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INTRALOCUS SEXUAL CONFLICT AND THE GENETIC ARCHITECTURE OF SEXUALLY DIMORPHIC TRAITS IN *PROCHYLIZA XANTHOSTOMA* (DIPTERA: PIOPHILIDAE)

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Abstract.—Because homologous traits of males and females are likely to have a common genetic basis, sex-specific selection (often resulting from sexual selection on one sex) may generate an evolutionary tug-of-war known as intralocus sexual conflict, which will constrain the adaptive divergence of the sexes. Theory suggests that intralocus sexual conflict can be mitigated through reduction of the intersexual genetic correlation (r_{MF}), predicting negative covariation between r_{MF} and sexual dimorphism. In addition, recent work showed that selection should favor reduced expression of alleles inherited from the opposite-sex parent (intersexual inheritance) in traits subject to intralocus sexual conflict. For traits under sexual selection in males, this should be manifested either in reduced maternal heritability or, when conflict is severe, in reduced heritability through the opposite-sex parent in offspring of both sexes. However, because we do not know how far these hypothesized evolutionary responses can actually proceed, the importance of intralocus sexual conflict as a long-term constraint on adaptive evolution remains unclear. In this study, we investigated the genetic architecture of sexual and nonsexual morphological traits in *Prochyliza xanthostoma*. The lowest r_{MF} and greatest dimorphism were exhibited by two sexual traits (head length and antenna length) and, among all traits, the degree of sexual dimorphism was correlated negatively with r_{MF}. Moreover, sexual traits exhibited reduced maternal heritabilities, and the most strongly dimorphic sexual trait (antenna length) was heritable only through the same-sex parent in offspring of both sexes. Our results support theory and suggest that intralocus sexual conflict can be resolved substantially by genomic adaptation. Further work is required to identify the proximate mechanisms underlying these patterns.

Key words.—Heritability, intersexual genetic correlation, intralocus sexual conflict, sex-specific selection, sexual dimorphism, sexual selection, sexually antagonistic selection.

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Theory suggests that intralocus sexual conflict is a pervasive consequence of sex-specific selection in sexually reproducing species (Fisher 1930a,b, 1931; Lande 1980; Rice and Chippindale 2001). This idea is supported by experimental evidence of such conflict in *Drosophila melanogaster* (Chippindale et al. 2001; Rand et al. 2001). Intralocus sexual conflict is mitigated through the independent evolution of the sexes toward their sex-specific phenotypic optima, resulting in sexual dimorphism (Fisher 1930a, 1931; Lande 1980). However, much remains to be learned about the genetic mechanisms involved in the evolution of sexual dimorphism and the extent to which they can resolve the conflict (Rhen 2000; Rice and Chippindale 2001, 2002; Day and Bonduriansky 2004; Bonduriansky and Rowe 2005). Consequently, it is not clear whether intralocus sexual conflict represents a transient constraint or a permanent and severe impediment to the adaptive divergence of the sexes.

Building on ideas developed by Fisher (1930a,b, 1931), Lande (1980, 1987) showed that sex-specific selection may result in a displacement of both sexes from their phenotypic optima because homologous traits of males and females tend to be affected by similar sets of genes (i.e., the intersexual genetic correlation, r_{MF}, tends to be high). When r_{MF} is high, sexual selection on a trait in males (i.e., a sexual trait that functions in competition for mates) will affect the phenotypic means of both sexes, and this process will continue until net selection on males is balanced by opposing viability selection on females, with neither sex at its phenotypic optimum. Although Lande treated r_{MF} as a constant, it is clear that genetic correlations can evolve under selection (Cheverud 1984; Archer et al. 2003; Phelan et al. 2003). By showing that r_{MF} impedes the evolution of sexual dimorphism, the work of Lande and Fisher establishes r_{MF} as a target of selection on genetic architecture (i.e., the genetic basis of the mapping between genotype and phenotype) of traits subject to intralocus sexual conflict. Selection should reduce r_{MF} in such traits because this will allow the sexes to evolve further or more rapidly toward their sex-specific phenotypic optima. This hypothesis also predicts negative covariation between r_{MF} and the degree of sexual dimorphism, because stronger sexual selection should favor a greater reduction in r_{MF} and more pronounced dimorphism.

Nonetheless, the evolution of r_{MF} remains a contentious issue. Lande (1980, 1987) argued that r_{MF} should be near zero in traits exhibiting advanced or extreme sexual dimorphism. In contrast, a simulation suggested that r_{MF} may decrease only slightly and transiently as sexual dimorphism evolves (Reeve and Fairbairn 2001). However, Reeve and Fairbairn (2001) allowed change in allele frequencies only, constraining the genetic architecture to remain constant. As they pointed out, allowing for changes in genetic architecture (e.g., evolution of novel mechanisms of sex-specific epistasis) may lead to different results. Empirical research has shown that sexually dimorphic traits can exhibit a wide range of r_{MF} values (Cowley and Atchley 1988; Simmons and Ward 1991; Price and Burley 1993; Merilä et al. 1998; Chenoweth and Blows 2003), so the evidence remains equivocal.

Moreover, new theory has shown that intralocus sexual

conflict may persist, despite a reduction in r_{MF}, as a result of intersexual inheritance of genes affecting traits subject to the conflict (Day and Bonduriansky 2004). This is because sex-specific selection results in a higher probability of inheriting high-fitness alleles from the same-sex parent than from the opposite-sex parent in one or both sexes. For a trait under sexual selection in males, a male offspring will be more likely to inherit a high male-fitness allele from his father, who succeeded in male sexual competition, than from his mother, who did not experience sexual selection. In other words, paternally inherited alleles will confer higher male fitness, on average, because sexual selection will remove the most unfavorable male genotypes from the breeding population in each generation (even though some paternally inherited alleles may be passed down from the paternal grandmother). This situation pertains even to loci that are male limited in expression because mutation, gene flow, or drift will cause some females to pass deleterious alleles to their male offspring. Thus, in such traits, the expression of maternally inherited alleles (i.e., maternal heritability) should be reduced through selection in male offspring. However, if genetic constraints prevent the evolution of reduced maternal heritability in offspring of one sex only, selection may favor reduced maternal heritability in offspring of both sexes if the benefit to males exceeds the cost to females. Conversely, for a trait under strong, opposing directional selection in both sexes (i.e., sexually antagonistic selection; Rice 1984; Rice and Chippindale 2001) resulting in severe conflict, a female will also be more likely to inherit a high female-fitness allele from her mother, who passed the tests of female-specific selection. In such traits, selection should favor reduced heritability through the opposite-sex parent in offspring of both sexes (Day and Bonduriansky 2004).

Parent-of-origin effects on gene expression (i.e., genomic imprinting in the broad sense applied to *Drosophila*: Golic et al. 1998; Lloyd et al. 1999; Lloyd 2000; mammals: Moore and Haig 1991; Moore 2001; birds: Tuiskula-Haavisto et al. 2004; and plants: Alleman and Doctor 2000) can produce differential heritability through the mother and father (Spencer 2002) and, thus, offer a potential proximate solution to this evolutionary problem (Day and Bonduriansky 2004). The fitness advantage conferred by genomic imprinting is proportional to the genetic variance at loci affecting the expression of the trait subject to sex-specific selection. However, as long as variation is maintained at these loci (e.g., by mutation, drift, gene flow, or sexually antagonistic selection), genomic imprinting is predicted to evolve under a broad range of conditions, including various forms of selection on the two sexes (see above) and different patterns of dominance (Day and Bonduriansky 2004). Note, however, that the above predictions reflect fitness maximization strategies and, thus, are consistent with any mechanism that can yield the predicted patterns.

These predictions apply broadly to loci that experience sex-specific selection and are present in the genomes of both sexes, segregating on the autosomes or the X or Z sex chromosome. Such genes may be numerous because the sexes pursue different reproductive strategies but share much of the genome. However, the predicted genetic modifications are most likely to evolve in the smaller subset of genes subject to very different selection pressures in the two sexes, such as genes targeted by sexual selection in one sex.

The relation between r_{MF} and heritability can be expressed as

$$r_{MF} = \sqrt{\frac{h^2_{FD} \cdot h^2_{MS}}{h^2_{MD} \cdot h^2_{FS}}}. \quad (1)$$

where h^2 represents heritability calculated from father-daughter (FD), mother-son (MS), mother-daughter (MD), and father-son (FS) covariances (Becker 1992; Lynch and Walsh 1998). r_{MF} may be reduced through a divergence in the sets of loci involved in trait expression in the two sexes, which could reflect the evolution of sex linkage (Rice 1984, 1987; Reinhold 1998; Roldan and Gomendio 1999; Rice and Chippindale 2002), the duplication and subsequent sex limitation of autosomal loci (Rhen 2000; Rice and Chippindale 2002), the evolution of condition dependence (Bonduriansky and Rowe 2005), or other epistatic mechanisms with sex-specific effects. These adaptations may mitigate intralocus sexual conflict. However, as long as some loci under sex-specific selection (including sex-limited loci) continue to be inherited from the opposite-sex parent, intralocus sexual conflict will persist to some degree, selecting for parent-of-origin effects that reduce intersexual inheritance at those loci (Day and Bonduriansky 2004). If this results in reduced heritability through the opposite-sex parent in offspring of both sexes (as expected for traits under strong sexually antagonistic selection) or reduced maternal heritability in male offspring only, then r_{MF} will also be reduced. However, if maternal heritability is reduced in offspring of both sexes, then r_{MF} may remain unchanged (eq. 1).

The four heritabilities (h^2_{FD}, h^2_{MS}, h^2_{FS}, h^2_{MD}) thus contain more information than r_{MF} and can be used to test the theory of Day and Bonduriansky (2004). Reduced heritability through the opposite-sex parent is a necessary facet of the reduction in r_{MF} (eq. 1) and, thus, follows from the work of Fisher and Lande. However, the theory of Day and Bonduriansky (2004) yields novel predictions. First, for traits under sexual selection in males, selection should target the maternal heritability most directly, resulting in reduced maternal heritability and correspondingly strengthened paternal heritability in such traits, at least in male offspring. This prediction does not follow from previous work because reductions in r_{MF} can be achieved through either reduced maternal heritability in male offspring, reduced paternal heritability in female offspring, or both. Second, under some circumstances (see above), selection is predicted to favor reduced maternal heritability in offspring of both sexes. This prediction does not follow from previous work because, in this case, intersexual inheritance may be reduced without concomitant reduction in r_{MF}. Thus, although it is compatible with the work of Lande (1980, 1987), the theory of Day and Bonduriansky (2004) yields distinct and testable predictions.

We tested these predictions for seven shape traits (i.e., linear dimensions corrected for body size) and body size in the fly *Prochyliza xanthostoma*. The shape traits included four sexual traits used directly in male combat and courtship (Bonduriansky 2003; Bonduriansky and Rowe 2003) and, hence, subject to sex-specific selection, and three nonsexual traits that play no direct role in male sexual competition and prob-

TABLE 1. Function, mean size (mm × 100) with standard deviation (SD), and degree of sexual dimorphism for body size (thorax length) and seven body shape traits in *Prochyliza xanthostoma*.

Trait	Females			Males			Sexual dimorphism
	Function	Mean size	SD	Function	Mean size	SD	
Body size (TL)	—	171	4.28	—	156	5.10	0.081
Head length (HL)	nonsexual	104	2.88	sexual	124	5.61	0.305
Head width (HW)	nonsexual	104	2.36	sexual	84	2.53	0.117
Antenna length (AL)	nonsexual	61	2.33	sexual	98	4.76	0.761
Foretibia length (FL)	nonsexual	95	2.51	sexual	104	3.22	0.203
Midtibia length (ML)	nonsexual	103	2.74	nonsexual	113	3.59	0.201
Wing-vein length (WL)	nonsexual	195	6.33	nonsexual	175	7.27	0.022
Intersetal width (IS)	nonsexual	84	2.53	nonsexual	72	2.82	0.082

ably experience more similar selection in the two sexes (Table 1). We also used variation among sexual traits in the degree of sexual dimorphism to infer variation in the severity of intralocus sexual conflict, assuming that the most pronounced sexual dimorphism will occur in traits presently or formerly under the strongest sexually antagonistic selection, whereas weaker dimorphism may characterize traits subject to directional selection in males only. Thus, we predicted that the most dimorphic sexual trait(s) would exhibit reduced heritability through the opposite-sex parent in offspring of both sexes, whereas less strongly dimorphic sexual traits would exhibit reduced maternal heritability in male offspring only or in offspring of both sexes. For nonsexual traits, we predicted similar heritabilities through the mother and father. Among all traits, we predicted a negative correlation between r_{MF} and sexual dimorphism. We also examined coefficients of additive genetic variance (CV_A) and residual variance (CV_R) for each trait.

MATERIALS AND METHODS

Rearing of Flies

We collected gravid females of *P. xanthostoma* (Walker) from carcasses of moose (*Alces alces*) at the Wildlife Research Station in Algonquin Park, Ontario, Canada, and transferred them to 1.5-l cages with mesh windows. Cages contained water, sugar, and petri dishes with larval medium (extralean organic ground beef, aged at room temperature for about 5 days) for oviposition. When the larvae from these dishes were ready to pupate (i.e., they left the dishes in response to misting with water), they were transferred individually to mesh-covered shell vials containing soil. Emerging adults were transferred in same-sex groups of about 10 to 1.5-l cages containing water, sugar, and ground beef. At age 10 days, females were paired with randomly selected males in glass cages (3-cm diameter × 9-cm height) containing water, sugar, and a punctured Eppendorf tube with larval medium.

We provided the F_1 and F_2 descendants of these flies with standardized quantities of larval medium to minimize environmental variation and increase power to detect genetic effects (see Simmons and Ward 1991). From the first clutch of each of 20 females, we transferred 40 randomly selected first-instar larvae to a petri dish containing 5 g of larval medium placed on a layer of soil inside a mesh-covered cup. Dishes were misted daily until the larvae had pupated, and pupae were transferred individually to shell vials. At about age 10 days, randomly selected adult flies (parents) were paired with nonsibling partners in separate cages (see above) containing larval medium. Each male-female pair represented a different combination of families. Males were frozen after 24 h, and females were frozen after laying their first clutch. From each brood, we randomly selected 40 first-instar larvae (where possible) and provided them with 5 g of larval medium (see above). Adults (offspring) were frozen 2 to 4 days following emergence (i.e., once their exoskeletons were fully sclerotized).

Morphometric and Quantitative Genetic Analysis

Although several broods contained fewer than 40 larvae, all broods received excess food and there was no evidence of an effect of number of larvae transferred on survivorship or mean adult body size ($P > 0.5$ for both tests). In total, 58 broods yielded at least five adult offspring of each sex. All parents and five randomly selected adults of each sex from each brood were glued to entomological pins by the right mesopleuron. We measured each fly's thorax length (TL), head length (HL), and width (HW), antenna length (AL), foretibia length (FL), midtibia length (ML), the length of the R_{4+5} wing-vein from the r–m cross-vein to the wing margin (WL), and the distance between the bases of the presutural intra-alar setae (IS) (see fig. 1 in Bonduriansky and Rowe 2005), using a dissecting microscope with an ocular micrometer (Table 1). Our quantitative genetic analysis is based on relative trait sizes (i.e., shape) because the use of absolute trait sizes would confound the genetic architectures of body shape and body size (see Discussion). We used thorax length as an index of body size because this trait loads most strongly on PC1 (Bonduriansky and Rowe 2003) and calculated relative trait sizes as standardized residuals from least-squares regressions of trait size on thorax length for each trait, performed separately by sex with parents and offspring pooled. Although measurement error is similar on both axes (so that, technically, reduced major axis regression is more appropriate), least-squares residuals are more clearly interpretable as deviations in trait size from the expectation at a given body size. Moreover, because all traits scale tightly with thorax length (see fig. 3 in Bonduriansky and Rowe 2005), the difference between least-squares and reduced major axis regressions is negligible. Thorax length is henceforth referred to as ''body size,'' and the names of the other traits refer to

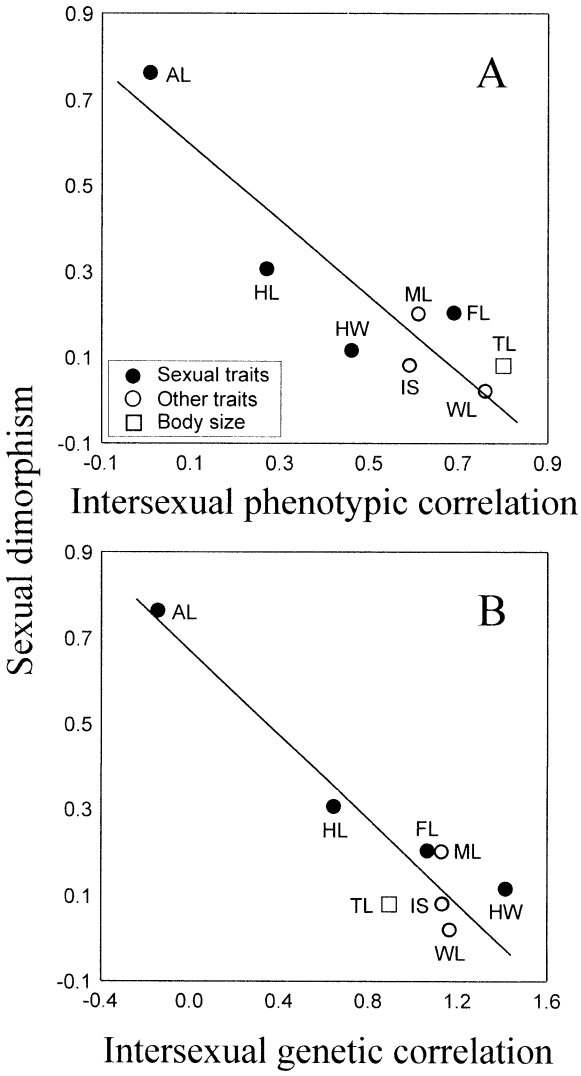

FIG. 1. Degree of sexual dimorphism for body size (open square) and seven body shape traits in *Prochyliza xanthostoma*, including sexual traits (closed circles) and nonsexual traits (open circles), plotted against the (A) intersexual phenotypic correlation (reduced major axis: $y = -0.88x + 0.68$) and (B) intersexual genetic correlation (reduced major axis: $y = -0.48x + 0.67$).

their relative sizes. The degree of sexual dimorphism for each shape trait i was calculated as

$$D_i = \left| 1 - \frac{(\bar{X}_i^m / \bar{X}_{TL}^m)}{(X_i^f / X_{TL}^f)} \right|, \quad (2)$$

where $\bar{X}_i^j$ is the mean absolute size of trait i in sex j, and $\bar{X}_{TL}^j$ is the mean thorax length of sex j. Sexual dimorphism for body size was estimated from the male:female thorax length ratio.

CV_A and CV_R were calculated using the method of Houle (1992). Data were converted to Z-scores prior to calculation of heritabilities and r_{MF} to standardize male and female phenotypic variances, so that observed differences between maternal and paternal estimates could be interpreted as differences between the sexes in genetic components of trait variance (Lynch and Walsh 1998). Narrow-sense heritabilities (h^2) were estimated separately through the mother and father for male and female offspring from regressions of offspring means on one parent (Falconer and Mackay 1996), providing maximum power for comparisons of maternal and paternal heritabilities. Each estimate and each comparison of maternal and paternal heritabilities was based on 10,000 bootstrap iterations. r_{MF} was estimated from parent-offspring covariances (eq. 1). For comparison, we also computed intersexual phenotypic correlations based on homologous traits of male and female full-sibs. We used nonparametric tests (Spearman rank correlations) to analyze the relation-ship between r_{MF} and sexual dimorphism because these variables do not appear to be normally distributed. We did not employ Bonferroni-type adjustments for multiple tests because each comparison tested a separate null hypothesis (Perneger 1998; Bender and Lange 2001).

RESULTS

As predicted, the intersexual genetic correlation (r_{MF}) was a decreasing function of the degree of sexual dimorphism (Spearman rank correlation: $N = 8$, $R = -0.74$, $t = -2.68$, $P = 0.0366$; Fig. 1, Table 2). A similar pattern, albeit slightly weaker, was exhibited by the intersexual phenotypic correlation (Spearman rank correlation: $N = 8$, $R = -0.67$, $t = -2.19$, $P = 0.0710$). Qualitatively identical results were obtained with only the seven shape traits included in the analysis (i.e., with body size excluded). This pattern is not driven by any single trait; if any trait was excluded, the resulting re-

TABLE 2. Intersexual phenotypic correlations with standard errors (SE) and significance based on t-tests and genetic correlations with significance based on bootstrap for body size and seven body shape traits in *Prochyliza xanthostoma*.

	Phenotypic			Genetic	
Trait	Correlation	SE	Significance	Correlation	Significance
Body size (TL)	0.80	0.081	*	0.90	*
Head length (HL)	0.27	0.129	*	0.64	
Head width (HW)	0.46	0.119	*	1.42	
Antenna length (AL)	0.01	0.134	†	−0.15	†
Foretibia length (FL)	0.69	0.097	*	1.06	*
Midtibia length (ML)	0.61	0.106	*	1.13	*
Wing-vein length (WL)	0.76	0.087	*	1.17	*
Intersetal length (IS)	0.59	0.108	*	1.13	

* Differs from zero ($P < 0.05$).
† Differs from one ($P < 0.05$).

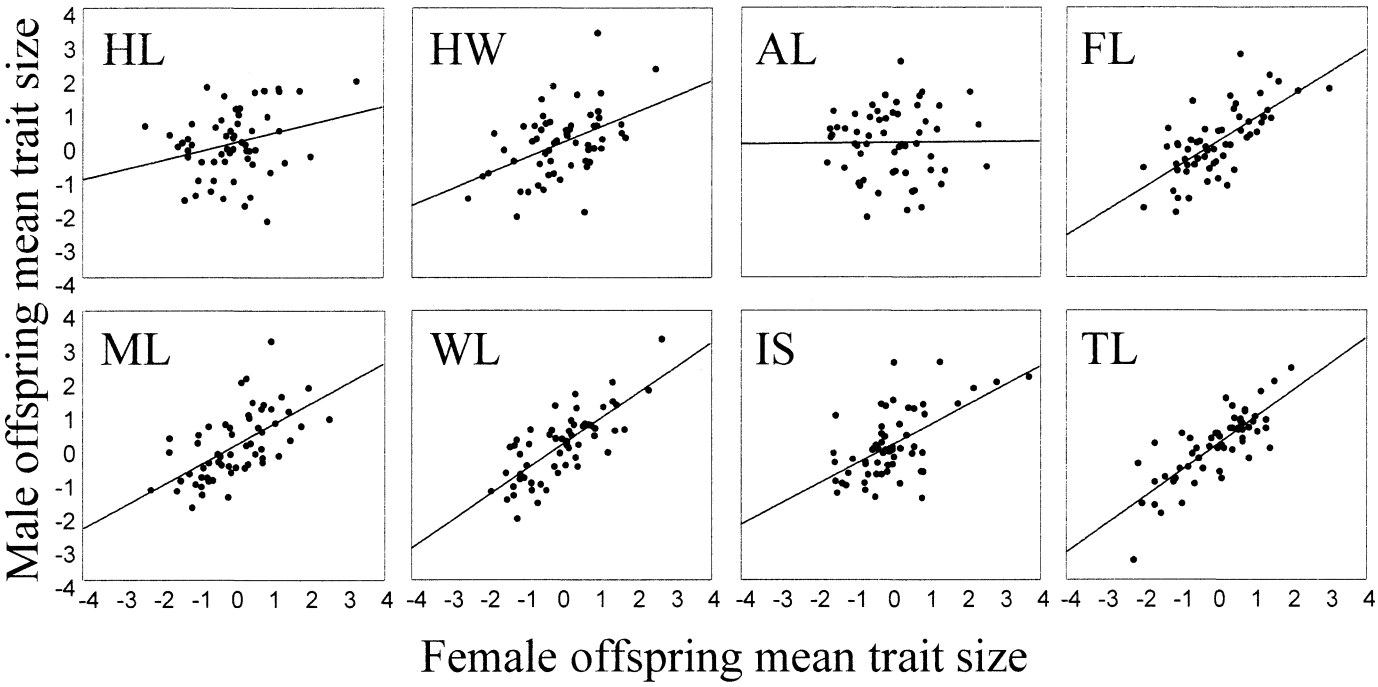

FIG. 2. Intersexual phenotypic correlations based on male and female full-sibs for seven body shape traits and body size (TL) in *Prochyliza xanthostoma*, with fitted ordinary least-squares regressions. For shape-trait abbreviations, see Materials and Methods.

duced major axis slope (range: −0.45 to −0.57) did not differ significantly (all bootstrap $P > 0.15$) from the slope for the full sample of eight traits (−0.48). For example, excluding antenna length changed the slope from −0.48 to −0.46 (bootstrap $P = 0.3$). The lowest r_{MF} and greatest dimorphism occurred in two traits that serve sexual functions in males (head length and antenna length). Although the r_{MF} for head length (0.64) did not differ significantly from one (bootstrap $P = 0.1$), the estimated genetic and phenotypic correlations between sexes for this trait were both substantially lower than those for any other trait except for antenna length (see Table 2, Fig. 2), suggesting that the lack of a significant difference from one results from insufficient statistical power. Other traits exhibited high r_{MF} and moderate sexual dimorphism (Table 2, Fig. 2).

Comparisons of maternal and paternal heritabilities (Table 3) revealed a breakdown in intersexual inheritance, particularly in sexual traits. The most dimorphic sexual trait (antenna length) exhibited no detectable heritability through the opposite-sex parent in offspring of either sex (Fig. 3). The nonsignificant, negative father-daughter heritability (and r_{MF}) for this trait approached zero if a single outlier (circled point

TABLE 3. Narrow-sense heritabilities (h^2) and their standard errors (SE), calculated separately through the mother and father and through male and female offspring, for body size (thorax length) and seven body shape traits in *Prochyliza xanthostoma*. Differences between heritability estimates (paternal-maternal) and associated bootstrap probabilities are also shown.

		Maternal estimates		Paternal estimates			
Trait	Offspring	h^2	SE	h^2	SE	Difference	P
Body size (TL)	male	0.52*	0.28	0.51*	0.31	−0.01	0.5177
	female	0.78***	0.24	0.61*	0.30	−0.17	0.6774
Head length (HL)	male	0.38	0.27	0.80***	0.23	0.42	0.0811
	female	0.61*	0.28	0.56*	0.29	−0.06	0.5778
Head width (HW)	male	0.32	0.23	0.49*	0.25	0.17	0.2604
	female	0.33	0.23	0.98***	0.22	0.65	0.0112
Antenna length (AL)	male	0.03	0.23	0.91***	0.21	0.87	0.0003
	female	0.62**	0.22	−0.38	0.31	−1.01	0.0026
Foretibia length (FL)	male	0.52**	0.20	1.04***	0.24	0.52	0.0267
	female	0.39	0.25	0.87***	0.22	0.49	0.0649
Midtibia length (ML)	male	0.71**	0.21	0.68***	0.24	−0.04	0.5768
	female	0.69**	0.20	0.83**	0.29	0.14	0.3482
Wing-vein length (WL)	male	0.80**	0.29	1.04***	0.25	0.25	0.1843
	female	0.67*	0.28	1.20***	0.23	0.53	0.0328
Intersetal width (IS)	male	0.11	0.26	0.48*	0.24	0.37	0.1427
	female	0.14	0.34	0.76***	0.26	0.63	0.0946

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

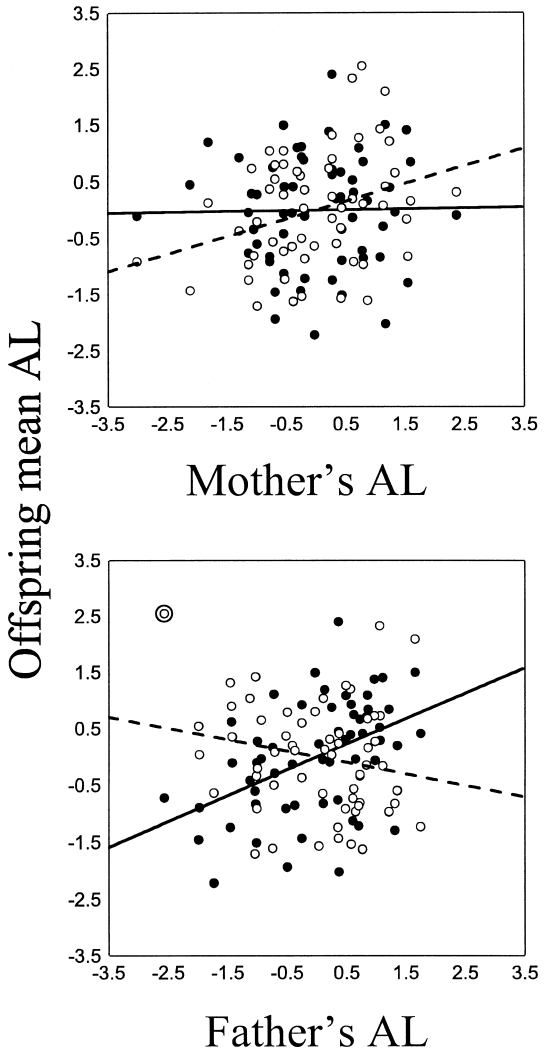

FIG. 3. Maternal and paternal heritabilities for antenna length (AL) in *Prochyliza xanthostoma* males (closed circles, solid line) and females (open circles, dashed line), with fitted ordinary least-squares regressions.

in Fig. 3) was excluded. Less strongly dimorphic sexual traits exhibited reduced maternal heritability, either in male offspring only (possibly head length) or in offspring of both sexes (foretibia length and, possibly, head width). None of the nonsexual traits exhibited significant differences between maternal and paternal heritabilities in offspring of both sexes, although wing-vein length and intersetal width exhibited a trend toward stronger paternal heritabilities. The two leg traits provide an interesting contrast: foretibia length (a sexual trait) was more strongly heritable through the father in offspring of both sexes, whereas midtibia length (a nonsexual trait) exhibited similar maternal and paternal heritabilities in offspring of both sexes (Fig. 4). The difference between paternal and maternal heritabilities was significantly greater for foretibia length than for midtibia length in male offspring (bootstrap $P < 0.05$) but not in female offspring (bootstrap $P = 0.14$). On average, paternal heritabilities (mean = 0.70, SD = 0.26) were stronger than maternal heritabilities (mean = 0.48, SD = 0.22; paired sample *t*-test: $t = -2.52$, df = 7, $P = 0.0396$). Among all traits, the degree of sexual dimorphism was positively correlated with the paternal-maternal heritability difference in males ($n = 8$, $r = 0.77$, $t = 2.98$, $P = 0.0245$) but negatively correlated with the paternal-maternal heritability difference in females ($n = 8$, $r = -0.86$, $t = -4.10$, $P = 0.0063$). Thus, traits that are relatively larger in males (perhaps because of stronger sexual selection) tended to exhibit stronger h^2 through the father than through the mother in male offspring but weaker h^2 through the father than through the mother in female offspring.

Maternal and paternal estimates of CV_A and CV_R also differed substantially for some traits (Table 4). The greatest difference was in a sexual trait (antenna length), but head length, head width, and intersetal length also exhibited substantial differences. Paternal CV_A estimates exceeded maternal estimates in 13 of the 15 comparisons (8 traits $\times$ 2 offspring sexes, excluding the father-daughter estimate for antenna length; sign test: $Z = 2.58$, $P = 0.0098$), whereas CV_R exhibited the converse pattern. The largest CV_A was exhibited by antenna length (father-son and mother-daughter estimates). Head length also exhibited a large father-son CV_A, whereas wing-vein length (a nonsexual trait) exhibited large maternal and paternal CV_A estimates through offspring of both sexes. The largest CV_R values were obtained for head length (father-son and mother-son estimates).

DISCUSSION

Intersexual Genetic Correlation and Sexual Dimorphism

Because a high intersexual genetic correlation (r_{MF}) will impede the evolution of sexual dimorphism (Fisher 1930a,b, 1931; Lande 1980, 1987), sex-specific selection should favor reduced r_{MF}. This hypothesis should be manifested in a negative correlation between r_{MF} and degree of sexual dimorphism, with the greatest dimorphism and lowest r_{MF} exhibited by traits directly targeted by sexual selection. Our findings are largely consistent with these predictions. We observed a strong negative correlation between r_{MF} and the degree of sexual dimorphism (Fig. 1). The greatest dimorphism and lowest r_{MF} were exhibited by two sexual traits: antenna length and head length. Thus, in *P. xanthostoma*, r_{MF} is reduced substantially in at least some traits under sex-specific selection, as predicted by Lande (1980, 1987), and the degree of sexual dimorphism is inversely proportional to r_{MF}. These findings suggest that sex-specific selection favors genetic modifications that reduce r_{MF}, thereby permitting a more rapid or more complete resolution of intralocus sexual conflict.

Note, however, that there is an alternative explanation for the negative correlation between r_{MF} and sexual dimorphism: traits that exhibit lower r_{MF} for reasons unrelated to sex-specific selection could evolve dimorphic phenotypes more rapidly than traits with higher r_{MF}. We regard this explanation as implausible because there is no evidence to suggest that traits vary substantially in r_{MF} for reasons unrelated to sex-specific selection. Homologous traits of males and females typically exhibit $r_{MF} \approx 1$ (Lande 1980, 1987). The known exceptions to this rule are traits that appear to be targeted by sexual selection in one sex (see below). It seems unlikely that the association between sex-specific selection and low r_{MF} is merely coincidental or spurious. More plausibly, low

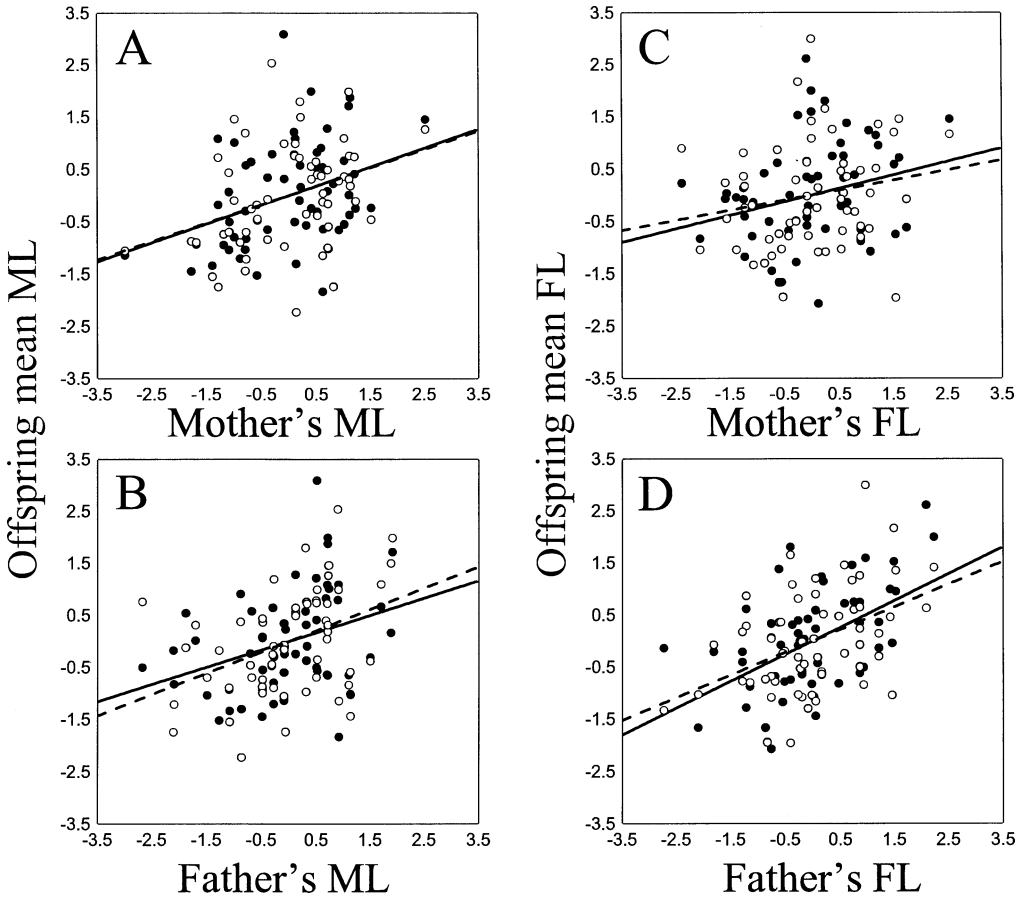

FIG. 4. Maternal and paternal heritabilities for midtibia length (A, B) and foretibia length (C, D) in *Prochyliza xanthostoma* males (closed circles, solid line) and females (open circles, dashed line), with fitted ordinary least-squares regressions. For shape-trait abbreviations, see Materials and Methods.

r_{MF} evolves in traits subject to sex-specific selection because reductions in r_{MF} allow the sexes to evolve further or more rapidly toward their sex-specific phenotypic optima.

The role of sex-specific selection in the breakdown of r_{MF} is also supported by the data of Chenoweth and Blows (2003). A reanalysis of their data showed that traits subject to substantially different sexual selection vectors (β-coefficients) in the two sexes tended to exhibit lower r_{MF} than traits subject to similar sexual selection. In contrast with our results, however, their data exhibited no correlation between r_{MF} and sexual dimorphism.

The view that sex-specific selection favors reduced r_{MF} appears to conflict with a simulation suggesting that pronounced sexual dimorphism can evolve without substantial reduction in r_{MF} and that traits in advanced stages of sexual dimorphism evolution will exhibit high r_{MF} (Reeve and Fairbairn 2001). However, the model of Reeve and Fairbairn (2001) did not permit the genetic architecture itself to evolve, and this may account for the difference between their results and ours. Modifications to the genetic architecture (in particular, novel mechanisms of epistasis with sex-specific effects, which may often reduce r_{MF}) may be favored by selection because they would allow sexual dimorphism to evolve more rapidly (Wright 1993) or result in a more complete resolution of intralocus sexual conflict (Day and Bonduriansky 2004).

We observed considerable variation in r_{MF} among sexual traits (Table 2), as have previous studies. For example, Chenoweth and Blows (2003) reported values ranging from about 1.0 to −0.277 for cuticular hydrocarbons in *Drosophila serrata*, Preziosi and Roff (1998) reported a range of 0.17 to 1.01 for morphological traits in a water strider, and Meagher (1992) reported a range of 0.50 to 0.96 for sexual traits in *Silene latifolia*. In addition, Møller (1993) gave an estimate of 0.54 for tail length in barn swallows, Simmons and Ward (1991) gave an estimate of 0.38 for body size in yellow dung flies (*Scathophaga stercoraria*), and Wilkinson (1993) provided an estimate of 0.29 for eye-stalk span in the diopsid fly *Cyrtodiopsis dalmanni*. This variation may reflect the intensity or duration of sexual selection on these traits and the degree to which intralocus sexual conflict has been resolved by evolution of the genetic architecture.

Our study also suggests that, like within-sex genetic correlations (Cheverud 1988; Roff 1995, 1996), intersexual genetic correlations for morphological traits can be estimated by the phenotypic correlations. Genetic correlations were substantially larger than the corresponding phenotypic correlations (Table 2), a pattern reported previously by Cheverud

TABLE 4. Phenotypic variances (V_P), estimates of additive genetic variance (V_A), coefficients of additive genetic variance (CV_A), and coefficients of residual variance (CV_R) based on maternal (mother-offspring) and paternal (father-offspring) covariances (COV) for body size (thorax length) and seven body shape traits of *Prochyliza xanthostoma* male and female offspring. Because the father-daughter covariance for antenna length is negative, corresponding V_A, CV_A, and CV_R estimates were not computed.

			Maternal estimates				Paternal estimates			
Trait	Offspring	V_P	COV	V_A	CV_A	CV_R	COV	V_A	CV_A	CV_R
Body size	male	26.05	4.50	9.00	1.92	2.64	4.99	9.97	2.02	2.57
	female	18.32	5.93	11.85	2.01	1.49	5.26	10.52	1.89	1.63
Head length	male	31.49	1.42	2.83	1.36	4.32	4.75	9.50	2.48	3.78
	female	8.31	1.39	2.78	1.60	2.26	1.92	3.85	1.88	2.03
Head width	male	6.40	0.44	0.87	1.11	2.79	0.75	1.49	1.45	2.63
	female	5.55	0.43	0.86	0.89	2.07	1.47	2.93	1.64	1.55
Antenna length	male	22.70	0.09	0.19	0.45	4.86	5.35	10.70	3.35	3.55
	female	5.45	1.22	2.44	2.56	2.84	−1.44	—	—	—
Foretibia length	male	10.34	0.76	1.51	1.18	2.85	1.76	3.42	1.77	2.52
	female	6.29	0.62	1.24	1.17	2.37	1.63	3.27	1.90	1.83
Midtibia length	male	12.87	1.46	2.92	1.51	2.80	1.34	2.69	1.45	2.83
	female	7.51	1.53	3.07	1.70	2.05	1.79	3.59	1.84	1.92
Wing-vein length	male	52.82	9.04	18.07	2.43	3.37	14.23	27.28	2.98	2.89
	female	40.11	8.72	17.43	2.14	2.44	18.62	30.91	2.85	1.56
Intersetal length	male	7.97	0.19	0.38	0.86	3.85	0.88	1.76	1.85	3.48
	female	6.39	0.30	0.60	0.92	2.85	1.78	3.56	2.23	1.99

(1988). However, the two sets of estimates were strongly correlated ($n = 8$, $r = 0.77$, $t = 2.92$, $P = 0.0267$), and their analysis yielded similar results (Fig. 1).

In our analysis, we treated each trait as an independent observation, even though each trait does not evolve completely independently of the others. However, within-sex genetic correlations among these traits are mostly weak (Bonduriansky and Rowe 2005) and, more importantly, their pattern cannot explain our results. For example, the strongest genetic correlation was between head length and antenna length, but these traits differ substantially in r_{MF} (bootstrap $P < 0.05$) and sexual dimorphism (Table 1). Similar conclusions follow from an inspection of the within-sex phenotypic correlations among traits (Bonduriansky and Rowe 2005). Our analysis also follows the convention for studies that compare multiple traits (e.g., Møller and Mousseau 2003).

Evolution of Heritability

Day and Bonduriansky (2004) hypothesized that intralocus sexual conflict selects for reduced intersexual inheritance. Our results support this hypothesis. In the nonsexual traits, differences between maternal and paternal heritabilities were weak. In contrast, the most dimorphic sexual trait (antenna length) was heritable only through the same-sex parent in offspring of both sexes (Fig. 3). These patterns follow from the hypothesis that sex-specific selection favors reduced r_{MF} (see above), as well as from the theory of Day and Bonduriansky (2004). However, less strongly dimorphic sexual traits (head length, head width and, particularly, foretibia length) exhibited stronger paternal heritability, either in male offspring only or in offspring of both sexes. No trait exhibited reduced paternal heritability in female offspring without also exhibiting reduced maternal heritability in male offspring (Table 3). These patterns are consistent with novel predictions of Day and Bonduriansky (2004). The probable role of sexual selection in shaping these heritability patterns is illustrated by a comparison of the fore- and midtibia. These traits exhibit a strong within-sex genetic correlation (Bonduriansky and Rowe 2005) and similar r_{MF} (Table 2) but differ in function: the midleg is used only in walking and standing, whereas the foreleg is used as a weapon and signal by males (Bonduriansky 2003). In light of the theory of Day and Bonduriansky (2004), this difference in function accounts for the contrasting heritability patterns exhibited by these traits (Fig. 4).

Our results suggest that traits vary in the form and extent of genetic adaptation to intralocus sexual conflict. The two most dimorphic sexual traits (antenna length and head length) appeared to exhibit reductions in both intersexual inheritance and r_{MF}. In contrast, less strongly dimorphic traits (such as foretibia length) exhibited reductions in intersexual inheritance only. Even in the absence of any reduction in r_{MF}, reduced maternal heritability in offspring of both sexes will mitigate intralocus sexual conflict as long as the benefit to male offspring more than compensates for any cost to female offspring (Day and Bonduriansky 2004). Such variation may reflect the conflict's severity during the evolutionary history of these traits.

Body size exhibited similar maternal and paternal heritabilities in offspring of both sexes. Although body size is under sexual selection in males (Bonduriansky and Rowe 2003), it is also subject to strong fecundity selection in females (Bonduriansky and Brooks 1999). Consequently, body size is unlikely to be under strong sexually antagonistic selection in *P. xanthostoma*, so that little difference between maternal and paternal heritabilities is to be expected for this trait.

Estimates of CV_A and CV_R, calculated separately through the mother and father, were consistent with these conclusions (Table 4) but also revealed substantial differences between traits in components of variance (Table 4). The observation that sexual traits tend to exhibit higher CV_A and CV_R than nonsexual traits accords with previous findings (Pomiankowski and Møller 1995; Rowe and Houle 1996). However, considerable variation among sexual traits in these parame-

ters and in condition dependence (Bonduriansky and Rowe 2005) calls for further research.

We used variation among traits in function (in males) and in the degree of sexual dimorphism to infer variation in the severity of intralocus sexual conflict. It might be possible to quantify intralocus sexual conflict more precisely by comparing selection on homologous traits in males and females. Very few attempts have been made to obtain such data (but see Chenoweth and Blows 2003), so it is not clear whether traits under sexual selection in males are typically subject to opposing directional selection in females, stabilizing selection in females, or stabilizing selection in both sexes. An alternative approach is to generate intralocus sexual conflict de novo by imposing artificial sexually antagonistic selection and to track the evolution of the genetic architecture over several generations. Few such experiments have been attempted (but see Bird and Schaffer 1972; Meagher 1994). New tools of genomic analysis (e.g., Yang et al. 2003) may facilitate this type of work.

Our heritability comparisons are not confounded by sex differences in morphometric repeatabilities (Bonduriansky and Rowe 2005). Furthermore, although additive genetic variances and r_{MF} may be affected by rearing environment (Conner et al. 2003) and different traits may be affected differently, there is no reason to believe that our results are artifacts of laboratory rearing conditions. Even if laboratory conditions affect r_{MF} and heritabilities of morphological traits, there is no known reason to expect this effect to differ with trait function or degree of sexual dimorphism so as to generate the observed patterns. Likewise, nongenetic maternal effects probably had little effect on our results because maternal heritabilities were substantially weaker, on average, than paternal heritabilities. Nongenetic paternal effects are possible because *P. xanthostoma* females ingest postcopulatory ejaculate meals that affect their fecundity (Bonduriansky et al. 2005) and could affect offspring phenotype. However, although this factor may strengthen paternal heritability on average, it cannot explain the association between paternal heritability and trait function or sexual dimorphism.

Note, however, that our heritability estimates are based on the assumption that alleles from both parents are expressed to an equal degree in each offspring, whereas our predictions are based on the hypothesis that one parent's alleles may be silenced to some degree in some traits (Day and Bonduriansky 2004). The assumption of equal maternal and paternal genetic contributions is also challenged by very high heritabilities through one parent (Table 3; see also Houde 1992). If alleles from one parent are partially or completely silenced in offspring, then some heritability estimates may exceed the true values. This may also explain the excessively high r_{MF} estimates for some traits.

Our findings can be compared with few published studies because heritabilities are typically reported either through the same-sex parent only or through a midparent analysis (e.g., Alatalo and Lundberg 1986; Arnqvist 1986, 1990; Møller 1991; Sakaluk et al. 1992). Moreover, of the few studies reporting separate maternal and paternal heritabilities, most have focused on body size or the absolute sizes of morphological traits uncorrected for (and, therefore, usually genetically correlated with) body size. Such data are difficult to interpret because they confound the genetic architectures of body size and shape and because selection on body size may be similar in both sexes (see above).

Nonetheless, several published results appear to be consistent with our predictions, although some do not. Simmons and Ward (1991) found that absolute hind tibia length was much more strongly heritable through the same-sex parent in both sexes, whereas Wilcockson et al. (1995) obtained stronger paternal heritability in both sexes (but especially in males) for absolute wing length. Likewise, of several sexual traits in a grasshopper, most were more strongly heritable through the father than through the mother (Butlin and Hewitt 1986). These patterns accord with our predictions for traits under sexual selection in males. In addition, male color patterns in *Poecilia reticulata* (guppies) and *Poecilia parae* are much more strongly heritable through the father, although this pattern has been attributed to Y-linkage (Winge 1927; Fisher 1930a; Houde 1992; Brooks 2000; Brooks and Endler 2001; Lindholm and Breden 2002; Lindholm et al. 2004). Conversely, Simmons (1987) obtained similar maternal and paternal heritabilities for absolute hind/leg length, body length, body weight, and pronotum width in the field cricket *Gryllus bimaculatus*, and Merilä et al. (1998) found similar maternal and paternal heritabilities for absolute tarsus length in collared flycatchers (*Ficedula albicollis*). These results contrast with our predictions for sexual traits but accord with our findings for body size. Contrary to our predictions, Price and Burley (1993) obtained similar maternal and paternal heritabilities for bill color in zebra finches, whereas Ritchie and Kyriacou (1994) found that male courtship song traits in *D. melanogaster* were nonheritable through the father. Clearly, much more evidence is required.

Proximate Mechanisms

An understanding of proximate mechanisms is key to a more rigorous test of the theory of Day and Bonduriansky (2004). This is because heritability patterns for quantitative morphological traits are likely to reflect the effects of multiple genes, permitting only weak inferences about the heritability effects of constituent loci. For example, although sex-limited gene expression may result in reduced heritability through the opposite-sex parent, it will not eliminate intersexual inheritance, and selection is expected to favor further genetic modifications that prevent the expression of alleles inherited from the opposite-sex parent through parent-of-origin effects. Our analysis does not allow us to distinguish such mechanisms conclusively. However, some tentative inferences can be made.

In *P. xanthostoma*, males are probably heterogametic (XY), as in the closely related *Piophila casei* (Canoval et al. 1987). Stronger paternal heritability in male offspring only (observed tentatively for head length) is thus consistent with Y-linked modifiers, male-limited expression of autosomal genes, or parent-of-origin effects. Stronger paternal heritability also argues against a major role for X-linked modifiers, which would strengthen maternal heritability. Reduced heritability through the opposite-sex parent in offspring of both sexes could result from sex-limited autosomal genes, although reduction of these heritabilities to zero (as observed

for antenna length) would require the duplication and sex-limitation of all underlying loci. An alternative explanation for this pattern is a sexually dimorphic form of genomic imprinting predicted by Day and Bonduriansky (2004). Particularly intriguing is stronger paternal heritability in offspring of both sexes (Table 1, Fig. 4). This pattern cannot be explained as a result of sex limitation or sex linkage but could readily result from genomic imprinting, with silencing of maternally inherited alleles in all offspring (Spencer 2002). Such parent-of-origin effects are known in a wide range of taxa, including *Drosophila* (Lloyd 2000), mammals (Monk 1987; Moore and Haig 1991; de Koning et al. 2000; Rattink et al. 2000; Goos and Silverman 2001; Moore 2001), birds (Tuiskula-Haavisto et al. 2004), and plants (Alleman and Doctor 2000), and typically appear to play a role in the regulation of organ development and growth. Reduced autosomal recombination rate in males (Lenormand 2003) could contribute to this result. However, the relatively minor consequences of this factor for heritabilities in *D. melanogaster* (Cowley and Atchley 1988) suggest that it is unlikely to account for our results. Nonetheless, we cannot rule out the possibility that other, more complex forms of epistasis underlie the observed patterns.

Conclusions

Our findings support the hypotheses that sexual selection contributes to the breakdown of intersexual genetic correlations and provide empirical support for a new theory on the breakdown of intersexual inheritance in sexually selected traits. Both factors contribute to the resolution of intralocus sexual conflict, allowing for the adaptive divergence of the sexes. Indeed, the extreme degree to which these evolutionary processes have progressed in one trait (antenna length) suggests that intralocus sexual conflict and the constraints it imposes on the adaptive evolution of the sexes can be resolved to a substantial degree through the evolution of genetic architecture.

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