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SEX CHROMOSOME LINKED GENETIC VARIANCE AND THE EVOLUTION OF SEXUAL DIMORPHISM OF QUANTITATIVE TRAITS

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Theory predicts that sex chromsome linkage should reduce intersexual genetic correlations thereby allowing the evolution of sexual dimorphism. Empirical evidence for sex linkage has come largely from crosses and few studies have examined how sexual dimorphism and sex linkage are related within outbred populations. Here, we use data on an array of different traits measured on over 10,000 individuals from two pedigreed populations of birds (collared flycatcher and zebra finch) to estimate the amount of sex-linked genetic variance (h^2_Z). Of 17 traits examined, eight showed a nonzero h^2_Z estimate but only four were significantly different from zero (wing patch size and tarsus length in collared flycatchers, wing length and beak color in zebra finches). We further tested how sexual dimorphism and the mode of selection operating on the trait relate to the proportion of sex-linked genetic variance. Sexually selected traits did not show higher h^2_Z than morphological traits and there was only a weak positive relationship between h^2_Z and sexual dimorphism. However, given the relative scarcity of empirical studies, it is premature to make conclusions about the role of sex chromosome linkage in the evolution of sexual dimorphism.

KEY WORDS: **Animal model, collared flycatcher, quantitative genetics, sex linkage, sexual antagonism, sexual dimorphism, zebra finch.**

A common observation in many different taxa is that sexual and/or natural selection favors different phenotypic trait optima in males and females, leading to the evolution of sexual dimorphism (Arnqvist and Rowe 2005; Fairbairn et al. 2007). Because the two sexes share the same genome, intersexual genetic correlations may impede the evolution of sexual dimorphism and prevent a resolution of sexual conflict (Fisher 1931). Much research has therefore been directed toward understanding the genetic details that allow sexual dimorphisms to evolve (Bonduriansky 2007; Williams and Carroll 2009).

One possible mechanism that could solve, or substantially reduce, intersexual conflict in trait optima is sex chromosome linkage (hereafter referred to as sex linkage), that is, when sexually antagonistic genes are located on the sex chromosomes (Rice 1984). For example, male beneficial alleles located on the Y chromosome in XY-systems or female beneficial alleles on the W chromosome in ZW-systems would immediately lead to sex-specific expression and resolution of the intersexual conflict. Similarly, sexually antagonistic alleles could increase in frequency more easily if they are located on the macro sex chromosomes (X and Z), as shown analytically by Rice (1984), and this should facilitate evolution of sexual dimorphism.

The theoretical work by Fisher (1931) and Rice (1984) suggests that: first, genetic variants influencing sexually selected

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traits should be overrepresented on the sex chromosomes when compared to morphological traits because sexually selected traits are more frequently under sex-specific (antagonistic) selection. Second, the degree of sex-linked genetic variance should correlate positively with the amount of sexual dimorphism at the phenotypic level (Fairbairn and Roff 2006) because sex linkage should reduce intersexual genetic correlations and therefore allow sexual dimorphism to evolve.

Three main approaches have been used to test for sex linkage in the context of sexual dimorphism: reciprocal crosses between populations or closely related species (reviewed in Reinhold 1998), within-population quantitative genetic studies (Fairbairn and Roff 2006), and, more recently, studies that have examined the genomic location of genes with sex-biased expression (Mank 2009; Mank and Ellegren 2009).

These approaches target slightly different stages in the evolution of sexual dimorphism and should therefore be considered complementary rather than exclusive to each other. Reciprocal crosses are likely to detect genes on the sex chromosomes that are fixed or show large differences in allele frequency between populations and although such genes may contribute to sexual dimorphism, they are no longer important for the continued evolution of sexual dimorphism within that population. Studies using reciprocal crosses thus focus on the later stages in the evolution of sexual dimorphism. Because genes that are divergent between populations are often not polymorphic within populations they remain undetectable in a quantitative genetic framework. The quantitative genetic approach in contrast can provide insight into the amount of standing variation in sexually antagonistic loci and thus focus on short-term potential for sexual dimorphism to evolve. Rice (1984) showed analytically that polymorphic sexually antagonistic loci should be overrepresented on the sex chromosomes because X-linked recessive alleles beneficial to the hetergametic sex (or dominant alleles detrimental to the homogametic sex) could increase to higher frequency than if they were located on autosomes, thereby allowing sexual dimorphism to evolve. Quantitative genetic approaches thus allow us to test for association between sex-linked genetic variance and sexual dimorphism as predicted from the theoretical models developed by Rice (1984).

Once the sexually antagonistic allele have increased in frequency so that fitness in the two sexes is maximized, Rice (1984) predicted that "modifier genes" should evolve to restrict expression of the sexually antagonistic allele to the sex in which it is beneficial, thereby removing intralocus conflict. To test this, gene expression studies (using microarray or RNA sequencing methods) have recently examined the genomic location of so-called sex-biased genes, that is, genes that are differentially expressed between the sexes (Ellegren and Parsch 2007). Sex-biased gene expression can be taken to infer past sexual antagonism (Connallon and Knowles 2005) and have the benefit that a larger proportion of the genome can be tested because information on variation in the sexually antagonistic phenotype or the underlying loci is not needed (Mank and Ellegren 2009). However, gene expression studies alone are not able to relate the loci to phenotypes and thus make it difficult to relate to observed levels of sexual dimorphism in phenotypic traits.

Most empirical knowledge about the extent of sex linkage has come from reciprocal line crosses, either between populations of the same species or between closely related species, and the majority of these studies have been conducted in mice and *Drosophila* (Reinhold 1998). However, despite some clear examples of sex linkage of sexually dimorphic traits in some taxa (Lindholm and Breden 2002), there is overall inconclusive evidence for the idea of elevated sex linkage in sexually dimorphic traits (Mank 2009). More empirical data are thus needed to settle the issue of the role of the sex chromosomes in generating sexual dimorphism. In particular, we know very little about the amount of standing genetic variation that resides on the sex chromosomes, even though such information is required to understand the microevolution of sexual dimorphism. It is therefore important that sex linkage and sexual dimorphism is tested also within populations and not only inferred from crosses (Fairbairn and Roff 2006).

In this paper, we focus on estimating sex-linked genetic variance and sexual dimorphism within populations using a quantitative genetic approach. Genetic variance can be estimated in different ways but the development of statistical methods that simultaneously use many different types of relatives ("animal model," Henderson 1950) has during the last decade reinvigorated the study of quantitative genetics in natural populations (Kruuk 2004). These studies have provided important insight into the genetic underpinnings of topics ranging from phenotypic plasticity (e.g., Husby et al. 2011) and mating preferences (e.g., Qvarnström et al. 2006), to sexually antagonistic selection (e.g., Foerster et al. 2007; Forstmeier et al. 2011). However, these studies have estimated the additive genetic variance assuming only autosomal inheritance and therefore ignored the potential that genes may be sex linked. Sex-linked genetic variation might inflate the estimate of autosomal additive genetic variance, but may also be pooled with the residual variance. Both situations are not desirable. Moreover, lack of explicit modeling of sex-linked variation is unfortunate because sex-linked genes have many interesting and unique evolutionary properties. For example, the presence of sex linkage has direct consequences for models of sexual selection and theoretical work has shown that if genes for sexual ornaments and female preference are both linked to the sex chromosomes, the possibility of Fisher's runaway selection (under a ZW system) or "good genes model" (under an XY system) is much more likely to take place than under autosomal inheritance (Kirkpatrick and Hall 2004). Moreover, empirical studies have clearly demonstrated that sex-linked effects can be substantial, as shown in studies ranging

from *Drosophila* (Cowley et al. 1986) to humans (Pan et al. 2007) and in some species, the sex chromosome can contain a substantial fraction of genes. For example, approximately 16% of all genes in *Drosophila melanogaster* are located on the X chromosome and approximately 4% are on the Z chromosome in the zebra finch. This suggests that sex-linked effects should be modeled explicitly when examining the quantitative genetic basis of trait variation, particularly for traits evolving under sexual selection where there are indications that sex-linked genes are particularly prevalent.

However, to our knowledge, only very few studies have used a pedigree-based approach to estimate sex-linked standing genetic variation on the sex chromosomes (e.g., Pan et al. 2007; Roulin et al. 2010). Consequently, the presence and extent of sex-linked genetic inheritance within populations remains largely unexplored and lags far behind theoretical developments in the field.

Our aim in this study was to provide increased knowledge of the extent of Z-linked genetic variance using a quantitative genetic approach and how within-population sexual dimorphism is related to Z-linked genetic variance. To avoid the complications introduced by dosage compensation (see e.g., Mank et al. 2011), we chose two long-term pedigree studies of birds as study systems: a natural population of collared flycatchers (*Ficedula albicollis*) and a captive population of zebra finches (*Taeniopygia guttata*). These populations are particularly well suited to study autosomal and sex-linked patterns of inheritance because of their well-characterized pedigree structure and the availability of detailed measurements on a wide range of morphological and sexually selected traits. In total, we included information on 17 morphological and sexually selected traits measured on over 10,000 different individuals across the two study species. Based on theoretical models, we predicted that sexually selected traits should show a higher degree of sex linkage than nonsexually selected traits (Rice 1984) and that the extent of sex linkage should increase with increasing sexual dimorphism (Fairbairn and Roff 2006). We thus focus explicitly on testing predictions from theoretical models for how sex linkage should be associated with observed levels of sexual dimorphism for different types of traits assuming presence of segregating sexually antagonistic loci.

Methods

STUDY POPULATIONS AND DATA COLLECTION

Data on the collared flycatcher were collected as part of a long-term study on Gotland, Sweden, that started in 1980 (see Gustafsson 1986). Each year, parents and offspring were measured and ringed and morphological measurements, laying date, and clutch size recorded (see Table 1). Detailed information about the study population and how measurements were taken is given in Gustafsson (1989).

Data on zebra finches were collected in a captive population maintained in Seewiesen, Germany. Our population of zebra finches originated from population that had been maintained at Sheffield university since 1985 (Forstmeier et al. 2007) and had prior to that date been living in captivity for a large, but unknown number of generations. Morphological data were routinely collected at independence (age 35 days), at 100 days of age, and on a few other occasions during adult life and behavioral data were collected in standardized trials (Forstmeier et al. 2012) and reproductive success in competitive environments was quantified during approximately three-month breeding seasons in aviaries (Schielzeth et al. 2011). The number of individuals measured, number of records, trait means, and SDs for the different traits can be found in Table 1.

TRAIT CLASSIFICATION AND SEXUAL DIMORPHISM

We classified traits in two broad categories as either "naturally selected" or "sexually selected" (see Table 1). The first category comprises morphological traits that show very little sexual dimorphism, whereas the second category comprises traits that are strongly sexually dimorphic or entirely sex limited, which suggests that they are or have been under sexual or sexually antagonistic selection. We note that this classification as sexually selected does not necessarily imply that the trait is currently under directional sexual selection in the studied population (see Schielzeth et al. 2012b), but at least past sexually antagonistic selection seems likely for all of the traits included in this category. For example, both a correlational and an experimental manipulation study have found the wing patch in collared flycatchers to be under sexual selection (Sheldon and Ellegren 1999; de Heij et al. 2011). The current evidence for zebra finch beak color being sexually selected is somewhat ambiguous and differs between populations (discussed in Schielzeth et al. 2012b), but because the long-term evolutionary history of sexual selection matters in the context discussed here, we included beak color as a sexually selected trait. Note that we were unable to classify some traits as sexually or naturally selected. These traits are therefore only included in the analyses looking at the overall extent of sex-linked genetic variance.

The degree of sexual dimorphism for traits expressed in both sexes was calculated using two different approaches. First, we estimated sexual dimorphism as the absolute value of the ratio of the mean trait value of the sex with the larger mean trait value divided by the mean trait value in the sex with smaller trait value minus one, that is, $SD = |\mu_{larger\_sex}/\mu_{smaller\_sex} - 1|$ as in Poissant et al. (2010). However, because standardization to the mean value is not meaningful for zebra finch beak color, we cannot apply the formula above in a meaningful way. Therefore, we also used a standardized effect size expressed as Cohen's

Table 1. Summary statistics of the traits used in the collared flycatcher population (a) and zebra finch population (b). Note that some traits are sex limited (expressed only in one sex) or were analyzed separately for the two sexes (see column "Sex," F, females; M, males). Trait type abbreviations: S, sexually selected traits; M, morphological traits; NA, not applicable.

Trait	Trait type	Sex	Individuals	Records	Mean	SD
(a) Collared flycatcher						
Male forehead patch	S	M	3091	4621	76.67	15.18
Female choice—forehead patch	NA	F	3280	4603	76.04	16.08
Female choice—wing patch	NA	F	3364	4727	40.34	16.89
Body mass	M	F+M	6771	9995	13.68	1.33
		F	3718	5382	14.28	1.45
		M	3053	4613	12.98	0.70
Tarsus	M	F+M	7206	10,781	19.37	0.58
		F	4066	6030	19.40	0.56
		M	3140	4751	19.34	0.59
Tail	M	F+M	6302	9124	51.48	1.63
		F	3595	5153	51.05	1.51
		M	2707	3971	52.05	1.61
Wing patch	S	F+M	7177	10,694	29.41	15.89
		F	4035	5943	20.38	7.03
		M	3142	4751	40.71	16.63
Wing length	M	F+M	7295	10,953	81.72	2.18
		F	4116	6134	80.98	1.96
		M	3179	4819	82.67	2.07
(b) Zebra finch						
Tarsus length	M	F+M	2254	2254	17.14	0.58
		F	1093	1093	17.09	0.58
		M	1161	1161	17.19	0.57
Wing length	M	F+M	2502	2502	58.19	1.53
		F	1220	1220	58.02	1.55
		M	1282	1282	58.36	1.50
Adult weight	M	F+M	2510	7181	15.88	2.11
		F	1226	3521	16.10	2.21
		M	1284	3660	15.67	1.98
Mass day 8	M	F+M	2319	2319	7.23	1.72
		F	1128	1128	7.30	1.73
		M	1191	1191	7.15	1.70
Beak color	S	F+M	1017	1864	−0.35	1.70
		F	487	816	−1.89	1.0
		M	530	1048	0.85	1.04
Choice activity	NA	F	552	846	19.54	9.30
Fecundity in cages	S	F	270	546	12.89[1]	6.62[2]
Courtship rate	S	M	1109	4984	3.76	2.52
Male attractiveness	S	M	582	582	0.62	0.16

[1]Predicted for a 12-week breeding period.

[2]After accounting for variation in the duration of the breeding season.

D, that is, $D = |\mu_{\text{males}} - \mu_{\text{females}}| / \sqrt{(((n_{\text{males}} - 1) \times \sigma^2_{\text{males}} + (n_{\text{females}} - 1) \times \sigma^2_{\text{females}})/(n_{\text{males}} + n_{\text{females}} - 2))}$ (Nakagawa and Cuthill 2007) as an alternative measure of sexual dimorphism. This allowed us to include also beak color when assessing the relationship between sexual dimorphism and sex-linked genetic variance.

PEDIGREE INFORMATION

In the collared flycatchers, relatedness was calculated from a pedigree based on field observations and will therefore contain some errors through the paternal line due to extra pair paternity (15% of nestlings, Sheldon and Ellegren 1999). Simulations have shown that estimates of genetic variance are relatively robust even with

pedigree errors as large as 20% (Charmantier and Réale 2005) and that errors should lead to a downward bias in the estimates, meaning they will be conservative. Pedigrees reconstructed based on molecular markers such as microsatellites also typically have error rates in the range of 10–20% (see e.g., Kruuk et al. 2000). In the zebra finch, pairs were allowed to breed separately in cages or in flocks in aviaries. All individuals were genotyped for microsatellites and parentage was assigned by exclusion and extensive SNP genotyping did not reveal a single pedigree error in the zebra finch dataset (Forstmeier et al. 2012; Schielzeth and Bolund 2010). The zebra finch pedigree goes back to the parents of the first generation whose data are included in the current study.

Information on pedigree statistics for the two populations was obtained using the R package pedantics (Morrissey and Wilson 2010). These statistics depend on the individuals that were phenotyped and therefore differs between traits. In Table S1, we show summaries using the tarsus length dataset as an example in both species.

ESTIMATING AUTOSOMAL ADDITIVE GENETIC VARIANCE

A mixed model incorporating relatedness information ("animal model," Henderson 1950; Kruuk 2004) was used to partition phenotypic variance into autosomal additive genetic and environmental components of variance according to the following model:

$$\mathrm{y} = \mathbf{X}\mathrm{b} + \mathbf{Z_1}\mathrm{a} + \mathbf{Z_2}\mathrm{pe} + \mathbf{Z_3}\mathrm{m} + e. \quad (1)$$

Here, y refers to the vector of individual trait observations (see Table 1), $\mathbf{X}$ and $\mathbf{Z_i}$ are design matrices that relate the fixed and random effects to the observations. b is the vector of fixed effects and a, pe, m are vectors of random effects related to the autosomal additive genetic effect, permanent environmental effects, and maternal effect, respectively, and e is a vector of residual deviations. Although maternal effects are often weak for adult traits, if present, they could be partly confounded with a sex-linked effect (Fairbairn and Roff 2006). To avoid this, we included the dam component in all models even if not statistically significant.

The flexibility of the animal model allows the inclusion of additional fixed and random effects that we added depending on the trait and the sampling design. As additional random effect we included for the collared flycatcher year as random effect to take into account yearly fluctuations in environmental conditions and an area random effect to take into account spatial variation in environmental conditions. For the zebra finch we included a session random effect for traits that were measured in batches (weight, fecundity, courtship rate). This controls for shared current environment effects. Furthermore, all zebra finch models include foster pair identity as a random effect, because most subjects were individually cross-fostered at the egg stage. This allows for variation caused by rearing environment.

We included sex as fixed effect in all analyses that combined data from females and males. For the collared flycatcher, we controlled for age as a two-level factor ("young" = less than 2 years and "old" = 2 years or older). In addition, we included the interaction between sex and age in the analyses of wing patch size in collared flycatcher to control for differences in age effects between the sexes. Inbreeding coefficient as calculated from the pedigree was included as a covariate in the zebra finch analyses, but not in the collared flycatcher analyses as inbreeding events are extremely rare in this population (approximately 1% of breeding events, Kruuk et al. 2002). An indicator of feather generation (juvenile versus adult flight feathers) was fitted as a two-level factor in the analysis of zebra finch wing length (see Schielzeth et al. 2012a) and subadult age was also controlled for in the analysis of zebra finch beak color (as a continuous predictor with all adult individuals older than 120 days set to age = 120, because beak color does no longer change systematically beyond that age, see Schielzeth et al. 2012b for details) and adult weight. The model for zebra finch female fecundity is controlled for the duration of the breeding season by adding this factor as a continuous covariate. The analyses of mass at day 8 fits hatch order as fixed effects (1 df) and choice activity (the number of hops in front of males in a four-way choice chamber) included trial number of each female (value 1–3), which accounts for systematic changes due to familiarity with the setup.

ESTIMATING Z-LINKED GENETIC VARIANCE

To separate autosomal inheritance from sex-linked inheritance, we constructed a relatedness matrix specific for Z-linked genes. Unlike autosomal inheritance where daughters will have a coefficient of relatedness of 0.5 with their mothers, for a Z-linked gene the coefficient of relatedness between daughters and their mothers will be 0 because females receive their single Z chromosome from their father. Similarly, a granddaughter will be unrelated with respect to the Z chromosome to her maternal grandfather but have a 0.5 chance of inheriting the Z chromosome from her paternal grandfather. A table of pairwise relatedness coefficients for relatives for an X-linked gene can be found in Grossman and Eisen (1989) and a worked example in Fernando and Grossman (1990). These expressions can be accordingly modified for Z-linkage. In contrast to autosomal inheritance, it can be seen that not all relatives are informative with respect to a sex-linked analysis and, consequently, the power to detect sex linkage besides autosomal genetic variation is smaller than that of detecting autosomal genetic variance itself. This is particularly problematic for sex-limited traits (i.e., traits only expressed in one sex) where data are available only on same-sex relatives. In the Supporting Information, we present pedigree statistics for traits expressed in

both sexes (tarsus length in both species) and for subsets of the same traits as if they were limited to one sex only (Table S1).

In the REML mixed model framework, the inverse of the Z-linked relatedness matrix can be fitted as a separate random effect, similar to fitting the inverse of the autosomal relatedness matrix when estimating autosomal additive genetic variance. This makes testing for sex linkage and its statistical significance straightforward. The null hypothesis is therefore that of no sex linkage, that is, a pure autosomal inheritance model, whereas the alternative hypothesis is that the trait is sex linked. Hence to assess sex linkage for the different traits, we compared the following two models:

$$H_0: y = \mathbf{X}b + \mathbf{Z_1}a + \mathbf{Z_2}pe + \mathbf{Z_3}m + e, \quad (2)$$

$$H_1: y = \mathbf{X}b + \mathbf{Z_1}a + \mathbf{Z_2}pe + \mathbf{Z_3}m + \mathbf{Z_4}s + e. \quad (3)$$

Most parameters are defined as in equation (1) with the exception of Z_4 and s, which refer to the design matrix and random effects (respectively) of Z chromosome linked inheritance.

Variance components were estimated using ASREML version 3.0 (Gilmour et al. 2009). In cases where estimated variance components were negative, we constrained them to be within the theoretical parameter space (i.e., constraining matrices to be positive semidefinite). Statistical testing of the variance components was done using likelihood ratio tests of H_1 against H_0 (Pinheiro and Bates 2000) and *P*-values multiplied by 0.5 to account for the fact that variances are always positive and hence we are testing on the boundary of the parameter space (Self and Liang 1987).

Results

THE EXTENT OF Z-LINKED GENETIC VARIANCE.

When we used data on homologous traits in both sexes, the estimated h^2_z was zero for nine of the 17 traits examined (Table 2). Moreover, with the exception of wing patch size in collared flycatchers ($h^2_z = 0.40 \pm 0.03$, Table 2) most estimates were small, with Z-linked genetic variance explaining less than 10% of the phenotypic variance. This indicates that there were in general few or no causal polymorphisms located on the Z chromosome and that any causal variants located on the Z chromosome had small effects and/or were present at low frequencies.

IS Z-LINKAGE MORE PRONOUNCED FOR SEXUALLY SELECTED COMPARED TO NATURALLY SELECTED TRAITS?

Theory predicts that genes for sexually selected traits should more often be located on the sex chromosomes (Rice 1984) and we tested this prediction by classifying traits as being either under sexual or natural selection (Table 1; note that not all traits can be reasonably classified) and taking the central tendency of the estimated h^2_z for each of the two trait classes. Although the median h^2_z of sexually selected traits was marginally higher than for naturally selected traits, this was clearly not statistically significant (Fig. 1 , Wilcox rank tests: $W = 24.5$, 95% CI: –0.071, 0.05, $P = 1$). Moreover, when grouping traits according to whether h^2_z was estimated larger than zero and using a Fisher's exact test there was again no indication that sexually selected traits showed a larger degree of sex linkage than naturally selected traits (sexually selected: 3/6, naturally selected: 5/8; Fisher's exact test: odds-ratio 1.61, $P = 1$). Taken together our results suggest that the degree of sex-linked genetic variance need not consistently be higher for sexually selected traits compared to naturally selected traits (see discussion).

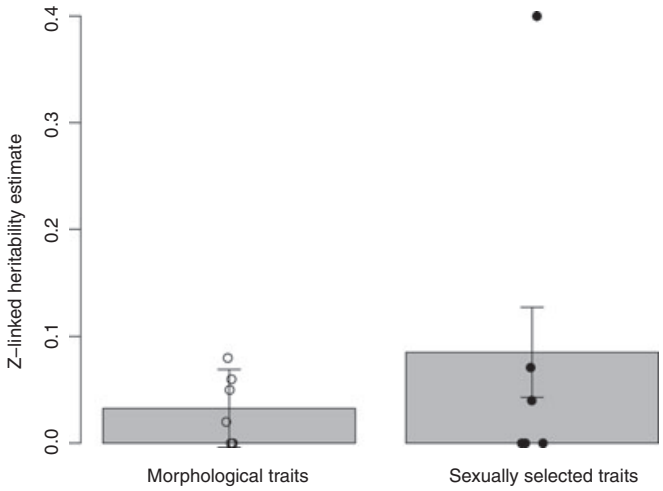

Figure 1. Mean of h^2_z estimates for morphological traits (M) and sexually selected traits (S) estimated using data on both sexes.

DOES THE DEGREE OF Z-LINKED GENETIC VARIANCE CORRELATE WITH INCREASING SEXUAL DIMORPHISM?

Theory also predicts that increasing sexual dimorphism should be associated with higher degree of sex-linked genetic variance (Fairbairn and Roff 2006). Because sexual dimorphism can only be estimated for traits expressed in both sexes, we tested the above prediction by correlating the degree of sexual dimorphism (measured both as SD and as Cohen's *D*, see Methods) with estimated h^2_z. We found no significant association between the degree of sexual dimorphism and estimated h^2_z neither when measuring sexual dimorphism as SD (excluding beak color, $r_s = -0.09$, $n = 9$, $P = 0.81$, Fig. 2A) nor when using Cohens *D* (including beak color, $r_s = 0.21$, $n = 10$, $P = 0.56$, Fig 2B).

Discussion

By taking a large-scale approach using data from two species of birds and measurements on 17 naturally and sexually selected

Table 2. Estimated variance components for the autosomal additive genetic effect (V_A), Z-linked effect (V_Z), and the ratios with phenotypic variance ($h^2_A = V_A/V_P$ and $h^2_Z = V_Z/V_P$). Significant variance components are highlighted in bold. All traits expressed in both sexes are controlled for sex differences in means. For further details of the model and estimated variance components, see Methods section and Table S2.

Trait	Sex	V_A (±SE)	h^2_A (±SE)	V_Z (±SE)	h^2_Z (±SE)	V_P (±SE)	χ^2_1	*P*-value
(a) Collared flycatcher								
Forehead patch	M	**86.49 (11.92)**	**0.37 (0.05)**	0	0	232.19 (9.63)	0	1
Female choice—forehead patch	F	**10.33 (5.06)**	**0.04 (0.02)**	0	0	263.17 (10.38)	0.02	0.44
Female choice—wing patch	F	**16.57 (5.84)**	**0.06 (0.02)**	0	0	277.13 (6.35)	0	0.5
Body mass	F+M	**0.27 (0.03)**	**0.20 (0.02)**	0	0	1.33 (0.05)	0	0.5
Tarsus length	F+M	**0.12 (0.01)**	**0.35 (0.04)**	0.03 (0.02)	**0.08 (0.04)**	0.35 (0.01)	**3.08**	**0.040**
Tail length	F+M	**0.88 (0.10)**	**0.36 (0.04)**	0.12 (0.10)	0.05 (0.04)	2.47 (0.10)	1.26	0.13
Wing patch	F+M	**7.13 (2.07)**	**0.11 (0.03)**	**26.59 (2.57)**	**0.40 (0.03)**	66.87 (1.52)	**107.46**	**0**
Wing length	F+M	**1.03 (0.13)**	**0.28 (0.04)**	0.20 (0.14)	0.05 (0.04)	3.72 (0.12)	2.54	0.056
(b) Zebra finch								
Tarsus length	M+F	**0.18 (0.02)**	**0.52 (0.05)**	0 (0)	0 (0)	0.34 (0.014)	0	1
Wing length	M+F	**1.42 (0.13)**	**0.65 (0.05)**	**0.14 (0.09)**	**0.06 (0.04)**	2.21 (0.09)	**3.46**	**0.03**
Adult mass	M+F	**0.67 (0.10)**	**0.25 (0.03)**	0.05 (0.05)	0.02 (0.02)	2.74 (0.13)	1.2	0.14
Mass day 8	M+F	**0.24 (0.11)**	**0.08 (0.04)**	0 (0)	0 (0)	2.85 (0.11)	0	1
Beak redness	M+F	**0.21 (0.06)**	**0.22 (0.06)**	**0.07 (0.05)**	**0.07 (0.05)**	0.95 (0.07)	**3.53**	**0.03**
Choice activity	F	**22.63 (7.45)**	**0.26 (0.08)**	0 (0)	0 (0)	87.33 (5.07)	0	1
Fecundity in cages	F	5.22 (3.20)	0.12 (0.07)	0 (0)	0 (0)	44.14 (6.48)	0	1
Courtship rate	M	**1.05 (0.26)**	**0.18 (0.04)**	0 (0)	0 (0)	6.04 (0.41)	0	1
Male attractiveness	M	0 (0)	0 (0)	0.001 (0.001)	0.04 (0.04)	0.03 (0.01)	0.70	0.20

traits, we demonstrate here that Z chromosome linked genetic variation was, in general, small (<10%) for most traits (Table 2). However, wing patch size in collared flycatchers (Fig. 3) showed a relatively large Z-linked genetic variance and, interestingly, this trait is known to be under sexual selection. Three more traits (beak color and wing length in zebra finches and tarsus length in collared flycatchers) showed low, but significant Z-linked genetic variation. Of these traits beak color in zebra finches is presumably under sexual selection at least in some populations and/or it has been under sexual selection in the past as suggested by the sexual dimorphism in this trait (see Schielzeth et al. 2012 for discussion).

There was no statistically significant relationship between the amount of sex-linked genetic variance and the level of sexual dimorphism (Fig. 2). Our analyses provide a rare large-scale test of the theoretical predictions for the genomic location of segregating sexually antagonistic loci influencing sexual dimorphism. These are in contrast to conclusions obtained from reciprocal line crosses (Reinhold 1998) but support other studies (e.g., Fitzpatrick 2004) that fail to find support for the theoretical expectations of sex chromosome linked genetic variation of antagonistic loci. Below we discuss our findings and potential reasons for the difference in conclusions reached with different approaches when testing for sex chromosome linkage of sexually antagonistic loci.

THE EXTENT OF SEX-LINKED GENETIC VARIANCE

Most estimates of sex linkage have come from reciprocal crosses while estimates of sex-linked genetic variance within populations are rare. Two recent studies have estimated standing genetic variation on the sex chromosome limited to one sex. In guppies, Y-linked genetic variance for different male ornamentation traits explained between 2% and 23% of the phenotypic variance (Postma et al. 2011), whereas for zebra finch beak color 7% of the phenotypic variation is explained by the maternal line that include genetic variation on W and extranuclear variants (Schielzeth et al. 2012b). A few other studies have estimated standing genetic variation on the sex chromosome that is not limited to one sex (as in our study): 13% of the phenotypic variance in a female ornament in barn owls was due to variation in genes located on the Z chromosome (Roulin et al. 2010) and in humans, Pan et al. (2007) found evidence for X-linked genetic variance in seven different morphological and physiological traits, including human height ($h^2_X = 0.09$) and a measure of lipoprotein A ($h^2_X = 0.47$).

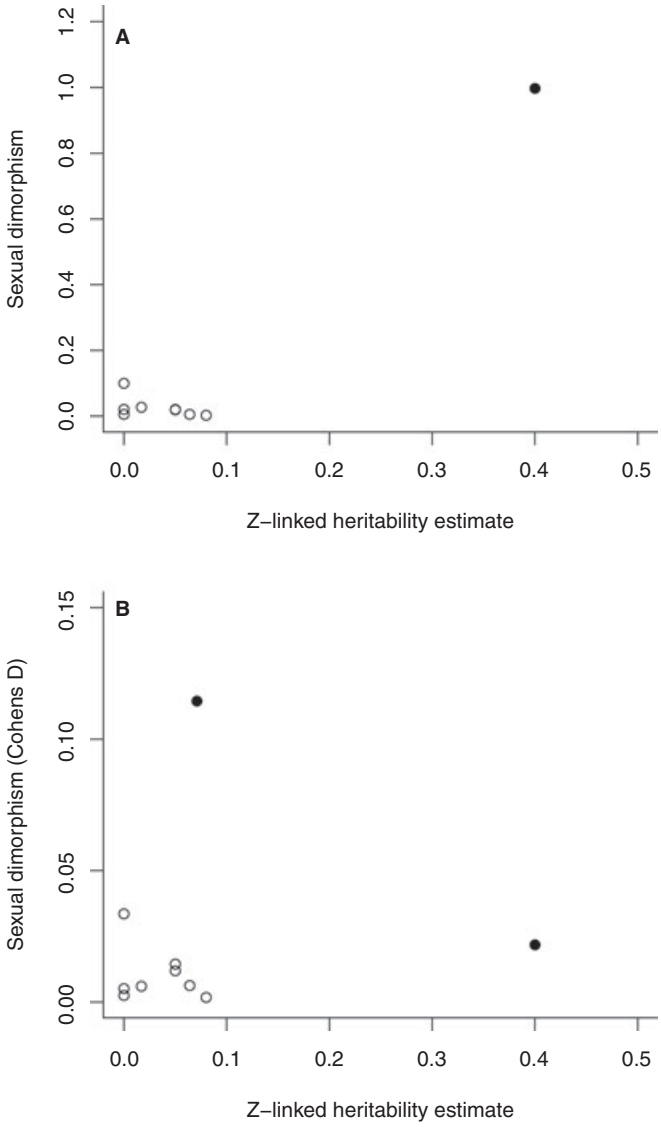

Figure 2. Estimated Z-linked heritability (h^2_Z) plotted against sexual dimorphism in the trait estimated as SD (A) and Cohen's D (B). Solid circles are sexually selected traits (see Table 1) and open circles are morphological traits. Note that this figure is limited to traits expressed in both sexes.

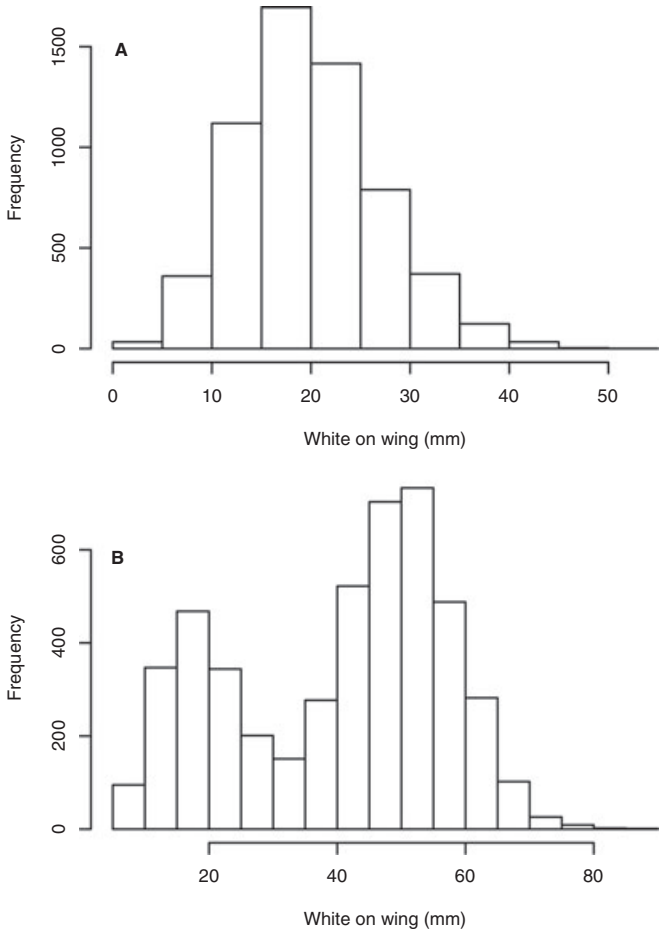

Figure 3. Phenotypic distribution of wing patch size in collared flycatcher females (A) and males (B). Note that the bimodality in males is largely an age effect (young males have smaller ornament than older males).

The proportion of phenotypic variation explained by sex chromosome linked genes in the above studies is similar to that found in our study where h^2_Z ranged from 0% to 40%, although most estimates being around 0–10% (Table 2). Most estimates were not statistically different from zero and of the morphological traits examined we only found statistically significant support for Z-linkage of wing length ($h^2_Z = 0.06 \pm 0.039$, $P = 0.031$) and beak redness ($h^2_Z = 0.071 \pm 0.047$, $P = 0.030$) in the zebra finch and tarsus length ($h^2_Z = 0.08 \pm 0.04$, $P = 0.040$) and wing patch size ($h^2_Z = 0.40 \pm 0.03$, $P < 0.001$) in the collared flycatcher. Wing patch size in collared flycatchers is under sexual selection (Sheldon and Ellegren 1999; de Heij et al. 2011), and the male mean size of the ornament is approximately twice that of females (Fig. 3). This might be in line with a sex-linked gene in a system with no dosage compensation, which seems to be lacking in birds, or only occur on a gene-by-gene basis (Ellegren et al. 2007; Wolf and Bryk 2011). Because the ornament is produced by lack of melanin production, it is possible that a Z-linked melanin inhibitor is present in this species that give rise to the observed sexual dimorphism in this trait.

We found no indication of sex-linked genetic variation for another sexually selected trait in the collared flycatcher; the size of the male-limited forehead patch (Qvarnström et al. 2006). For this trait, all segregating variation seems to be located on the autosomes ($h^2_A = 0.37$). However, as discussed in the Introduction this does not necessarily mean that there are no genes with potential effect on forehead patch size on the Z chromosome. Fixed genes on the Z chromosome that have no allelic variation may still contribute to the expression of this trait, but would not be detectable with our approach. Indeed a previous study in this species

has found support for Z-linkage of male forehead patch size in a species comparison (Sætre et al. 2003). Moreover, Sætre et al. also did not find support for Z-genotype to predict the size of the wing patch. It seems therefore that there are both fixed Z-linked alleles and segregating genetic variants on the autosomes that determine the size of the forehead patch in male collared flycatchers whereas segregating genes on the Z chromosome clearly influence wing patch size in this species.

Z-linkage of forehead patch size has also been suggested in a within-population study on the closely related pied flycatcher (*F. hypoleuca*). In this species, heritability of forehead patch size was higher between paternal grandfather and grandson than the maternal grandfather and grandson and, second, maternal half-brothers were more similar than paternal half-brothers (Potti and Canal 2011).

Some independent support for the relatively high h^2_z estimate of zebra finch wing length comes from a recent QTL scan in the same population. Schielzeth et al. (2012a) found one significant QTL and five suggestive QTLs that explained in sum approximately 36% of the genetic variance in wing length. Although the significant QTLs were not located on the Z chromosome, the estimated variance components and likelihood ratios were generally high across the entire Z chromosome even if it did not reach the significance threshold (see Fig. 1 in Schielzeth et al. 2012a).

SEX LINKAGE GENETIC VARIANCE, SEXUAL SELECTION, AND SEXUAL DIMORPHISM

Our within-population study of sex-linked genetic variance did not reveal an overrepresentation of sex-linked genetic variance for sexually selected traits compared to morphological traits, although there was a weak trend for sexually selected traits to have more genetic variance located on the Z chromosome (Fig. 1). A study on *Drosophila* (Fitzpatrick 2004) also did not find loci affecting sexually selected traits to be overrepresented on the sex chromosomes. Although sex linkage of sexually selected traits seems particularly prevalent in some groups, such as guppies (reviewed in Lindhom & Breden 2002), the empirical support for this prediction is still low. One possible interpretation of this is that genes that have the potential to solve sexual conflicts are strongly favored by selection and therefore relatively quickly become fixed within populations, thereby eliminating standing genetic variation. Because the X/Z chromosome has a smaller effective population size than autosomes (Whitlock and Wade 1995) this chromosome is expected to typically contain relatively low levels of standing genetic variation. Thus, the evolution of sexual size dimorphism may proceed at different rates across the genome whereby favorable genes go to fixation faster on the sex chromosome. This might also explain why crosses of divergent populations tend to find evidence of sex linkage (Reinhold 1998). However, given the relative scarcity of empirical studies it seems too early to conclude about the generality and nature of sex linkage of sexually selected traits.

Fairbairn and Roff (2006) predicted that sexual dimorphism should correlate positively with sex-linked genetic variance on the basis that once sexually antagonistic loci are sex linked this should facilitate increasing dimorphism. We did not find any support for this prediction in our data however (Fig. 2). Although a positive correlation between sex-linked genetic variation and sexual dimorphism might be expected, it seems unlikely that this should be a strong relationship. The molecular genetic underpinnings of sexual dimorphism has been well studied in *Drosophila* and in this species it seems to be largely autosomal-linked loci that lead to the expression of sexually dimorphic phenotypes such as, for example, abdominal bristle pigmentation (Williams and Carroll 2009). Moreover, gene expression studies that have examined the genomic location of sex-biased genes (which presumably are under sexual antagonistic selection) have had mixed success demonstrating that such genes are predominately sex linked (Mank 2009). Thus, although the conditions under which sexually antagonistic loci should be sex linked are clear from a theoretic point of view (Fisher 1931, Rice 1984), more empirical studies are needed to demonstrate the generality of these predictions.

POWER TO SEPARATE AUTOSOMAL AND Z-LINKED INHERITANCE PATTERNS

Coefficients of coancestry between individuals for loci on the sex chromosomes only differ from those on autosomal loci for some types of relatives (see Lynch and Walsh 1998; Grossman & Eisen 1989). As a result, separating autosomal and sex chromosome inheritance require large sample sizes and ideally specific pedigree designs to maximize power (Fairbairn and Roff 2006). This includes crosses to obtain both full sibs, half sibs, and single and double first cousins (see Fig. 3 in Fairbairn and Roff 2006). Although such designs have been shown to have good power for detecting sex linkage (Meyer 2008), it is often difficult or even impossible to implement for many study organisms. Our analyses rely on the occurrence of classes of relatives for which coefficient of relatedness differs for autosomal and sex-linked genes, which allows us to separate autosomal- and sex-linked effects. One plausible reason for the relatively low support for sex linkage could be due to low power to separate autosomal and sex chromosomal inheritance. Nevertheless, at least for traits expressed in both sexes, we have relatively large number of relatives that are informative also for sex-linked inheritance (see Table S1). The possibility to separate autosomal- and sex-linked variance in our study is also demonstrated by the fact that we did find support for significant and, in some cases substantial, sex chromosome linkage in both systems (Table 2) despite large differences in

pedigree structure. For traits with sex-limited expression, however, power is likely low to detect Z-linkage and for these traits our results should be interpreted with caution and not as refuting Z-linkage.

Conclusion

Here, we have provided a detailed and comprehensive analysis of the degree to which genetic variation in naturally and sexually selected traits are sex linked using two different bird species with contrasting population history and pedigree structure. Our results show that sex-linked genetic variance was modest, although we did find statistical support for sex-linked genetic variants segregating for wing length and beak color in the zebra finch and for wing patch size and tarsus length in the collared flycatcher. A possible explanation for the absence of a consistent higher degree of sex-linked genetic variance for sexually selected traits could be that favorable genes go to fixation faster on the sex chromosome (thereby removing standing genetic variation). However, given the relative scarcity of empirical studies, it seems too early to conclude about the generality and nature of sex linkage of sexually selected traits. Our results have substantially increased the number of traits for which Z-linkage has been estimated thereby contributing to a better understanding of the extent of Z chromosome linked genetic variance of quantitative traits. Our study also demonstrates the feasibility of using long-term pedigree data to separate autosomal and sex chromosomal patterns of inheritance, although this require well-connected pedigrees. Quantifying the extent to which genetic variance is located on the autosomes or the sex chromosomes can also aid researcher who plan to develop molecular markers for linkage and/or association mapping. If there are indications of sex linkage, one should ensure that also the sex chromosomes have a sufficiently high marker density to detect potential QTLs.

Moreover, we highlight that birds are an ideal group in which to study sex linkage due to the lack of dosage compensation and hope that the work presented will stimulate further work on sex chromosome linkage in other pedigreed populations.

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LITERATURE CITED

Arnqvist, G., and L. Rowe. 2005, Sexual conflict. Princeton Univ. Press, Princeton, NJ.

Bonduriansky, R. 2007. The genetic architecture of sexual dimorphism: the potential roles of genomic imprinting and condition dependence, Pp. 176–184 *in* D. J. Fairbairn, W. U. Blanckenhorn, and T. Szekely, eds. Sex, size and gender roles: evolutionary studies of sexual size dimorphism. Oxford Univ. Press, Oxford, U.K.

Charmantier, A., and D. Réale. 2005. How do misassigned paternities affect the estimation of heritability in the wild? Mol. Ecol. 14:2839–2850.

Connallon, T., and L. L. Knowles. 2005. Intergenomic conflict revealed by patterns of sex-biased gene expression. Trends Genet. 21:495–499.

Cowley, D. E., W. R. Atchley, and J. J. Rutledge. 1986. Quantitative genetics of *Drosophila melanogaster* 1. Sexual dimorphism in genetic parameters for wing traits. Genetics 114:549–566.

de Heij, M. E., L. Gustafsson, and J. E. Brommer. 2011. Experimental manipulation shows that the white wing patch in collared flycatchers is a male sexual ornament. Ecol. Evol. 1:546–555.

Ellegren, H., and J. Parsch. 2007. The evolution of sex-biased genes and sex-biased gene expression. Nat. Rev. Genet. 8:689–698.

Ellegren, H., L. Hultin-Rosenberg, B. Brunström, L. Dencker, K. Kultima, and B. Scholz. 2007. Faced with inequality: chicken do not have a general dosage compensation of sex-linked genes. BMC Biol. 5: doi:10.1186/1741-7007-5-40.

Fairbairn, D. J., and D. A. Roff. 2006. The quantitative genetics of sexual dimorphism: assessing the importance of sex-linkage. Heredity 97: 319–328.

Fairbairn, D. J., W. U. Blanckenhorn, and T. Szekely. 2007. Sex, size and geneder roles: evolutionary studies of sexual size dimorphism. Oxford Univ. Press, Oxford, New York.

Fernando, R. L., and M. Grossman. 1990. Genetic evaluation with autosomal and X-chromosomal inheritance. Theor. Appl. Genet. 80:75–80.

Fisher, R. A. 1931. The evolution of dominance. Biol. Rev. Biol. Proc. Camb. Philos. Soc. 6:345–368.

Fitzpatrick, M. J. 2004. Pleiotropy and the genomic location of sexually selected genes. Am. Nat. 163:800–808.

Foerster, K., T. Coulson, B. C. Sheldon, J. M. Pemberton, T. H. Clutton-Brock, and L. E. B. Kruuk. 2007. Sexually antagonistic genetic variation for fitness in red deer. Nature 447:1107-U1109.

Forstmeier, W., K. Martin, E. Bolund, H. Schielzeth, and B. Kempenaers. 2011. Female extrapair mating behavior can evolve via indirect selection on males. Proc. Natl. Acad. Sci. USA 108:10608–10613.

Forstmeier, W., H. Schielzeth, J. C. Mueller, H. Ellegren, and B. Kempenaers. 2012. Heterozygosity-fitness correlations in zebra finches: microsatellite markers can be better than their reputation. Mol. Ecol. 21:3237–3249.

Gilmour, A. R., B. J. Gogel, B. R. Cullis, S. J. Welham, and R. Thompson. 2009. ASReml user guide. Relase 3.0.VSN International. Hemel Hempstead, U.K.

Grossman, M., and E. J. Eisen. 1989. Inbreeding, coancestry and covariance between relatives for X-chromosomal loci. J. Hered. 80:137–142.

Gustafsson, L. 1986. Lifetime reproductive success and heritability—empirical support for Fisher's fundamental theorem. Am. Nat. 128: 761–764.

———. 1989. Collared flycatcher, Pp. 75–88 *in* I. Newton, ed. Lifetime reproduction in birds. Academic Press, London.

Henderson, C. R. 1950. Estimation of genetic parameters. Ann. Math. Stat. 21:309–310.

Husby, A., M. E. Visser, and L. E. B. Kruuk. 2011. Speeding up microevolution: the effects of increasing temperature on selection and genetic variance in a wild bird population. Plos Biol. 9: e1000585.

Kirkpatrick, M., and D. W. Hall. 2004. Sexual selection and sex linkage. Evolution 58:683–691.

Kruuk, L. E. B. 2004. Estimating genetic parameters in natural populations using the 'animal model'. Philos. Transac. R. Soc. Lond. Ser. B Biol. Sci. 359:873–890.

Kruuk, L. E. B., T. H. Clutton-Brock, J. Slate, J. M. Pemberton, S. Brotherstone, and F. E. Guinness. 2000. Heritability of fitness in a wild mammal population. Proc. Natl. Acad. Sci. USA 97:698–703.

Kruuk, L. E. B., B. C. Sheldon, and J. Merilä. 2002. Severe inbreeding depression in collared flycatchers (*Ficedula albicollis*). Proc. R. Soc. Lond. Ser. B Biol. Sci. 269:1581–1589.

Lindholm, A., and F. Breden. 2002. Sex chromosomes and sexual selection in poeciliid fishes. Am. Nat. 160:S214-S224.

Lynch, M., and B. Walsh. 1998. Genetics and analysis of quantitative traits. Sinauer, Sunderland, MA.

Mank, J. E. 2009. Sex chromosomes and the evolution of sexual dimorphism: lessons from the genome. Am. Nat. 173:141–150.

Mank, J. E., and H. Ellegren. 2009. Sex-linkage of sexually antagonistic genes is predicted by female, but not male, effects in birds. Evolution 63:1464–1472.

Mank, J. E., D. J. Hosken, and N. Wedell. 2011. Some inconvenient truths about sex chromosome dosage compensation and the potential role of sexual conflict. Evolution 65:2133–2144.

Meyer, K. 2008. Likelihood calculations to evaluate experimental designs to estimate genetic variances. Heredity 101:212–221.

Morrissey, M. B., and A. J. Wilson. 2010. pedantics: an R package for pedigree-based genetic simulation and pedigree manipulation, characterization and viewing. Mol. Ecol. Res. 10:711–719.

Nakagawa, S., and I. C. Cuthill. 2007. Effect size, confidence interval and statistical significance: a practical guide for biologists. Biol. Rev. 82: 591–605.

Pan, L., C. Ober, and M. Abney. 2007. Heritability estimation of sex-specific effects on human quantitative traits. Genet. Epidemiol. 31: 338–347.

Pinheiro, J. C., and D. Bates. 2000, Mixed-effects models in S and S-Plus. Springer, New York.

Poissant, J., A. J. Wilson, and D. W. Coltman. 2010. Sex-specific genetic variance and the evolution of sexual dimorphism: a systematic review of cross sex genetic correlations. Evolution 64:97–107.

Postma, E., N. Spyrou, L. A. Rollins, and R. C. Brooks. 2011. Sex-dependent selection differentially shapes genetic variation on and off the guppy Y chromosome. Evolution 65:2145–2156.

Potti, J., and D. Canal. 2011. Heritability and genetic correlation between the sexes in a songbird sexual ornament. Heredity 106:945–954.

Qvarnström, A., J. E. Brommer, and L. Gustafsson. 2006. Testing the genetics underlying the co-evolution of mate choice and ornament in the wild. Nature 441:84–86.

Reinhold, K. 1998. Sex linkage among genes controlling sexually selected traits. Behav. Ecol. Sociobiol. 44:1–7.

Rice, W. R. 1984. Sex chromosomes and the evolution of sexual dimorphism. Evolution 38:735–742.

Roulin, A., R. Altwegg, H. Jensen, I. Steinsland, and M. Schaub. 2010. Sex-dependent selection on an autosomal melanic female ornament promotes the evolution of sex ratio bias. Ecol. Lett. 13:616–626.

Sætre, G. P., T. Borge, K. Lindroos, J. Haavie, B. C. Sheldon, C. Primmer, and A. C. Syvanen. 2003. Sex chromosome evolution and speciation in Ficedula flycatchers. Proc. R. Soc. Lond. Ser. B Biol. Sci. 270: 53–59.

Schielzeth, H., and E. Bolund. 2010. Patterns of conspecific brood parasitism in zebra finches. Anim. Behav. 79:1329–1337.

Schielzeth, H., E. Bolund, B. Kempenaers, and W. Forstmeier. 2011. Quantitative genetics and fitness consequences of novelty-seeking in zebra finches. Behav. Ecol. 22:126–134.

Schielzeth, H., W. Forstmeier, B. Kempenaers, and H. Ellegren. 2012a. QTL mapping of wing length in zebra finch using genome-wide single nucleotide polymorphisms markers. Mol. Ecol. 21:229–239.

Schielzeth, H., B. Kempenaers, H. Ellegren, and W. Forstmeier. 2012b. QTL linkage mapping of zebra finch beak colour shows an oligogenic control of a sexually selected trait. Evolution 66:18–30.

Self, S., and K. Y. Liang. 1987. Asymptotic properties of maximum likelihood estimators and likelihood ratio tests under nonstandard conditions. J. Am. Stat. Assoc. 82:605–610.

Sheldon, B. C., and H. Ellegren. 1999. Sexual selection resulting from extra-pair paternity in collared flycatchers. Anim. Behav. 57:285–298.

Whitlock, M., and M. J. Wade. 1995. Speciation–founder events and their effects on X-linked and autosomal loci. Am. Nat. 145:676–685.

Williams, T. M., and S. B. Carroll. 2009. Genetic and molecular insights into the development and evolution of sexual dimorphism. Nat. Rev. Genet. 10:797–804.

Wolf, J. B. W., and J. Bryk. 2011. General lack of global dosage compensation in ZZ/ZW systems? Broadening the perspective with RNA-seq. BMC Genom. 12: doi:10.1186/1471-2164-12-91.

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Supporting Information

Additional Supporting information may be found in the online version of this article at the publisher's website:

Table S1. Pruned pedigree statistics for tarsus length when including records from both sexes, females only, and males only in the collared flycatcher (a) and the zebra finch (b).

Table S2. Estimated variance components of the full models for components not reported in main text.