Evolution, 59(1), 2005, pp. 138–151

SEXUAL SELECTION, GENETIC ARCHITECTURE, AND THE CONDITION DEPENDENCE OF BODY SHAPE IN THE SEXUALLY DIMORPHIC FLY *PROCHYLIZA XANTHOSTOMA* (PIOPHILIDAE)

Russell Bonduriansky[1] and Locke Rowe
Department of Zoology, University of Toronto, Toronto, Ontario M5S 3G5, Canada

Abstract.—The hypothesis that sexual selection drives the evolution of condition dependence is not firmly supported by empirical evidence, and the process remains poorly understood. First, even though sexual competition typically involves multiple traits, studies usually compare a single sexual trait with a single ''control'' trait, ignoring variation among sexual traits and raising the possibility of sampling bias. Second, few studies have addressed the genetic basis of condition dependence. Third, even though condition dependence is thought to result from a form of sex-specific epistasis, the evolution of condition dependence has never been considered in relation to intralocus sexual conflict. We argue that condition dependence may weaken intersexual genetic correlations and facilitate the evolution of sexual dimorphism. To address these questions, we manipulated an environmental factor affecting condition (larval diet) and examined its effects on four sexual and four nonsexual traits in *Prochyliza xanthostoma* adults. As predicted by theory, the strength of condition dependence increased with degree of exaggeration among male traits. Body shape was more condition dependent in males than in females and, perhaps as a result, genetic and environmental effects on body shape were congruent in males, but not in females. However, of the four male sexual traits, only head length was significantly larger in high-condition males after controlling for body size. Strong condition dependence was associated with reduced intersexual genetic correlation. However, homologous male and female traits exhibited correlated responses to condition, suggesting an intersexual genetic correlation for condition dependence itself. Our findings support the role of sexual selection in the evolution of condition dependence, but reveal considerable variation in condition dependence among sexual traits. It is not clear whether the evolution of condition dependence has mitigated or exacerbated intralocus sexual conflict in this species.

Key words.—Condition dependence, diet, genetic architecture, intralocus sexual conflict, *Prochyliza xanthostoma*, sexual dimorphism, sexual selection.

Received July 15, 2004. Accepted October 1, 2004.

Condition can be defined as the quantity of metabolic resources that an individual can accumulate, and the efficiency with which it can translate them into fitness. Thus, condition represents an individual's potential fitness, or that combination of environmentally and genetically determined phenotypic parameters that best predicts individual reproductive success. Condition dependence—a form of developmental plasticity that links phenotypic trait expression to condition—is an important source of intraspecific variation in body shape and a key element of many sexual selection models (see Darwin 1874; Fisher 1915; Zahavi 1975, 1977; Andersson 1982; Nur and Hasson 1984; West-Eberhard 2003).

Theory predicts that condition dependence will evolve in traits subject to a directional selection vector, such as life-history traits or sexually selected traits, because a component of fitness is an increasing function of investment in such traits, so that an individual will benefit by allocating as much as it can afford to such a trait (Andersson 1982; Nur and Hasson 1984). The amount that an individual should invest may be determined by the direct viability costs of trait expression, trade-offs with other traits, and expected fitness pay-offs (Getty 1998a,b, 2002), all of which may be functions of condition. Thus, condition dependence enables individuals to express their sexually selected traits at the maximum level that they can afford given their condition, optimizing the trade-off between mating success and viability (McAlpine 1979). Because an individual in good condition will incur lower costs for a given level of sexual trait expression than an individual in poor condition, the individual in good condition will be able to afford a larger sexual trait. In sexually selected traits, selection for condition dependence may result from mate choice, which should lead to exaggeration of reliable (''honest'') signals of mate quality, and/or from intrasexual agonistic interactions whose outcome depends on the expression of intrasexual signals or weapons (Nur and Hasson 1984; Johnstone and Grafen 1992, 1993; Price et al. 1993; Rowe and Houle 1996). Consequently, such traits are expected to ''capture'' genetic and phenotypic variation in condition (Rowe and Houle 1996). In contrast, ordinary ''metric'' traits under weak stabilizing selection are not expected to evolve condition-dependent expression, because fitness is not increased by allocating more resources to such traits (Schluter et al. 1991). Indeed, in ordinary metric traits, selection is expected to favor condition-independent expression through increased developmental buffering (i.e., canalization) against environmental or genetic factors (de Visser et al. 2003). Thus, the strength of condition dependence is expected to reflect both the intensity of sexual selection on a trait, and the costs of expressing that trait.

Nonetheless, compelling evidence for a role of sexual selection in the evolution of condition dependence is relatively sparse, and very little is known about the genetics of condition dependence (Cotton et al. 2004a,b; Hunt et al. 2004; Tomkins et al. 2004). Likewise, although the genic capture model has some empirical support (e.g., Wilkinson and Taper 1999; Kotiaho et al. 2001), much remains to be learned about intertrait variation in condition dependence, as well as its

---

[1] Corresponding author. Present address: School of Biological, Earth and Environmental Sciences, University of New South Wales, Sydney 2052, Australia; E-mail: r.bonduriansky@unsw.edu.au.

genetic architecture (i.e., the set of genetic factors that determine the mapping between genotype and phenotype). Recent evidence suggests large variation in condition dependence strength among sexually selected traits within a species (Møller and Petrie 2002). Moreover, sexually selected traits may exhibit strong condition dependence in some species (e.g., Carranza and Detrucios 1993; David et al. 1998; Kotiaho 2000, 2002; Kotiaho et al. 2001; Uetz et al. 2002), whereas traits with apparently similar functions are not significantly condition dependent in others (e.g., Gray and Eckhardt 2001; Perrier et al. 2002). Finally, nonsexual morphological traits may also be affected by condition (see David et al. 2000). These puzzles form part of the larger problem of integrating developmental plasticity with evolutionary theory (West-Eberhard 1986, 1989, 2003).

A fruitful approach to the study of condition dependence is the comparison of traits with different functions within a single species (e.g., Møller and Petrie 2002; Møller and Mousseau 2003; Cotton et al. 2004b). Unfortunately, most such studies have contrasted a single sexually selected trait with a single control trait (see Cotton et al. 2004a). Given that multiple traits are typically involved in sexual competition (since even a single organ or structure comprises multiple dimensions or characteristics that can be regarded as distinct traits), the utility of the comparative approach increases with the number of traits included in the analysis. Because the sample of traits included in the comparison may determine the conclusions (as illustrated by the results of the present study), comparisons involving a single pair of traits are particularly susceptible to sampling bias. Thus, comparisons of multiple traits are more likely to reveal the nature of intertrait variation in condition dependence.

In traits expressed in both sexes but subject to sexual selection in one sex only, some underlying loci are likely to experience sex-specific selection (Rice 1984; Lande 1987). Because the shared genetic basis of such traits may generate an intersexual genetic correlation that impedes the evolutionary divergence of male and female phenotypes (Lande 1980, 1987), this situation can result in an evolutionary tug-of-war known as intralocus sexual conflict. Although it remains unclear how genetic architecture responds to sex-specific selection so as to break intersexual genetic correlations and resolve intralocus sexual conflict (see Lande 1987; Rice and Chippindale 2001), this process is thought to involve the evolution of epistatic mechanisms with sex-specific effects (e.g., see Rice 1984; Rhen 2000; Rice and Chippindale 2002; Day and Bonduriansky 2004). Thus, given that condition dependence is thought to result from sex-specific epistasis (Rowe and Houle 1996), it may contribute to the breakdown of intersexual genetic correlations. The intersexual genetic correlation (r_{MF}) reflects the extent of overlap between the sets of genes affecting homologous traits in males and females (Lande 1980): when $r_{MF} = 1$, exactly the same genes affect the trait in males and females; but when $r_{MF} = 0$, completely different genes affect the trait in the two sexes. If condition dependence evolves in a given trait in males only, it must be assumed that some genetic factors contribute to trait expression in one sex but not in the other sex (i.e., $r_{MF} < 1$). The genic capture model (Rowe and Houle 1996) postulates that loci involved in resource acquisition and allocation efficiency will modify trait expression in males, producing larger traits in males of higher condition and thereby increasing phenotypic variance. However, these modifiers are assumed to be sex limited in their expression, since increased condition dependence is not expected to evolve in the homologous traits of females. Thus, the evolution of condition dependence may contribute to the breakdown of intersexual genetic correlations in sexually selected traits by producing sex-specific patterns of epistasis.

Nonetheless, condition dependence will only contribute to the resolution of intralocus sexual conflict if it can evolve in the sexually selected sex without correlated effects on the other sex. Alternatively, if genetic modifiers that evolve through selection on males are also expressed to some degree in females, then condition dependence itself will be subject to an intersexual genetic correlation. In such a case, it is not clear whether the evolution of condition dependence would mitigate or exacerbate the severity of the intralocus sexual conflict.

The carrion fly *Prochyliza xanthostoma* (Diptera: Piophilidae) possesses several morphological structures that are directly employed in male sexual competition, and exhibit varying degrees of sexual dimorphism. The greatly elongated head capsule, antennae, and forelegs of males are used as weapons in male-male combat, and as signals in male-female courtship (Bonduriansky 2003; Bonduriansky and Rowe 2003). However, *P. xanthostoma* also possesses many sexually dimorphic body parts that are not directly employed in male sexual competition (e.g., wings, thorax, mid- and hind-legs), in which sexual dimorphism may have evolved through correlated responses to sexual selection on other traits. *Prochyliza xanthostoma* females also possess elongated heads and antennae in comparison with related species (see McAlpine 1977), perhaps through a correlated response to selection on males.

Because diet quality is likely to represent an important source of variation in condition among individuals in the wild, diet manipulation is an effective method of investigating the effects of an environmental factor affecting condition (Emlen 1997; Cotton et al. 2004a). However, if we seek to understand how reaction norms are shaped by sexual selection, it is important to employ treatments that fall within the range of environments experienced in the wild by individuals that survive to adulthood (Cotton et al. 2004b). Thus, relatively subtle manipulations may provide more relevant insights than extreme contrasts that can only be achieved under laboratory conditions. We compared the morphology of adult *P. xanthostoma* males and females reared on a high-quality larval diet with the morphology of their full siblings reared on a low-quality larval diet, employing treatments that resulted in relatively small differences in mean survival and development rates (about 10%) and mean body lengths (5–6%) between siblings reared on contrasting diets. We examined diet effects on the expression of four sexual traits (i.e., linear dimensions of body parts employed directly in male sexual competition) and four nonsexual traits (i.e., linear dimensions of body parts that are not employed directly in male sexual competition; Table 1, Fig. 1). Note that, even though thorax length was previously found to be under sexual selection when used to represent body size in the analysis

TABLE 1. Morphological traits examined, indicating whether the trait functions directly in male combat and courtship (sexual) or not (nonsexual), the degree of trait exaggeration in males relative to females (i.e., sexual dimorphism) in absolute and relative trait size, and mean absolute trait sizes (mm × 100) for each sex in the low- and high-condition treatments, with standard errors (SE) in parentheses.

		Trait exaggeration in males		Mean size in females (SE)		Mean size in males (SE)	
Trait	Function	Absolute size	Relative size	Low food	High food	Low food	High food
Head length	sexual	1.20	1.30	98 (0.73)	104 (0.89)	117 (1.03)	127 (1.59)
Head width	sexual	0.81	0.88	100 (0.69)	104 (0.61)	83 (0.62)	85 (0.34)
Antenna length	sexual	1.62	1.76	58 (0.53)	61 (0.57)	94 (0.86)	101 (1.34)
Foretibia length	sexual	1.10	1.20	90 (0.68)	95 (0.83)	100 (0.72)	106 (0.93)
Midtibia length	nonsexual	1.10	1.20	98 (0.73)	103 (0.89)	108 (0.71)	115 (1.25)
Wing-vein length	nonsexual	0.90	0.98	188 (1.82)	193 (1.51)	168 (1.16)	174 (1.24)
Intersetal width	nonsexual	0.84	0.92	81 (0.83)	84 (0.70)	68 (0.55)	73 (0.58)
Thorax length	nonsexual	0.92	—	161 (1.32)	169 (1.09)	148 (0.81)	158 (1.21)

(Bonduriansky and Rowe 2003), it is considered a nonsexual trait because it does not play a direct role in male sexual competition (Bonduriansky 2003). Because the degree of trait exaggeration in males, relative to females, may reflect the importance of the trait in sexual competition as well as the viability costs of trait expression, we expected the most exaggerated traits to exhibit the strongest condition dependence. Moreover, we expected condition to affect males' sexual traits more than males' nonsexual traits, or the homologous traits of females. We also examined and compared environmental and genetic effects on body shape in males and females (see Cheverud 1988; West-Eberhard 2003). Finally, we asked whether the evolution of condition dependence in response to sexual selection might mitigate the intensity of intralocus sexual conflict in *P. xanthostoma*. Thus, we tested for negative covariation among traits between the strength of condition dependence in males and the intersexual genetic correlation, and compared the effects of condition on homologous traits of males and females.

We define condition dependence operationally as an effect of diet on trait size, quantified in two ways: (1) effects on absolute trait sizes; (2) effects on relative trait sizes, calculated in relation to a linear index of body size (thorax length; Fig. 2). The effect of diet on absolute trait size reflects the absolute condition dependence strength of a trait, whereas the effect of diet on relative trait size reflects the condition dependence strength of a trait relative to that of the body size index. Moreover, absolute trait sizes almost invariably in-

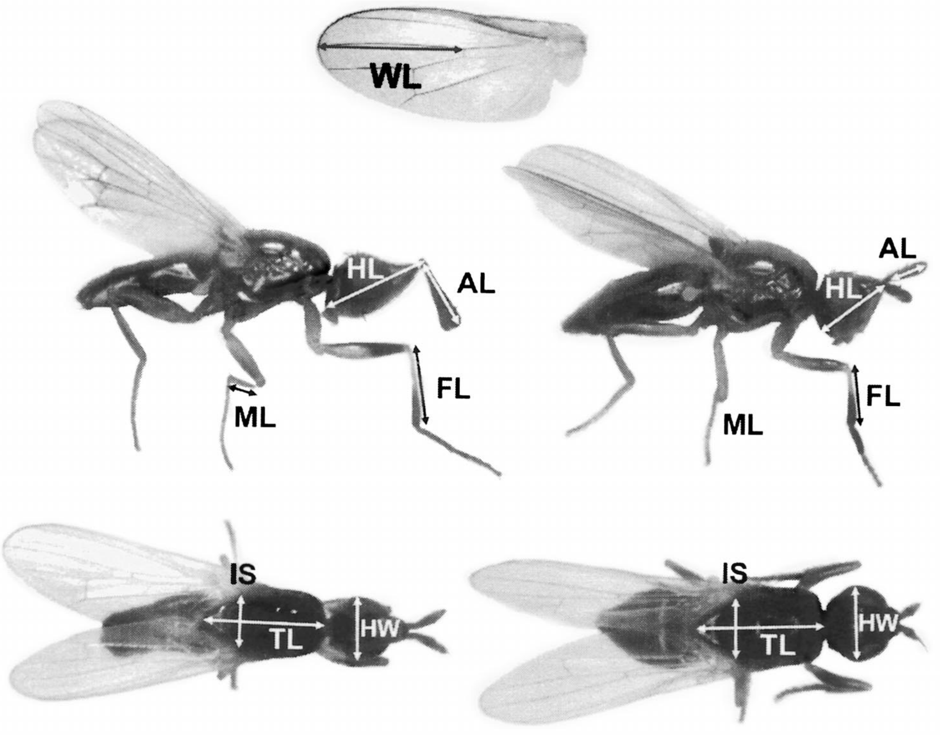

FIG. 1. Morphological traits measured in male (left) and female (right) *Prochyliza xanthostoma*, each viewed from the side (top) and from above (bottom): head length (HL), head width (HW), antenna length (AL), foretibia length (FL), midtibia length (ML), wing-vein length (WL), intersetal width (IS), and thorax length (TL) (see Materials and Methods).

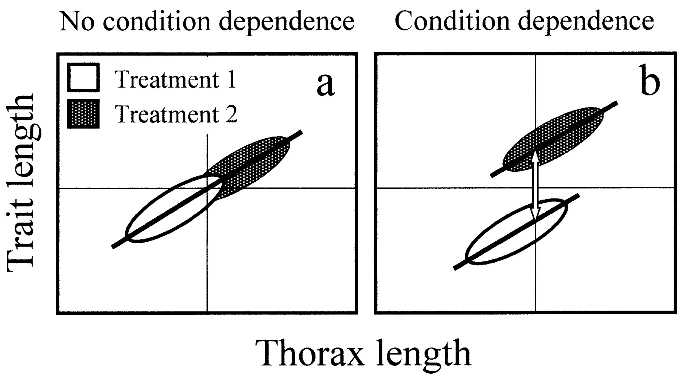

FIG. 2. Hypothetical effects of larval diet quality manipulation on relative sizes of morphological traits in *Prochyliza xanthostoma*, corrected for body size by plotting trait length against thorax length: (a) noncondition-dependent traits such as ordinary metric traits are expected to scale with similar intercept in both treatments, even if body size differs between treatments; (b) condition-dependent traits such as signal or weapon traits are expected to scale with larger intercepts in the high-condition treatment than in the low-condition treatment (the arrow indicates the magnitude of the response to condition manipulation).

crease with increasing body size, and thus nearly always exhibit positive genetic and phenotypic correlations. In contrast, relative trait sizes may exhibit negative genetic or phenotypic correlations, making it easier to interpret variation in body *shape*. Indeed, condition dependence of morphology is conventionally defined in terms of condition effects on body shape, with sexual traits expected to increase faster than nonsexual traits in relation to body size as condition increases (see Cotton et al. 2004a). Estimating the condition dependence of relative trait sizes also makes it possible to determine which traits, if any, signal condition more reliably than body size. Thus, we present analyses based on absolute or relative trait sizes or both, as appropriate.

MATERIALS AND METHODS

Rearing of Flies

Gravid *Prochyliza xanthostoma* females ($n = 37$) were collected off carcasses of moose (*Alces alces*) in Algonquin Park, Ontario, and transferred to 1.5 L cages containing water and sugar and petri dishes containing larval medium (''organic,'' nonhormone-treated extra-lean ground beef aged for about five days at room temperature), and maintained on an 18:6 light:dark cycle at about 60% humidity under a combination of incandescent and fluorescent (broad-spectrum, high flicker-frequency) lights. Larvae from these petri dishes were allowed to pupate in cups containing soil, and the pupae were transferred individually to scintillation vials. After emerging as adults, flies were maintained for 10 days in same-sex groups of about 10 in 1.5 L cages containing water, sugar, and small quantities of ground beef. Males and females were then paired randomly in 250 ml cages containing water, sugar, and perforated eppendorf tubes partially filled with larval medium. From the first brood produced by each of 20 females, 40 randomly selected first instar larvae were transferred to a 3.5 cm diameter petri dish containing 5 g of larval medium. Each petri dish was placed on a layer of soil inside a plastic cup, and misted daily with distilled water. Pupae were collected from these cups after about 15 days and transferred individually to shell vials containing soil. Adults emerging inside the shell vials were transferred in full-sibling, same-sex groups of about 10 to 1.5 L cages containing sources of water, sugar, and ground beef. After about 10 days, they were paired with opposite-sex, non-sibling partners inside 50 ml vials containing sources of water and sugar, as well as perforated eppendorf tubes with larval medium. Males were removed and frozen after 24 h, and females were frozen after laying their first clutch.

Manipulation of Condition

From each of 15 broods, 24–48 h after hatching, 20 randomly selected larvae were transferred to a petri dish (3.5 cm diameter) containing 3 g of ''organic'' extra-lean ground beef (high-condition treatment), and another 20 larvae were transferred to a petri dish containing 3 g of a mixture of three parts by weight of rehydrated texturized vegetable protein (TVP) to one part ''organic'' extra-lean ground beef (low-condition treatment). The TVP-beef mixture was thoroughly homogenized into a paste, then cut into small beads to create a texture similar to the ground beef. Each petri dish was covered with a perforated lid, placed on a layer of soil inside a mesh-covered plastic cup, and misted daily with distilled water. Adult flies were frozen approximately 48 h after emergence. Three broods failed to yield sufficient progeny, and were excluded from analyses to maintain equal power for all comparisons ($n = 12$ broods).

Morphometric Analysis

From each brood-treatment combination, five randomly selected individuals of each sex were thawed and glued to entomological pins. A dissecting microscope with an ocular micrometer was then used to measure each fly's thorax length, head capsule length (head length) and width (head width), the combined lengths of the second and third segments of the left antenna (antenna length), the lengths of the tibiae of the left fore- and midlegs, the length of the R_{4+5} vein from the r-m cross-vein to the margin of the left wing (wing-vein length), and the distance between the bases of the presutural intra-alar setae (intersetal width; Fig. 1). Fore- and midtibia lengths are strongly correlated with the total lengths of the fore- and midlegs, respectively, and wing-vein length is highly correlated with total wing length ($r > 0.98$ in each case). The ideal trait to use as a body size index is the trait that best summarizes the size variation in all traits. We used thorax length as an index of body size because, of 17 morphological traits examined, it exhibited the highest loading (0.97) on the first principal component for data standardized (converted to z-scores) within trait and sex to control for differences in phenotypic variance (Bonduriansky and Rowe 2003). The degree of male trait exaggeration in absolute size was calculated as the male:female ratio of mean absolute trait sizes, and the degree of exaggeration in relative trait size was calculated as the male:female ratio of mean trait size divided by mean thorax length. This yielded an index that is >1 for traits that are absolutely or relatively larger in males than in females, but <1 for traits that are absolutely or relatively smaller in males than in females. Because sex differences in repeatabilities of trait measurements could confound comparisons of male and female responses, we estimated repeatabilities by measuring the eight traits on 10 flies of each sex twice on two separate days in randomized sequence of individuals. We found no difference between sexes in repeatabilities (mean intraclass correlation coefficient = 0.98 for each sex; paired sample *t*-test: $t = 0.07$, df = 7, $P = 0.94$).

We quantified treatment effects on absolute trait sizes for each brood as the percent difference between mean trait sizes of same-sex full-siblings from high- and low-condition treatments, standardized by mean trait size (with treatments pooled) to yield a percent response to diet manipulation. The mean percent response for each trait represents the condition dependence strength. To quantify treatment effects on relative trait sizes, we first standardized the data for all broods by subtracting the brood mean body size and trait size from each individual's values, thus centering all broods at the origin while preserving differences between treatments. We then fitted a reduced major axis (RMA) line to the data for each brood-treatment-sex combination. Assuming linear scaling and similar slopes in both treatments, and that treatment effects exceed within-treatment variation, the intercept of this line will represent the mean relative trait size in each treatment (see Fig. 2), and the response to diet manipulation can be calculated for each brood as described above. We evaluated the linearity assumption by fitting polynomial regressions to the data for each trait (within treatment and sex, with all broods pooled), and testing for the significance of the quadratic term. Of the 28 tests (7 traits × 2 treatments × 2 sexes), five yielded marginally significant results (all $P > 0.02$), but the significance resulted in each case from a single outlier point, and none remained significant after adjustment for multiple testing. No significant intertreatment differences in scaling slope were observed for any trait in either sex, even without correcting for multiple testing. Note that we do not assume isometric scaling of trait sizes with thorax length because, if data for both treatments fall on a common regression line of any slope, no intertreatment difference in intercept will be observed (see Fig. 2). The overall mean treatment effect for each trait in each sex was estimated (as above) from RMA regressions fitted by a jackknife algorithm (software created by Andrew J. Bohonak; http://www.bio.sdsu.edu/pub/andy/rma.html).

We compared mean trait sizes of same-sex high- and low-condition siblings, as well as the percent responses of males and females for each trait, using paired sample *t*-tests on brood means. We also used brood means in an analysis of variance examining effects of trait, sex, and brood on responses to diet, with sex and trait as fixed factors and brood as a random factor (df error computed using the Satterthwaite method, and type III sums of squares). Likewise, we used brood means in analyses relating condition dependence with trait exaggeration and intersexual genetic correlation, and in tests for correlation between the responses of homologous male and female traits.

Since correction for multiple testing is appropriate only when testing the ''global'' null hypothesis that two groups are identical in all comparisons (Perneger 1998; Bender and Lange 2001), we did not adjust for multiple testing in cases where different a priori hypotheses were available for different tests. Thus, a correction was made when testing the assumptions of the analysis (see above), but not for tests of our main hypotheses (see Results). No correction was made for tests of the significance of phenotypic and genetic correlations because these tests are intended only as an index of the strength of the correlations, with no particular conclusions drawn from the significance of any single correlation. Also, note that numerous significant results are highly unlikely to come about by chance alone (Moran 2003). All probabilities reported are two-tailed.

Quantitative Genetic Analysis

From each of 58 broods, 40 randomly selected first-instar larvae were provided with 5 g of ''organic'' extra-lean ground beef. Adults were frozen 24–48 h after emergence. Five adults of each sex were randomly selected from each brood, and measured as described above. The parents of each brood were also measured. Prior to analysis, data were standardized (converted to *z*-scores) within trait and sex to eliminate sex-differences in phenotypic trait variance (see Lynch and Walsh 1998). We calculated standardized residuals from ordinary least-squares regressions of trait size on thorax length for each trait, performed separately for each sex (Fig. 3). Genetic correlations between environments were calculated for each trait using the covariance between trait sizes in low- and high-quality siblings and the variances in the low- and high-condition treatments, extracted from separate ANOVAs for each trait in each environment. The significance of these genetic correlations was based on the corresponding Pearson correlations among phenotypic means. We also used the residuals in regressions of offspring means on one parent to estimate parent-offspring covariances for relative trait sizes. Intertrait genetic correlations among relative trait sizes were then calculated from the offspring-parent covariances and ''cross-variances,'' and their standard errors were estimated using the trait heritabilities (Robertson 1959; Falconer and Mackay 1996, p. 316). Intersexual genetic correlations were estimated for absolute trait sizes using the geometric method of Becker (1992), and their standard errors were calculated using Falconer's approximation (Robertson 1959; Falconer and Mackay 1996, p. 316). For genetic correlations greater than 1.0, the value of $1 - r^2$ in Falconer's approximation of the standard error was replaced by its absolute value. Intersexual phenotypic correlations were based on trait means for opposite-sex full-siblings. Further quantitative genetic analysis will be presented in a forthcoming paper.

RESULTS

More larvae survived to adulthood in the high-condition treatment (mean = 90.4%, SD = 12.5%, $n = 12$ broods) than in the low-condition treatment (mean = 80.4%, SD = 18.0%, $n = 12$ broods; paired *t*-test: $t = 2.37$, df = 11, $P = 0.0372$). High-condition flies also developed faster from larval transfer to adult emergence (high-condition: mean = 26.5 days, SD = 1.38 days, $n = 12$ broods; low-condition: mean = 29.25 days, SD = 1.86 days, $n = 12$ broods; paired *t*-test: $t = 4.65$, df = 11, $P = 0.0007$). Nonetheless, high-condition adults of

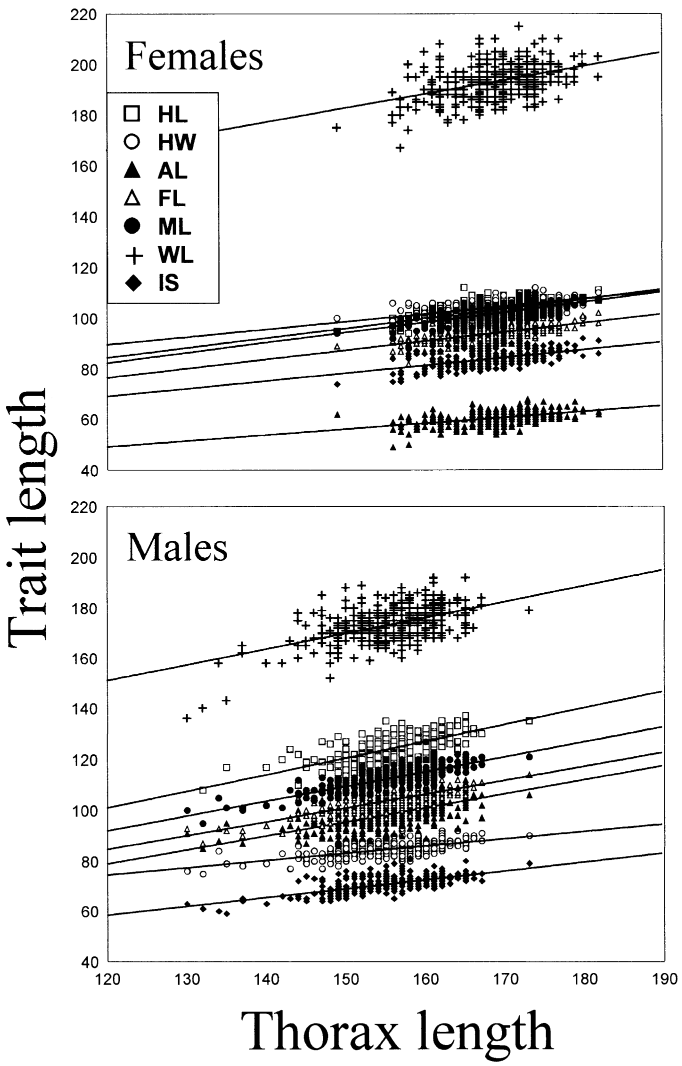

FIG. 3. Scaling of the linear dimensions of seven morphological traits with thorax length in *Prochyliza xanthostoma*, with ordinary least-squares regression lines shown for each trait.

TABLE 2. Genetic correlations between environments for absolute and relative trait sizes (significant correlations are highlighted in bold).

	Absolute trait sizes		Relative trait sizes	
Trait	Males	Females	Males	Females
Head length	0.39	**0.56**	**0.89**	**0.96**
Head width	0.02	**0.68**	0.17	**0.92**
Antenna length	0.42	0.72	**0.82**	**0.83**
Foretibia length	0.23	0.35	**1.11**	**1.05**
Midtibia length	0.33	0.41	**1.07**	**0.98**
Wing-vein length	0.28	0.38	**0.99**	**0.92**
Intersetal width	0.09	0.33	0.44	−0.02
Thorax length	0.05	0.37	—	—

both sexes were larger than their low-condition siblings (Table 1). These results for development rate and size accord with most diet manipulations (Berrigan and Charnov 1994; Day and Rowe 2002; Uetz et al. 2002).

For absolute trait sizes, genetic correlations between environments (Table 2) were stronger in females than in males for every trait, and on average (males: mean = 0.22; females: mean = 0.47; paired sample *t*-test: *n* = 8, *t* = −3.70, *P* = 0.0076). After correcting for body size, the correlations became stronger in both males (paired sample *t*-test: *n* = 7, *t* = −5.48, *P* = 0.0015) and females (paired sample *t*-test: *n* = 7, *t* = −2.36, *P* = 0.0561), but there was no longer a significant sex difference in their strength (males: mean = 0.78; females: mean = 0.81; paired sample *t*-test: *n* = 7, *t* = −0.16, *P* = 0.88).

In absolute terms (Table 3), all traits (both sexual and nonsexual) were larger in high-condition flies than in their low-condition siblings in both sexes. The strongest condition dependence was exhibited by two male sexual traits: head length and antenna length. Analysis of variance indicated significant effects of sex, trait, and brood on responses of absolute trait sizes to condition, and significant two-way interactions between factors (Table 4). Because treatments were not replicated within broods, brood effects may reflect either genetic variation or random environmental variation, or both. However, condition dependence was stronger in males than in females (main effect of sex), and varied among traits (main effect of trait). Most importantly, the sex-difference in condition dependence strength varied among traits (sex × trait interaction). Male responses exceeded female responses for seven of the eight traits (significantly so in five cases), but the greatest sex differences in condition dependence strength were observed in two sexual traits: head length and antenna length. However, after correcting for body size only head length was significantly larger in high-condition flies than in their low-condition siblings and exhibited significantly stronger condition dependence in males than in females (Table 5). Head length was more strongly condition dependent than any other sexual trait (paired sample *t*-tests, *n* = 12: compared to head width: *t* = 4.51, *P* = 0.0009; compared to antenna length; *t* = 2.11, *P* = 0.0584; compared to foretibia length: *t* = 2.28, *P* = 0.0436). However, head width (in males) and wing-vein length (in both sexes) were also significantly condition dependent, although these traits were reduced in high-condition flies.

We predicted that traits that are more exaggerated in males (relative to females) would exhibit stronger condition dependence. We also tested for (but did not expect to observe) a negative correlation between these variables in females. As predicted, a significant positive correlation was observed in males (*n* = 96, *r* = 0.29, *t* = 2.96, *P* = 0.0038), but no significant correlation was observed in females (*n* = 96, *r* = 0.09, *t* = 0.90, *P* = 0.37; Fig. 4).

If the evolution of condition dependence in males contributes to the breakdown of intersexual genetic correlations, the most strongly condition dependent traits should exhibit the lowest intersexual genetic correlations (Table 6). This prediction was supported: the strength of condition dependence in male traits covaried negatively with the intersexual genetic correlation (*n* = 96, *r* = −0.28, *t* = −2.79, *P* = 0.0063; Fig. 5). There was no significant correlation between condition dependence strength and the intersexual phenotypic correlation (*n* = 96, *r* = −0.10, *t* = −1.00, *P* = 0.32). Nonetheless, the responses to condition of homologous male

TABLE 3. Effects of diet manipulation on absolute trait sizes in *Prochyliza xanthostoma*: for each trait, the response represents the mean percent difference in trait size between high- and low-condition treatments (representing the absolute strength of condition dependence), followed by the standard error (SE), *t*-statistic (df = 11) and *P*-value (significant responses are highlighted in bold). Differences between effects in males and females are also shown, with corresponding *t*-statistics (df = 11) and *P*-values.

	Females				Males				Male-female comparison		
Trait	Effect	SE	*t*	*P*	Effect	SE	*t*	*P*	Difference	*t*	*P*
Head length	**5.1**	1.23	6.80	<0.0001	**9.0**	0.77	7.24	<0.0001	**3.83**	4.00	0.0021
Head width	**4.0**	0.87	8.67	<0.0001	**3.2**	0.48	3.81	0.0029	−0.75	−0.94	0.37
Antenna length	**4.5**	1.20	4.83	0.0005	**8.0**	0.95	6.63	<0.0001	**3.44**	3.06	0.0109
Foretibia length	**5.6**	0.93	6.10	<0.0001	**6.3**	0.91	6.96	<0.0001	0.73	0.96	0.36
Midtibia length	**4.4**	1.00	5.06	0.0004	**6.2**	0.88	6.23	<0.0001	**1.81**	2.36	0.0379
Wing-vein length	**2.5**	0.73	2.57	0.0260	**3.4**	0.97	4.70	0.0006	0.85	1.33	0.21
Intersetal width	**4.2**	1.13	3.82	0.0028	**7.6**	1.13	6.83	<0.0001	**3.37**	3.21	0.0083
Thorax length	**5.1**	0.95	6.07	<0.0001	**6.3**	0.85	6.69	<0.0001	**1.17**	2.84	0.0162

and female traits (see Tables 3 and 5) were positively correlated, both for absolute trait sizes ($n = 96$, $r = 0.60$, $t = 7.35$, $P < 0.0001$) and relative trait sizes ($n = 84$, $r = 0.34$, $t = 3.23$, $P = 0.0018$, Fig. 6).

To examine the relation between environmental and genetic effects on body shape, we also compared the genetic (and phenotypic) correlations among the body shape traits (Tables 7, 8) with the similarities among these traits in responses to diet manipulation. We computed a matrix of all possible pairwise comparisons among trait responses to condition (condition response similarities) as the ratio of the two responses, with the larger absolute value in the denominator. Thus, a condition response similarity was positive when both responses were of the same sign, but negative when the responses were of opposite sign, and its magnitude (varying between 1 and −1) reflected the relative magnitudes of the two responses. Intertrait genetic correlations were positively correlated with intertrait phenotypic correlations in both males ($n = 21$, $r = 0.88$, $t = 7.97$, $P < 0.0001$) and females ($n = 21$, $r = 0.58$, $t = 3.11$, $P = 0.0057$). In males, condition response similarities were positively correlated with the corresponding intertrait genetic correlations ($n = 21$, $r = 0.49$, $t = 2.45$, $P = 0.0244$) and phenotypic correlations ($n = 21$, $r = 0.52$, $t = 2.68$, $P = 0.0147$; Fig. 7). In contrast, females' condition response similarities were negatively correlated with intertrait genetic correlations ($n = 21$, $r = -0.45$, $t = -2.17$, $P = 0.0430$). However, the significance of this result rested on a single outlier: the unrealistically large genetic correlation (−1.49) between antenna length and intersetal width. Removing this point eliminated the significant correlation in females ($n = 20$, $r = -0.23$, $t = -1.01$, $P = 0.33$), but had no effect on the correlation in males ($n = 20$, $r = 0.49$, $t = 2.36$, $P = 0.0301$). Females' condition response similarities were not correlated significantly with intertrait phenotypic correlations ($n = 21$, $r = -0.31$, $t = -1.43$, $P = 0.17$; Fig. 7). In each of the above tests, the correlation was significantly stronger in males than in females (*Z*-tests: all $Z > 2.00$, all $P < 0.05$). Thus, environmental and genetic effects appear to be congruent in males, but there is no evidence of such a pattern in females.

DISCUSSION

Variation among Traits

Our findings support the hypothesis that sexual selection drives the evolution of condition dependence. Males exhibited stronger condition dependence than females. The strongest condition dependence and the largest sex differences in condition dependence were observed in two male sexual traits: head length and antenna length. After controlling for body size, the only trait that was more strongly condition dependent in males than in females, and the only trait that was significantly larger in high-condition flies, was male head length. This suggests that head length represents a more reliable signal of male condition than body size, a finding consistent with the observation that females prefer males with relatively elongated heads, but exhibit no preference for large males (Bonduriansky and Rowe 2003). Further support for the role of sexual selection comes from examining the relation between trait sexual dimorphism and condition dependence. Traits that are most directly targeted by sexual selection should generally exhibit the greatest size-exaggeration in males relative to females and, as a result, should also be the costliest to produce or maintain. Indeed, the traits most exaggerated in males are antenna length and head length (Table 1), both of which are employed directly in male com-

TABLE 4. Analysis of variance for mean responses of broods to diet treatment (i.e., difference in absolute trait sizes between high- and low-condition treatments), examining effects of trait, sex, and brood and their two-way interactions.

Effect	df effect	MS effect	df error	MS error	*F*	*P*
Sex	1	156.58	11.00	21.40	7.32	0.0205
Trait	7	44.53	77.00	5.02	8.88	<0.0001
Brood	11	105.44	14.43	24.62	4.28	0.0058
Sex × trait	7	15.60	77.00	1.80	8.68	<0.0001
Sex × brood	11	21.40	77.00	1.80	11.90	<0.0001
Trait × brood	77	5.02	77.00	1.80	2.79	<0.0001

TABLE 5. Effects of diet manipulation on relative trait sizes in *Prochyliza xanthostoma*: for each trait, the response represents the mean percent difference in trait size between high- and low-condition treatments (representing the strength of condition dependence relative to that of thorax length), followed by the standard error (SE), *t*-statistic (df = 11) and *P*-value (significant responses are highlighted in bold). Differences between effects in males are females are also shown, with corresponding *t*-statistics (df = 11) and *P*-values.

	Females				Males				Male-female comparison		
Trait	Effect	SE	*t*	*P*	Effect	SE	*t*	*P*	Difference	*t*	*P*
Head length	0.4	1.46	0.58	0.57	**2.3**	1.71	3.40	0.0060	**1.89**	2.54	0.0276
Head width	0.1	0.76	1.34	0.21	**−2.6**	0.48	2.72	0.0201	−2.71	−1.24	0.24
Antenna length	−1.6	0.71	1.46	0.17	0.7	0.47	0.04	0.97	2.26	1.38	0.20
Foretibia length	0.2	0.66	0.33	0.75	0.4	0.56	1.34	0.21	0.20	1.00	0.34
Midtibia length	−0.8	0.56	1.19	0.26	0.2	0.41	0.56	0.59	1.01	1.00	0.34
Wing-vein length	**−3.4**	0.64	3.28	0.0073	**−2.2**	0.45	2.57	0.0263	1.25	1.73	0.11
Intersetal width	−1.9	1.35	1.33	0.21	−0.4	1.60	1.51	0.1589	1.50	0.80	0.44

bat and courtship. We thus expected a positive correlation among male traits between the degree of trait exaggeration and the strength of condition dependence. This prediction was supported (Fig. 4). Moreover, increased condition accentuated the overall pattern of sexual dimorphism (i.e., caused males to assume a more ''male-like'' body shape), in that traits that are smaller in males than in females were reduced in size (relative to body size), whereas traits that are larger in males than in females were increased in size (see Tables 1 and 5). Females did not exhibit a clear pattern of this kind, although note that high-condition females tended to have shorter (i.e., more ''female-like'') antennae, relative to body size, than their low-condition sisters. If the entire body shape is considered as a single trait, then the female body shape may be assumed to approach the viability-selected optimum, whereas the male body shape may be regarded as a costly trait displaced from the viability-selected optimum by sexual selection. Hence, the finding that high-condition males exhibit a more male-like (i.e., more costly) body shape supports the role of sexual selection in the evolution of condition dependence.

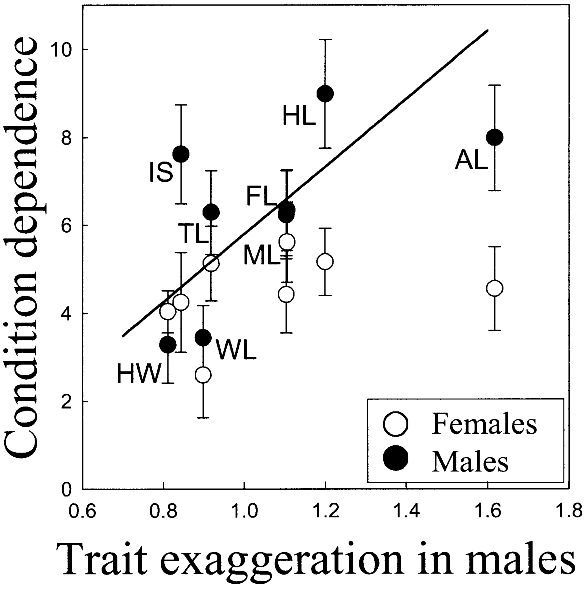

FIG. 4. Degree of trait exaggeration in males relative to females (values >1 denote traits that are larger in males) and the strength of condition dependence (with associated standard errors): male and female responses are shown for each trait, with reduced major axis regression fitted to the male data. Abbreviations as in Figure 1.

Nonetheless, we observed considerable variation in condition dependence among sexual traits, suggesting that the evolution of condition dependence may be contingent on subtle variation among traits in functions, costs or genetic architecture. The head capsule and antennae appear to function as a unit in male combat and courtship, along with the forelegs (Bonduriansky 2003; Bonduriansky and Rowe 2003), yet these traits vary considerably in condition dependence strength (Table 5): after controlling for body size, improved diet quality increased head length, decreased head width, but had little effect on antenna length or foretibia length. This variation is not explained by the intertrait genetic correlation structure (Table 7). For example, after controlling for body size, male head length and antenna length exhibit a strong, positive genetic correlation but differ in condition dependence, whereas male antenna length and foretibia length exhibit a weak genetic correlation but similar condition dependence. Nor is the variation likely to be explained by patterns of sexually antagonistic selection (see Lande 1980, 1987; Rice 1984): for example, the intersexual genetic correlation for antenna length is lower than that for head length (Table 6).

Variation in condition dependence may result from subtle differences in the form of selection, where some traits (i.e., relative head length) are under directional sexual selection, favoring strong condition dependence, while others (i.e., relative antenna length) are under net stabilizing sexual selection, favoring condition dependence of similar strength to that of thorax length. Variation in condition dependence

TABLE 6. Intersexual phenotypic (r_P) and genetic (r_{MF}) correlations and their standard errors (SE) for absolute trait sizes in *Prochyliza xanthostoma* (significant correlations are highlighted in bold).

Trait	r_P (SE)	r_{MF} (SE)
Head length	**0.56** (0.111)	**0.49** (0.179)
Head width	**0.46** (0.119)	1.62 (1.558)
Antenna length	**0.29** (0.128)	0.21 (0.597)
Foretibia length	**0.67** (0.099)	0.41 (2.622)
Midtibia length	**0.69** (0.097)	**1.22** (0.245)
Wing-vein length	**0.78** (0.084)	**0.97** (0.004)
Intersetal width	**0.74** (0.090)	**1.40** (0.222)
Thorax length	**0.80** (0.081)	**0.90** (0.027)

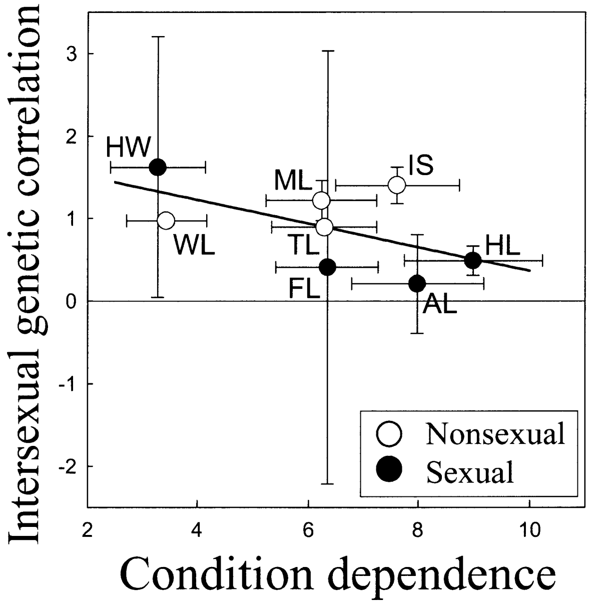

FIG. 5. Condition dependence strength in males and intersexual genetic correlation, with associated standard errors and fitted reduced major axis regression.

strength may also be related to the costs of trait expression. A trait that is more costly to build or maintain should reflect condition more reliably because, on average, high- and low-condition individuals will exhibit greater differences in the expression of such a trait (Rowe and Houle 1996). Increased head length may be more costly than increased antenna or foretibia length because the head capsule is a larger trait, or because its elongation imposes greater viability costs (e.g., by reducing visual acuity). Variation in condition dependence strength has also been observed among morphological traits associated with male display in the peacock (Møller and Petrie 2002). Likewise, diverse traits associated with male mating success in barn swallows were found to exhibit varying degrees of susceptibility to the effects of radiation, which appeared to affect the genetic component of condition (Møller and Mousseau 2003).

Interestingly, after correcting for body size, wing-vein length (a nonsexual trait) exhibited significant condition dependence in both sexes. However, the form of condition dependence exhibited by this trait clearly differs from that expected to evolve in sexually selected traits, and observed in head length, in that high-condition individuals invest relatively *less* in the trait, producing shorter wings than their low-condition siblings. The wings do not appear to be directly employed in male sexual competition, and are clearly not sexually selected in females (Bonduriansky 2003). Nor is wing-vein length strongly genetically correlated with any condition-dependent sexual trait (Table 7). Rather, assuming that variation in this trait reflects variation in overall wing shape, this response may have an adaptive function in compensating for condition effects on body shape or relative weight so as to maintain flight performance (although it is not clear how high-condition flies might benefit from having relatively shorter wings). This finding is consistent with the results of David et al. (2000), who detected significant condition dependence in relative wing length in male and female stalk-eyed flies (*Cyrtodiopsis dalmanii*). A similar pattern of condition dependence was also exhibited by head width, but in this case the pattern is probably a consequence of the negative genetic correlation between this trait and a strongly condition dependent trait (head length; Table 7).

Our results thus illustrate the need to compare multiple sexual and nonsexual traits. Had we compared a single sexual

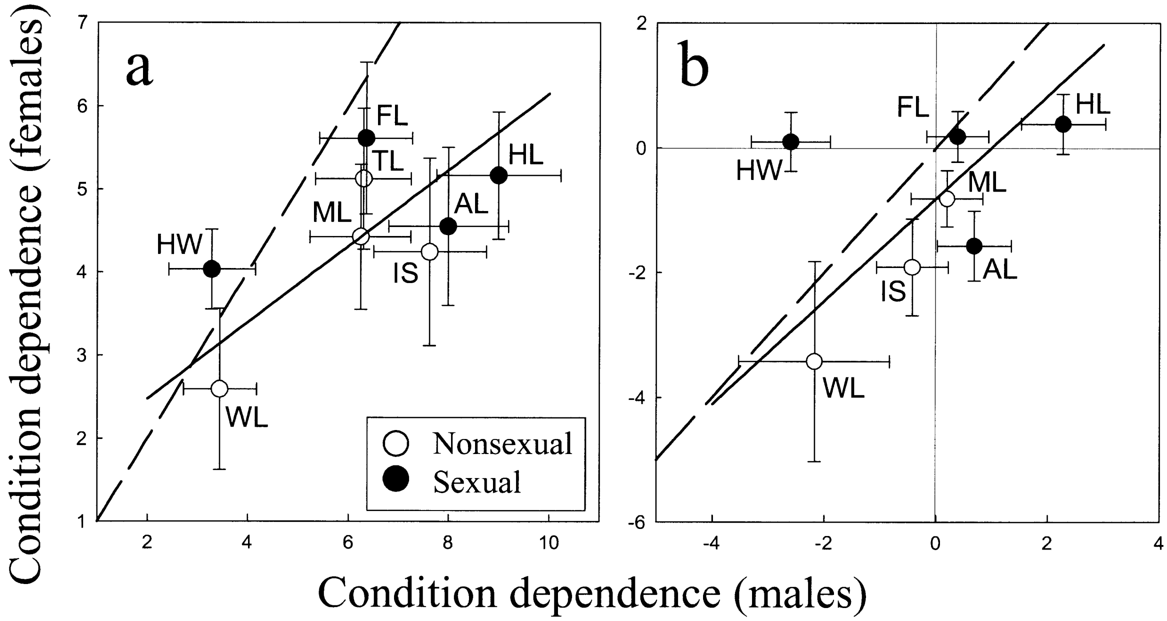

FIG. 6. Covariation of responses to condition in homologous male and female traits (a, absolute trait sizes; b, relative trait sizes), with associated standard errors and fitted reduced major axis regressions. Points below the dashed line in each plot (y = x) represent traits that are more strongly condition dependent in males than in females.

TABLE 7. Within-sex genetic correlations among relative trait sizes in *Prochyliza xanthostoma*. Values above the diagonal are based on father-son covariances, whereas values below the diagonal are based on mother-daughter covariances, with standard errors shown in parentheses (significant correlations are highlighted in bold).

Trait	HL	HW	AL	FL	ML	WL	IS
Head length (HL)	—	−0.39 (0.24)	**0.89** (0.04)	0.09 (0.18)	0.29 (0.22)	−0.18 (0.18)	0.20 (0.28)
Head width (HW)	−0.39 (0.35)	—	−**0.56** (0.18)	−0.17 (0.23)	−0.30 (0.29)	0.15 (0.23)	0.33 (0.34)
Antenna length (AL)	0.46 (0.23)	0.37 (0.35)	—	−0.00 (0.17)	0.22 (0.21)	−0.12 (0.17)	−0.10 (0.27)
Foretibia length (FL)	0.02 (0.37)	−**0.87** (0.13)	−0.21 (0.75)	—	**0.81** (0.07)	−0.10 (0.15)	0.08 (0.25)
Midtibia length (ML)	0.28 (0.25)	−0.24 (0.36)	0.34 (0.50)	**0.78** (0.14)	—	−0.18 (0.20)	−0.03 (0.33)
Wing-vein length (WL)	0.36 (0.24)	−0.54 (0.28)	0.34 (0.51)	−0.05 (0.36)	0.16 (0.25)	—	−0.33 (0.22)
Intersetal width (IS)	−0.24 (0.61)	−0.16 (0.89)	−1.49 (1.66)	−0.62 (0.51)	0.23 (0.57)	−0.41 (0.51)	—

trait with a single nonsexual trait (like most studies of condition dependence), it is easy to see that our results would have depended substantially on our choice of traits (see Table 5). Head length exhibits strong condition dependence after controlling for body size, but antenna length (the most sexually dimorphic of the sexual traits), foretibia length, and head width do not. Thus, for example, head length accords with the pattern exhibited by eyestalk length in diopsid flies, whereas the other sexual traits do not (see David et al. 2000; Cotton et al. 2004b). Comparisons of multiple traits, although less simple to interpret, are more likely to shed light on the factors that contribute to variation among traits in condition dependence.

Environmental and Genetic Effects on Body Shape

Genetic correlations between environments (Table 2) were stronger in females than in males when based on absolute trait sizes. After correcting for body size, however, the correlations became stronger in every trait except intersetal width, and very similar on average in the two sexes. Absolute trait sizes may yield low correlations as a result of weak genetic effects on body size, especially in males. Conversely, genetic effects on body shape appear to be strong and similar in both sexes, and there is no evidence that different sets of genes affect relative trait sizes in the different environments. The two traits measured perpendicular to the long axis of the body (head width and intersetal width), both of which were very weakly condition dependent, exhibited weak correlations in males both before and after controlling for body size. In contrast, the most condition-dependent traits (head length and antenna length) both exhibited high genetic correlations between environments, suggesting that sensitivity to environmental factors is not associated with diminished genetic effects on trait expression.

We found evidence of congruent responses to environmental and genetic factors in males (Fig. 7). This suggests that genetic variation at loci associated with the ability to acquire or use resources, and environmental variation in resource abundance, have similar effects on male body shape (see Griffith et al. 1999; Kotiaho et al. 2001; Tschirren et al. 2003). For example, male head length and width are negatively genetically correlated and exhibit opposite responses to condition, whereas head length and antenna length are positively genetically correlated and respond similarly to condition (although head length responds more strongly). Congruence of environmental and genetic factors can be accounted for by a simple model of condition-dependent resource allocation, whereby two factors—ambient resource abundance and genes that determine resource extraction efficiency—both affect the total quantity of available metabolic resources, which is an important component of condition, whereas the pattern of resource allocation is determined by a separate set of genes (Fig. 8). Consequently, increased availability of ambient resources (i.e., environmental factors) would have a similar effect on allocation to a condition-dependent trait as does increased quality of genes that determine resource extraction efficiency (i.e., genetic factors). Congruence of environmental and genetic effects on body shape variation has also been reported by Cheverud (1988) and Roff (1996), based on the covariation of genetic and phenotypic correlations (a pattern also observed in the present study). However, phenotypic correlations integrate both environmental and genetic effects, whereas the present study compared genetic correlations with patterns resulting from purely environmental factors.

In contrast, we found no evidence of congruence between environmental and genetic factors in females. We may have failed to detect the pattern in females because the standard

TABLE 8. Within-sex phenotypic correlations among relative trait sizes in *Prochyliza xanthostoma*. Values above the diagonal are for males, whereas values below the diagonal are for females, with standard errors shown in parentheses (significant correlations are highlighted in bold).

Trait	HL	HW	AL	FL	ML	WL	IS
Head length (HL)	—	−**0.14** (0.052)	**0.77** (0.033)	**0.24** (0.051)	**0.29** (0.050)	−0.02 (0.053)	**0.14** (0.052)
Head width (HW)	0.06 (0.053)	—	−0.05 (0.053)	**0.17** (0.052)	**0.22** (0.051)	0.02 (0.053)	**0.28** (0.051)
Antenna length (AL)	**0.43** (0.048)	0.08 (0.053)	—	**0.24** (0.051)	**0.33** (0.050)	−0.05 (0.053)	0.09 (0.052)
Foretibia length (FL)	**0.28** (0.051)	0.01 (0.053)	**0.28** (0.051)	—	**0.73** (0.036)	0.10 (0.052)	**0.17** (0.052)
Midtibia length (ML)	**0.22** (0.052)	**0.11** (0.052)	**0.30** (0.050)	**0.78** (0.033)	—	0.06 (0.053)	**0.14** (0.052)
Wing-vein length (WL)	0.07 (0.053)	−0.10 (0.053)	**0.16** (0.052)	**0.17** (0.052)	**0.17** (0.052)	—	**0.11** (0.052)
Intersetal width (IS)	**0.20** (0.052)	**0.36** (0.049)	0.09 (0.053)	0.10 (0.053)	**0.16** (0.052)	0.04 (0.053)	—

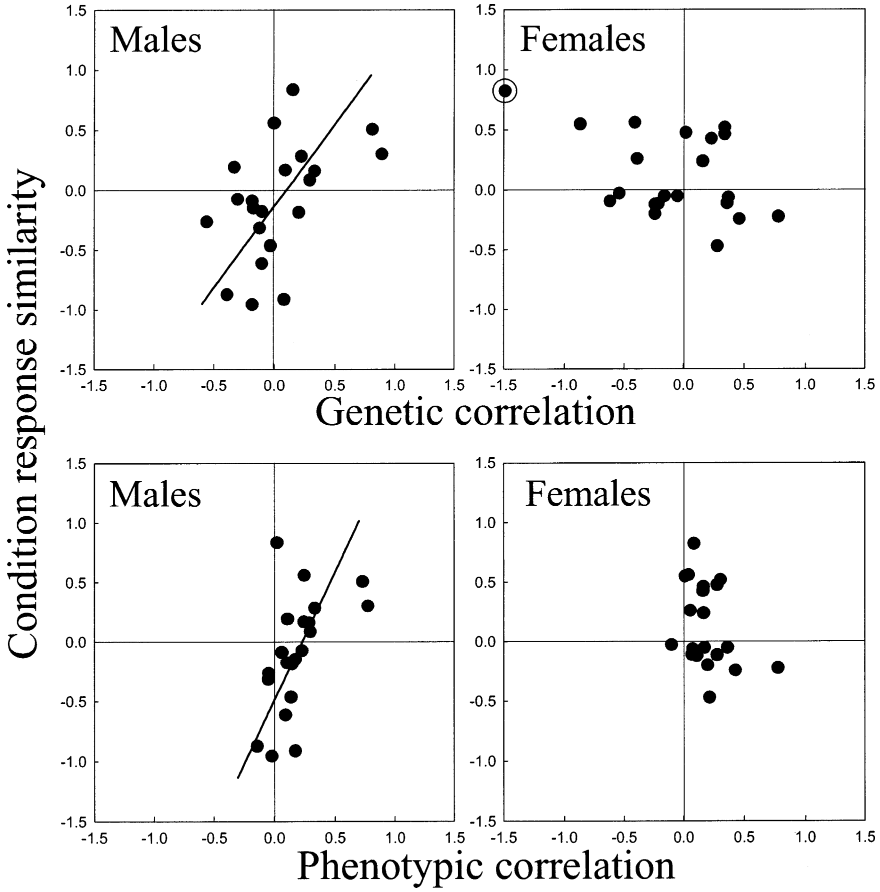

FIG. 7. Covariation of environmental and genetic effects: condition response similarities (see text) are positively correlated with both genetic and phenotypic correlations among traits in males, but no such pattern is evident in females. The circled outlier point represents an unrealistically large negative correlation (see text).

errors of mother-daughter covariances substantially exceeded the standard errors of father-son covariances (Table 7). However, this explanation is implausible because a similar sex difference was observed using phenotypic correlations, which exhibit very similar standard errors in males and females (Fig. 7, Table 8). Thus, the observed pattern appears to reflect a real sex difference in genetic architecture and may be related to the lesser importance of condition dependence in determining body shape in females, compared with males.

Intertrait genetic correlations may often result from pleiotropy engendered by patterns of developmental integration and trade-offs in resource allocation among traits (Rowe and Houle 1996; Nijhout and Emlen 1998; Emlen 2001; Conner 2002). However, when considering body shape (i.e., trait size in relation to body size), intertrait correlations may be determined by the relative condition dependence strengths of the traits of interest and of body size. This is because, although increasing condition causes all traits to increase in absolute size, whether it causes a given trait to increase or decrease in relative size (i.e., trait size in relation to body size) depends on the condition-dependent rate of allocation to that trait, relative to the condition-dependent rate of al-

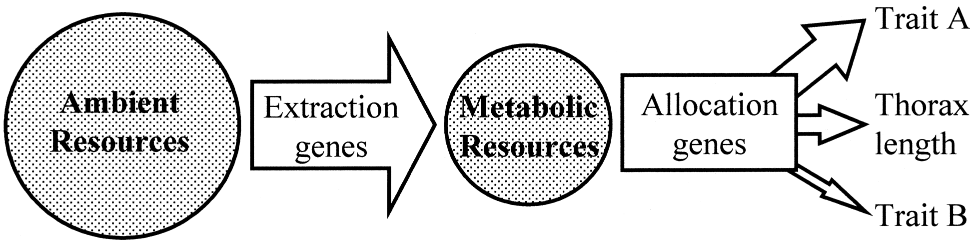

FIG. 8. A model of environmental and genetic effects on condition-dependent trait expression. Congruence of environmental and genetic effects is observed because ambient resource availability and resource extraction genes both affect the quantity of metabolic resources available for allocation to morphological traits, while allocation genes determine the relative rates of allocation to those traits. Thus, the quantity of metabolic resources can be increased either by augmenting the pool of ambient resources, or by augmenting the quality of the extraction genes.

location to the index of body size (e.g., thorax length). Thus, if two traits both exhibit greater (or lesser) condition dependence than thorax length, they will respond in the same direction to a change in condition, whether this results from genetic or environmental factors (see above). In contrast, if one trait is more condition dependent than thorax length (e.g., trait A in Fig. 8), whereas the other trait is less condition dependent than thorax length (e.g., trait B in Fig. 8), these traits will respond in opposite directions to a change in condition. To illustrate this more formally, we use a modified version of the model of condition-dependent allocation provided by Cotton et al. (2004a): $s = t + a(\upsilon) \times \upsilon$, where s is the size of a sexual trait, determined by a condition-independent constant; t, a condition-dependent allocation function, $a(\upsilon)$; and condition, υ (Fig. 9). Such patterns will arise in any study examining the condition dependence of body shape, where condition effects on trait size must be considered in relation to condition effects on body size. Indeed, this pattern suggests that the matrix of intertrait genetic or phenotypic correlations will depend, to some extent, on the choice of body size index, highlighting the importance of selecting an appropriate index (see Materials and Methods).

Condition Dependence and Intralocus Sexual Conflict

If the evolution of condition dependence in males contributes to the breakdown of intersexual genetic correlations, then condition dependence strength should covary negatively with intersexual genetic correlation. Our data are consistent with this hypothesis (Fig. 5). Nonetheless, the observed pattern is also predicted by an alternative hypothesis that reverses the direction of cause and effect: the extent to which condition dependence can evolve in a given trait may be constrained by the strength of the intersexual genetic correlation. Resolving this issue will require a more complete understanding of the genetic basis of condition dependence, as well as the genetic mechanisms involved in the breakdown of intersexual genetic correlations.

The responses of homologous male and female traits to condition were positively correlated. This result suggests that at least some loci involved in condition dependence are expressed in both sexes, resulting in an intersexual genetic correlation for condition dependence. Moreover, the stronger responses of males are consistent with the view that the evolution of condition dependence is driven primarily by sexual selection on males (see Bonduriansky 2003; Bonduriansky and Rowe 2003). The interpretation of the observed phenotypic correlation as reflecting an intersexual genetic correlation is supported by the strong covariation between intersexual phenotypic and genetic correlations for morphological traits in *P. xanthostoma* (see Table 6). An intersexual genetic correlation for condition dependence would represent a genetic constraint on the divergence of the sexes in their patterns of resource allocation to the precursors of adult morphological traits during the larval and pupal stages. Thus, it is not clear whether the evolution of condition dependence has mitigated or exacerbated the severity of intralocus sexual conflict in this species. As a further test for an intersexual genetic correlation for condition dependence, it would be useful to compare responses to diet manipulation in separately reared male and female siblings.

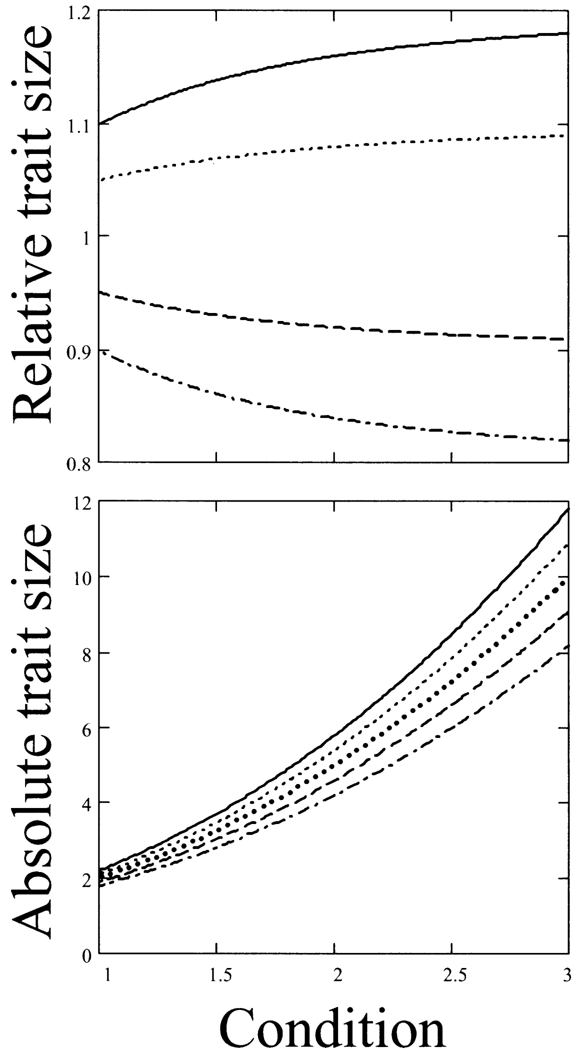

FIG. 9. Change with increasing condition in the absolute sizes of five hypothetical traits, and in the relative sizes of four of those traits, with one trait (heavy dotted line in the lower figure) acting as an index of body size: a numerical example, based on a modified version of the model of Cotton et al. (2004a; see text) with five traits of varying condition dependence ($t = 1.0$ for all traits; $a(\upsilon) = 0.8{\cdot}\upsilon$, $0.9{\cdot}\upsilon$, $1.0{\cdot}\upsilon$, $1.1{\cdot}\upsilon$, $1.2{\cdot}\upsilon$ for the five traits, respectively), shows that increasing condition causes all traits to increase in absolute size (lower panel), but not in relative size (upper panel). If one trait is used as an index of body size (in this example, the trait with ($a[\upsilon] > 1.0{\cdot}\upsilon$), less condition-dependent traits [$a(\upsilon) = 1.0{\cdot}\upsilon$] become relatively smaller as condition increases, whereas more condition-dependent traits [$a(\upsilon) < 1.0{\cdot}\upsilon$] become relatively larger.

Assumptions

We assume that treatment effects on trait expression did not result from differences in larval mortality (i.e., selection). Although mortality rate was about 10% lower in the high-condition treatment, this differential selection is unlikely to account for observed treatment effects because the traits compared are expressed only in adults (albeit the precursors of these traits may impose some viability costs at the larval stage). We also assume that intertreatment differences in development rate were of little importance. Although high-condition flies developed more quickly, they were significantly larger as adults (Table 3), suggesting that the effects of rapid development were overwhelmed by the effects of increased resource abundance. Finally, our intertrait comparisons (see Figs. 6, 7, 8) treat each trait as an independent observation, despite the fact that all traits are linked by a genetic correlation structure and correlated selection. However, the operational assumption of trait independence seems justified given that genetic correlations among traits are generally quite weak (Table 7), and neither the genetic correlations nor patterns of correlated selection seem sufficient to explain the variation among traits in condition dependence (see above). Our approach also follows the convention in studies that examine intertrait variation (e.g., see Møller and Mousseau 2003).

Conclusions

The strongest condition dependence was exhibited by two male sexual traits: head length and antenna length. However, only head length continued to exhibit strong condition dependence after controlling for body size. Other male sexual traits (head width and foretibia length) were weakly condition dependent, as were males' nonsexual traits and all female traits. Among all male traits, condition dependence strength was positively correlated with the degree of trait exaggeration, so that increased condition tended to accentuate the overall pattern of sexual dimorphism in body shape. Moreover, perhaps because of the considerable importance of condition dependence as a determinant of male (but not female) body shape, we detected a congruence between environmental and genetic effects on body shape in males, but no evidence of such a congruence in females. These findings are consistent with the hypothesis that sexual selection drives the evolution of condition dependence, but also suggest that the evolution of condition dependence is contingent on subtle variations in trait functions, costs, or genetic architecture. The variation in condition dependence observed among sexual traits also highlights the importance of comparing multiple traits. Our data are also consistent with the hypothesis that the evolution of condition dependence contributes to the breakdown of intersexual genetic correlations. Nonetheless, because selection for condition dependence in males appears to have produced a correlated response in females, it is not clear whether the evolution of condition dependence mitigates or exacerbates the intensity of intralocus sexual conflict in this species.

ACKNOWLEDGMENTS

We thank I. Dworkin, J. Kotiaho, T. Tregenza and an anonymous reviewer for insightful comments and suggestions. Funding was provided by the Natural Sciences and Engineering Research Council of Canada (NSERC) through Postgraduate Scholarship B and postdoctoral fellowships to RB, and through research funding to LR from NSERC, the Premier's Research Excellence Award, and the Canada Research Chairs.

LITERATURE CITED

Andersson, M. 1982. Sexual selection, natural selection and quality advertisement. Biol. J. Linn. Soc. 17:375–393.

Becker, W. A. 1992. Manual of quantitative genetics. 5th ed. Academic Enterprises, Pullman, WA.

Bender, R., and S. Lange. 2001. Adjusting for multiple testing—when and how? J. Clinical Epidemiol. 54:343–349.

Berrigan, D., and E. L. Charnov. 1994. Reaction norms for age and size at maturity in response to temperature—a puzzle for life historians. Oikos 70:474–478.

Bonduriansky, R. 2003. Layered sexual selection: a comparative analysis of sexual behaviour within an assemblage of piophilid flies. Can. J. Zool. 81:479–491.

Bonduriansky, R., and L. Rowe. 2003. Interactions among mechanisms of sexual selection on male body size and head shape in a sexually dimorphic fly. Evolution 57:2046–2053.

Carranza, J., and S. J. H. Detrucios. 1993. Condition dependence and sex traits in the male great bustard. Ethology 94:187–200.

Cheverud, J. M. 1988. A comparison of genetic and phenotypic correlations. Evolution 42:958–968.

Conner, J. K. 2002. Genetic mechanisms of floral trait correlations in a natural population. Nature 420:407–410.

Cotton, S., K. Fowler, and A. Pomiankowski. 2004a. Do sexual ornaments demonstrate heightened condition-dependent expression as predicted by the handicap hypothesis? Proc. R. Soc. Lond. B 271:771–783.

———. 2004b. Condition dependence of sexual ornament size and variation in the stalk-eyed fly *Cyrtodiopsis dalmanni* (Diptera: Diopsidae). Evolution 58:1038–1046.

Darwin, C. R. 1874. The descent of man and selection in relation to sex. 2nd ed. Hurst and Company, New York.

David, P., A. Hingle, D. Greig, A. Rutherford, A. Pomiankowski, and K. Fowler. 1998. Male sexual ornament size but not asymmetry reflects condition in stalk-eyed flies. Proc. R. Soc. Lond. B 265:2211–2216.

David, P., T. Bjorksten, K. Fowler, and A. Pomiankowski. 2000. Condition dependent signalling of genetic variation in stalk-eyes flies. Nature 406:186–188.

Day, T., and R. Bonduriansky. 2004. Intralocus sexual conflict can drive the evolution of genomic imprinting. Genetics 167:1537–1546.

Day, T., and L. Rowe. 2002. Developmental thresholds and the evolution of reaction norms for age and size at life-history transitions. Am. Nat. 159:338–350.

de Visser, J. A. G. M., J. Hermisson, G. P. Wagner, L. Ancel Meyers, H. Bagheri-Chaichian, J. L. Blanchard, L. Chao, J. M. Cheverud, S. F. Elena, W. Fontana, G. Gibson, T. F. Hansen, D. Krakauer, R. C. Lewontin, C. Ofria, S. H. Rice, G. von Dassow, A. Wagner, and M. C. Whitlock. 2003. Evolution and detection of genetic robustness. Evolution 57:1959–1972.

Emlen, D. J. 1997. Diet alters male horn allometry in the beetle *Onthophagus acuminatus* (Coleoptera: Scarabaeidae). Proc. R. Soc. Lond. B 264:567–574.

———. 2001. Costs and the diversification of exaggerated animal structures. Science 291:1534–1536.

Falconer, D. S. and T. F. C. Mackay. 1996. Introduction to quantitative genetics. 4th ed. Longman, New York.

Fisher, R. A. 1915. The evolution of sexual preference. Eugenics Rev. 7:184–192.

Getty, T. 1998a. Handicap signalling: when fecundity and viability do not add up. Anim. Behav. 56:127–130.

———. 1998b. Reliable signaling need not be a handicap. Anim. Behav. 56:253–255.

———. 2002. Signaling health versus parasites. Am. Nat. 159:363–371.

Gray, D. A. and G. Eckhardt. 2001. Is cricket courtship song condition dependent? Anim. Behav. 62:871–877.

Griffith, S. C., I. P. F. Owens, and T. Burke. 1999. Environmental determination of a sexually selected trait. Nature 400:358–360.

Hunt, J., L. F. Bussière, M. D. Jennions, and R. Brooks. 2004. What is genetic quality? Trends Ecol. Evol. 19:329–333.

Johnstone, R. A., and A. Grafen. 1992. Error-prone signaling. Proc. R. Soc. Lond. B 248:229–233.

———. 1993. Dishonesty and the handicap principle. Anim. Behav. 46:759–764.

Kotiaho, J. S. 2000. Testing the assumptions of condition dependent handicap theory: costs and condition dependence of a sexually selected trait. Behav. Ecol. Sociobiol. 48:188–194.

———. 2002. Sexual selection and condition dependence of courtship display in three species of horned beetles. Behav. Ecol. 13: 791–799.

Kotiaho, J. S., L. W. Simmons, and J. L. Tomkins. 2001. Towards a resolution of the lek paradox. Nature 410:684–686.

Lande, R. 1980. Sexual dimorphism, sexual selection, and adaptation in polygenic characters. Evolution 34:292–305.

———. 1987. Genetic correlations between the sexes in the evolution of sexual dimorphism and mating preferences. Pp. 83–94 *in* J. W. Bradbury and M. B. Andersson, eds. Sexual selection: testing the alternatives, John Wiley and Sons, New York.

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

McAlpine, D. K. 1979. Agonistic behaviour in *Achias australis* (Diptera, Platystomatidae) and the significance of eye-stalks. Pp. 221–230 *in* M. S. Blum and N. A. Blum, eds. Sexual selection and reproductive competition in insects, Academic Press, New York.

McAlpine, J. F. 1977. A revised classification of the Piophilidae, including ''Neottiophilidae'' and ''Thyreophoridae'' (Diptera: Schizophora). Mem. Entomol. Soc. Can. 103:1–66.

Møller, A. P., and T. A. Mousseau. 2003. Mutation and sexual selection: a test using barn swallows from Chernobyl. Evolution 57:2139–2146.

Møller, A. P., and M. Petrie. 2002. Condition dependence, multiple sexual signals, and immunocompetence in peacocks. Behav. Ecol. 13:248–253.

Moran, M. D. 2003. Arguments for rejecting the sequential Bonferroni in ecological studies. Oikos 100:403–405.

Nijhout, H. F., and D. J. Emlen. 1998. Competition among body parts in the development and evolution of insect morphology. Proc. Natl. Acad. Sci. USA 95:3685–3689.

Nur, N., and O. Hasson. 1984. Phenotypic plasticity and the handicap principle. J. Theor. Biol. 110:275–297.

Perneger, T. V. 1998. What's wrong with Bonferroni adjustments. Br. Med. J. 316:1236–1238.

Perrier, G., F. de Lope, A. P. Møller, and P. Ninni. 2002. Structural coloration and sexual selection in the barn swallow *Hurundo rustica*. Behav. Ecol. 13:728–736.

Price, T., D. Schluter, and N. E. Heckman. 1993. Sexual selection when the female directly benefits. Biol. J. Linn. Soc. 48: 187–211.

Rhen, T. 2000. Sex-limited mutations and the evolution of sexual dimorphism. Evolution 54:37–43.

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

Rice, W. R. and A. K. Chippindale. 2001. Intersexual ontogenetic conflict. J. Evol. Biol. 14:685–693.

———. 2002. The evolution of hybrid infertility: perpetual coevolution between gender-specific and sexually antagonistic genes. Genetica 116:179–188.

Robertson, A. 1959. The sampling variance and the genetic correlation coefficient. Biometrics 15:469–485.

Roff, D. A. 1996. The evolution of genetic correlations: an analysis of patterns. Evolution 50:1392–1403.

Rowe, L., and D. Houle. 1996. The lek paradox and the capture of genetic variance by condition dependent traits. Proc. R. Soc. Lond. B 263:1415–1421.

Schluter, D., T. D. Price, and L. Rowe. 1991. Conflicting selection pressures and life history trade-offs. Proc. R. Soc. Lond. B 246: 11–17.

Tomkins, J. L., J. Radwan, J. S. Kotiaho, and T. Tregenza. 2004. Genic capture and resolving the lek paradox. Trends Ecol. Evol. 19:323–328.

Tschirren, B., P. S. Fitze, and H. Richner. 2003. Proximate mechanisms of variation in the carotenoid-based plumage coloration of nestling great tits (*Parus major* L.). J. Evol. Biol. 16:91–100.

Uetz, G. W., R. Papke, and B. Kilinc. 2002. Influence of feeding regime on body size, body condition and a male secondary sexual character in *Schizocosa ocreata* wolf spiders (Araneae, Lycosidae): condition dependence in a visual signaling trait. J. Arachnol. 30:461–469.

West-Eberhard, M.-J. 1986. Alternative adaptations, speciation, and phylogeny (a review). Proc. Natl. Acad. Sci. USA 83: 1388–1392.

———. 1989. Phenotypic plasticity and the origins of diversity. Annu. Rev. Ecol. Syst. 20:249–278.

———. 2003. Developmental plasticity and evolution. Oxford Univ. Press, Oxford, U.K.

Wilkinson, G. S., and M. Taper. 1999. Evolution of genetic variation for condition dependent traits in stalk-eyed flies. Proc. R. Soc. Lond. B 266:1685–1690.

Zahavi, A. 1975. Mate selection—a selection for a handicap. J. Theor. Biol. 53:205–214.

———. 1977. The cost of honesty. J. Theor. Biol. 67:603–605.

Corresponding Editor: T. Tregenza