokurtic, so that many differences were close to 0 (fig. 1). Shared homologous traits may harbor similar amounts of genetic variance due to strong intralocus sexual conflict (Cox and Calsbeek 2009). However, even shared traits can have very high sexual dimorphism in additive genetic variances, as a significant male-biased skew was prevalent (fig. 1; tables 2, A2).

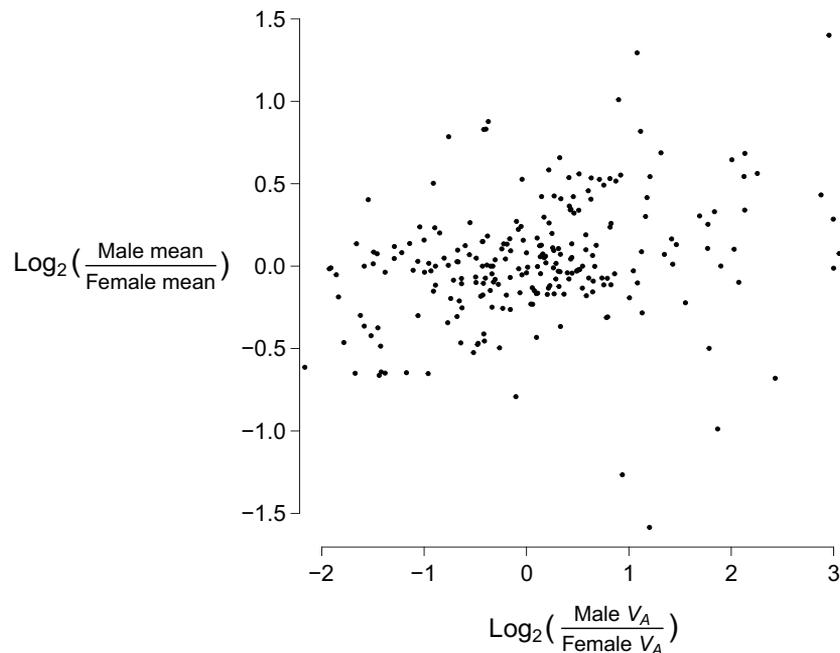
When reproduction-related and unrelated traits were considered separately, male  $CV_A$  – female  $CV_A$  in neither group was significantly different from 0, and the two groups did not differ from one another. The lack of difference between groups was surprising since Pomiankowski and Møller (1995) demonstrated that sexual traits

have more additive genetic variance than nonsexual traits. Differences in methodology may account for the discrepancy. We targeted shared homologous traits while Pomiankowski and Møller (1995) compared sexual and nonsexual traits that were not necessarily homologous between the sexes. Shared traits may have a higher intersexual genetic correlation, resulting in the nonsignificant male  $CV_A$  – female  $CV_A$  difference between reproduction-related and not reproduction-related traits. Consistent with Pomiankowski and Møller (1995) the mean male  $CV_P$  – female  $CV_P$  difference was greater in reproduction-related traits than in not reproduction-related traits. The mean male  $CV_R$  – female  $CV_R$  was also greater among reproduction-related traits. And unlike for  $h^2$  or  $CV_A$ , we found that male traits had more  $CV_P$  and  $CV_R$  than female traits, as measured by both the mean and the skew.

We found a positive correlation between the extent of sexual dimorphism in additive genetic variances and the extent of phenotypic sexual dimorphism. Male-biased traits have greater male  $V_A$ , and female-biased traits have greater female  $V_A$  (fig. 2). However, it is unclear how scalar relationships between the mean and variance in each sex may affect this positive correlation (see the appendix for further discussion). The positive correlation is consistent with greater male  $V_A$  facilitating male-biased phenotypic traits (and similarly for female-biased traits). Alternatively, strong sex-specific selection on phenotypic means may have caused the increased sex-specific differences in  $V_A$  via the evolution of condition dependence. Traits that come to rely on the bearer’s condition, such as sexually selected and life-history traits, will absorb some portion of the large additive genetic variance in condition when under strong directional selection (Rowe and Houle 1996).

#### *Discordance between $CV_A$ and $h^2$*

$CV_A$  and  $h^2$  are not expected to be correlated (Houle 1992; Hansen et al. 2011). We find that sexual dimorphism in  $CV_A$  and sexual dimorphism in  $h^2$  did not usually correspond in one-to-one comparisons (results not shown). We observed that  $h^2$  but not  $CV_A$  indicated more male bias in reproduction-related traits compared to not reproduction-



**Figure 2:** Phenotypic sexual dimorphism versus variance dimorphism. The amount of dimorphism in additive genetic variance is positively correlated with the extent of phenotypic sexual dimorphism ( $N = 252$ ,  $\rho = 0.194$ ,  $P = .007$ ).

related traits;  $h^2$  may be a poor predictor of evolvability (Hansen et al. 2011). However, sexual differences in  $h^2$  are still expected to be important to the degree of phenotypic sexual dimorphism (Reeve and Fairbairn 1996) in the variance-scaled formulation of quantitative genetics.

#### *Potential Causes of Greater Male $CV_R$ and $CV_P$*

Greater male  $CV_R$  may occur through more male-specific dominance and epistatic variances. Greater male  $CV_P$  may occur through more dominance, epistatic, and/or environmental variances. The increased  $CV_R$  and  $CV_P$  of male traits is a little mysterious because we did not focus on sexually selected traits. Unlike Pomiankowski and Møller (1995), we used only homologous traits expressed in both sexes to conduct our paired comparisons. Many of the remaining traits may have unrecognized roles in reproduction or may be correlated with such traits, accounting for their greater variation (Rowe and Houle 1996).

Interestingly, the pattern of greater male-related phenotypic variation is an emerging trend in the transcriptomics literature. In *Drosophila melanogaster*, males have greater additive genetic variance in gene expression than females (Wayne et al. 2007). Furthermore, phenotypic variance in gene expression is greater among male-biased genes than among female-biased genes at the interspecific and intraspecific level in whole-body assays (Meiklejohn

et al. 2003; Zhang et al. 2007; Wyman et al. 2012), although some differences may arise from sex differences in tissue proportions (Stewart et al. 2010).

Over long evolutionary timescales, gene duplications may contribute to the greater expression variation of male-biased genes. Male-biased genes have greater duplicate enrichment than female-biased or unbiased genes (Gnad and Parsch 2006; Gallach et al. 2010; Wyman et al. 2012; Assis and Bachtrog 2013). Male-biased genes are more common in late-replicating regions of the genome where duplication events are more likely (Cardoso-Moreira and Long 2010), and they are overrepresented among evolutionarily young genes (Zhang et al. 2010). Male-biased genes also have a higher substitution rate and bear signatures of positive selection, suggesting adaptation (Sella et al. 2009; Zhang et al. 2010; Andolfatto et al. 2011; Grath and Parsch 2012; Meisel et al. 2012). Both gene duplications and base substitutions may contribute to sex-specific differences in additive genetic and nonadditive genetic variances.

Even if mutational processes are biased toward generating genes with male functions, the same processes may generate both male-benefit and male-detriment genes, at least initially. The introduction of good and bad male alleles may account for the large variance we observe in male fitness and in traits related to male fitness. Recent data suggest that the extent of within-sex variation in sexual traits is nonrandom with respect to fitness. High-fitness

males have less additive genetic variance than low-fitness males in sexually selected traits in *Drosophila bunnanda* (McGuigan and Blows 2008) and *Drosophila serrata* (Sztepanacz and Rundle 2012). Males successful at obtaining mates not only possess the most attractive traits but are also constrained in their extent of variation—perhaps because they do not bear as many deleterious alleles with pleiotropic effects as the unsuccessful males (McGuigan et al. 2011). It is unclear whether similar patterns occur among females.

Finally, it is possible that sex linkage may underlie differences in male and female variance for quantitative traits and gene expression. Although male heterogamety versus female heterogamety may explain patterns of phenotypic variance (Reinhold and Engqvist 2013), we find little support for this effect in our data set for additive genetic, residual, or phenotypic variances. It is likely that many, if not all, of the traits in this study have a complex polygenic basis. If so, both autosomal and sex-linked loci may contribute to trait expression. Many more autosomal than sex-linked loci may dampen the signal of greater heterogamety variance. Partial or no dosage compensation is also expected to dampen the amount of additive genetic variance in the heterogametic sex (Reinhold and Engqvist 2013).

Whatever the cause of greater male than female non-additive genetic variance, we suggest an emerging convergence between classic phenotypic studies of sexual traits and genomic data on sex-biased gene expression. Further data from ZW and other XY systems will enable us to examine whether greater variation in male-biased gene expression is a peculiarity specific to *Drosophila*. Interestingly, the trend of greater variation among males may extend even to humans. Darwin (1874, p. 223) remarked on the greater morphological variation present among men than women. More recent data suggest that men may have greater variation in test scores (Hedges and Nowell 1995) and in other morphological traits (Lehre et al. 2009) than women, even though sex-specific means may differ very little. If true, why additive genetic variance and non-additive genetic variance might differ between the sexes in traits not clearly linked to reproduction is unclear—and it is an area of research that has made little progress since Darwin (1874, p. 224) first proposed it as a general phenomenon.

### Conclusions

The evolution of adaptive sexual dimorphism requires sex-specific selection, a low intersexual genetic covariance, and/or sexual dimorphism in sex-specific additive genetic variances (Lande 1980). We have shown that additive genetic variances do not differ overall between the sexes. However, additive genetic variances can differ in the ex-

tremes of the distribution, and sexual dimorphism in  $V_A$  is correlated with sexual dimorphism in phenotypes. Moreover, residual and total phenotypic variances are sexually dimorphic. It will be interesting to discern the ultimate and proximate causes of this greater sex-specific variation.

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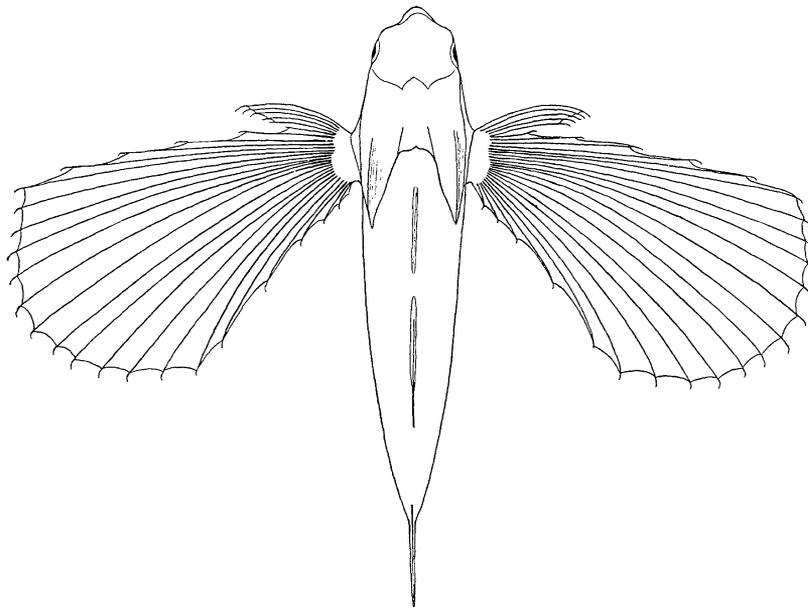
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“In *Dactylopterus* the pectoral fins are especially developed for flight and they alone give support to the body in the air as the pelvic fins, which are situated just beneath the pectorals, are not visible from above even when extended. The pectoral fins as a compensation are much broader than in *Exocoetus*, the relative alar expanse compared with the bulk of the body being much the same in the two genera [...]” From “Volant Adaptation in Vertebrates” by Richard S. Lull (*The American Naturalist*, 1906, 40:537–566).