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The Evolution of Condition-Dependent Sexual Dimorphism

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ABSTRACT: Theory suggests that the net benefit of allocating resources to a sexual trait depends both on the strength of sexual selection on that trait and on individual condition. This predicts a tight coevolution between sexual dimorphism and condition dependence and suggests that these patterns of within-sex and between-sex variation may share a common genetic and developmental basis. Although condition-dependent expression of sexual traits is widely documented, the extent of covariation between condition dependence and sexual dimorphism remains poorly known. I investigated the effects of condition (larval diet quality) on multivariate sexual dimorphism in the fly *Telostylinus angusticollis* (Neriidae). Condition determined the direction of sexual size dimorphism and modulated sexual shape dimorphism by affecting allometric slopes and/or intercepts of sexually homologous traits in both sexes. Although the greatest responses to condition manipulation were observed in male sexual traits, both sexual and nonsexual traits exhibited substantial variation in the nature and magnitude of condition effects. Nonetheless, condition dependence and sexual dimorphism were remarkably congruent: variation in the strength of condition effects on male traits explained more than 90% of the variation in the magnitude of sexual dimorphism, whether quantified in terms of trait size or allometric slope. The genetic mechanisms that give rise to multivariate sexual dimorphism in body shape thus function in a strongly condition-dependent manner in this species, suggesting a common genetic basis for body shape variation within and between sexes.

Keywords: body shape, body size, condition dependence, diet, genetic architecture, sexual selection, sexual dimorphism, *Telostylinus angusticollis*, Neriidae.

Theory predicts that sexual selection acting on males will favor exaggeration of the targeted male traits if individuals with larger traits achieve higher mating success (Darwin 1874). This leads to the evolution of sexual dimorphism, whereby male sexual traits are absolutely or relatively larger than homologous traits of females. However, theory also suggests that traits targeted by sexual selection should evolve heightened condition dependence, a form of developmental plasticity that links the degree of trait expression to the quantity of metabolic resources available to the individual, optimizing the trade-off between viability and reproduction (McAlpine 1979; Andersson 1982, 1986; Nur and Hasson 1984; Rowe and Houle 1996). This prediction applies to any trait exaggerated by sexual selection, including signal, weapon, or other devices employed in sexual competition. Thus, although virtually all traits may be affected by condition to some degree, sexually selected traits are expected to evolve greater sensitivity to condition than ordinary metric traits. Consequently, sexual selection on male traits is expected to result in the evolution of condition-dependent sexual dimorphism, where variation in trait expression among individual males (but not females) reflects variation in condition. Yet, despite the clear theoretical link between condition dependence and sexual dimorphism, the coevolution and degree of genetic and developmental integration of these traits remain poorly understood.

Condition dependence and sexual dimorphism both reflect patterns of phenotypic variation rather than a particular phenotypic state. Thus, unlike most traits, they cannot be measured on a single individual at a single point in time. Rather, both condition dependence and sexual dimorphism can be thought of as reaction norms, whereby condition dependence represents phenotypic variation across levels of metabolic resource availability, while sexual dimorphism represents phenotypic variation across sexes. These reaction norms are traits in that they reflect the phenotypic effects of a particular genotype. It is not clear to what extent the genetic mechanisms that produce condition dependence (within-sex variation) in a given trait may also contribute to sexual dimorphism (between-sex variation) in the same trait. In other words, the magnitude and developmental basis of the genetic correlation between condition dependence and sexual dimorphism are unknown.

Numerous studies have found that sexually dimorphic secondary sexual traits that function as courtship signals

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and/or weapons exhibit condition-dependent expression (McAlpine 1979; Andersson 1982, 1986; Emlen 1994, 1997; Griffith et al. 1999; Post et al. 1999; Møller and Petrie 2002; Cotton et al. 2004*a*, 2004*b*; Bonduriansky and Rowe 2005*b*), and a few studies have reported that the degree of sexual dimorphism is modulated by environmental factors (David et al. 1994; Post et al. 1999; Karan et al. 2000; Weladji et al. 2005). These findings are consistent with theory. However, as Cotton et al. (2004*b*) have concluded after reviewing the literature, few such studies provide compelling support for the hypothesis that sexual selection drives the evolution of heightened condition dependence. They pointed out that much of the evidence is correlational rather than experimental and that many studies failed to include a nonsexual control trait or to control for body size. It can be added that the traits examined (typically, the most interesting sexual trait and, sometimes, an arbitrarily chosen nonsexual trait) may represent a strongly biased sample. Because such single-trait or paired-trait studies cannot detect variation in condition dependence among sexual or nonsexual traits, they could yield misleading results. As Arnqvist and Thornhill (1998) pointed out, stronger inference can be achieved through analysis of variation in the magnitude of condition dependence among multiple traits within species (e.g., Møller and Petrie 2002; Bonduriansky and Rowe 2005*b*). Unlike single-trait or paired-trait studies, multitrait comparisons permit quantitative tests of complex predictions based on continuous variation in the parameters of interest. It is clear that both sexual and nonsexual traits can vary in the strength and nature of condition dependence (Arnqvist and Thornhill 1998; Bonduriansky and Rowe 2005*b*), but the causes of this variation have received little attention and remain poorly understood.

Theory predicts positive covariation among traits between the degree of sexual dimorphism and the degree of condition dependence. Because male secondary sexual traits are thought to be displaced from the viability optimum by sexual selection, whereas the homologous traits of females are assumed to be at or near the viability optimum, the extent of phenotypic divergence between the sexes (i.e., the degree of sexual dimorphism) for a given trait should, on average, reflect the viability costs of trait expression for males (although these costs may vary among traits; see "Discussion"). The magnitude of the viability costs is, in turn, expected to determine the degree of condition dependence that evolves (Rowe and Houle 1996). Consequently, the more dimorphic the trait, the more strongly it should respond to variation in condition. Only one previous study has tested this prediction: an analysis of body shape variation in *Prochyliza xanthostoma* (Diptera: Piophilidae) revealed positive covariation between condition dependence and sexual dimorphism (Bonduriansky and Rowe 2005*b*). However, this result was inconclusive because it explained only 8% of the variance.

The Australian fly *Telostylinus angusticollis* (Diptera: Neriidae) exhibits pronounced sexual dimorphism in mean body size and shape. On average, males are larger than females and have more elongated heads, antennae, and legs (Bonduriansky 2006). This sexual dimorphism appears to have evolved in response to sexual selection on males resulting from male-male and male-female interactions. As in other neriid species (de Meijere 1911; Mangan 1979; Eberhard 1998; Preston-Mafham 2001), *T. angusticollis* males form aggregations on rotting bark, where they fight other males for access to females. *Telostylinus angusticollis* males challenge rivals by raising the anterior section of the body to a steep angle with the substrate and, in escalated encounters, use their head, antennae, and forelegs to strike their opponent. There is little evidence of courtship, but males guard females by enclosing them within the span of their legs during copulation and oviposition (Bonduriansky 2006; R. Bonduriansky, unpublished data). I investigated the relation between condition dependence and sexual dimorphism in *T. angusticollis* by manipulating larval diet quality (condition) and examining its effects on body size and seven shape traits, represented by linear dimensions of body parts involved directly in male-male sexual competition (sexual traits: head length and width, antenna length, and foretibia length), body parts with no direct role in male-male sexual competition (nonsexual traits: midtibia length, wing-vein length, and the distance between a pair of thoracic bristles), and thorax length (body size). Because sexual selection on body shape has not yet been quantified in this species, sexual and nonsexual traits are differentiated on the basis of their observed roles in male-male sexual competition (R. Bonduriansky, unpublished data).

Material and Methods

Flies and Culturing Techniques

The analysis is based on outbred, laboratory-reared F3 descendants of male and female *Telostylinus angusticollis* (Enderlein) collected from beetle-damaged *Acacia longifolia* trunks at the Fred Hollows Reserve in Sydney, Australia, in March 2004. Unrelated F2 males and females were paired inside 250-mL plastic containers with the bottom lined about 2 cm deep with moistened shavings from coconut husks (cocopeat; Galuku, Sydney) and containing 1-cm-diameter dishes of molasses and soy protein as adult food and a 3.5-cm petri dish with oviposition medium. Petri dishes were checked daily for eggs. From each of 14 male-female pairs, 20 eggs were transferred to each of two larval diet treatments. Each fly was reared separately: eggs

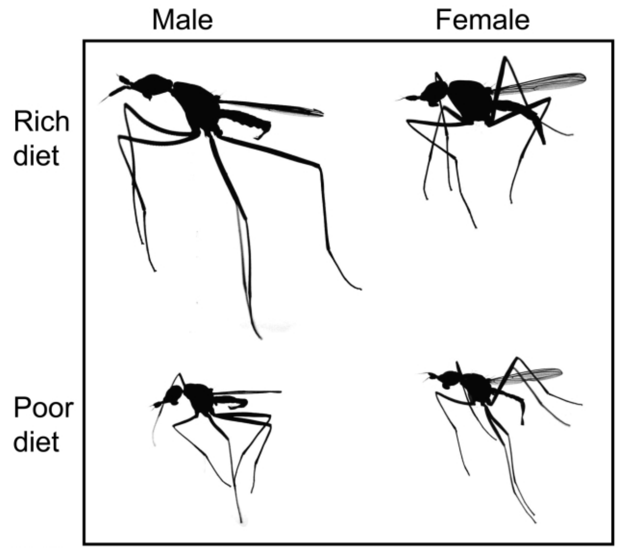

Figure 1: Male (*left*) and female (*right*) *Telostylinus angusticollis* siblings reared on rich (*top*) and poor (*bottom*) diets, shown as silhouettes viewed from the left. Specimens shown are representative individuals from a randomly selected family.

were placed individually into 20-mL glass vials containing 5 mL of food and were closed with mesh caps to permit air circulation. Vials were watered periodically to keep the food moist. Adults were allowed to emerge inside the vials, left to sclerotize for at least 24 h, and then frozen at −20°C.

Larval diet treatments differed in the concentration of sugars and protein in the hydrated cocopeat base. Rich food consisted of 30 mL blackstrap sugarcane molasses (Conga Foods, Preston, Australia), 30 mL liquid barley malt (Colonial Farms, Select Foods, Smithfield, Australia), and 32 g soy protein powder (Nature's Way, Pharm-a-Care, Warriewood, Australia) per liter of dry cocopeat hydrated with 800 mL of reverse-osmosis-treated water. The rich food also served as the oviposition medium. Poor food consisted of 10 mL of molasses, 10 mL of malt, and 10 g of soy protein per liter dry cocopeat and 800 mL water. Food mixtures were homogenized thoroughly using a handheld blender and were frozen at −20°C in 500-mL plastic containers until the day of use.

Morphometric Data

From each family, five offspring of each sex (where possible) were thawed and glued to entomological pins by the right mesopleuron. For each of these flies, eight linear measurements were made (illustrated in Bonduriansky 2006): thorax length (TL), head length (HL), head width at the widest point across the eyes (HW), antenna length (AL), foretibia length (FL), midtibia length (ML), wing-vein length (WL; R_{4+5} vein length from the r-m cross-vein to the wing margin), and intersetal width (IS; distance between the bases of the posterior dorsocentral setae). Measurements of TL, HL, HW, AL, and IS were made on a Leica MS 5 stereoscope with an ocular micrometer. Wings and legs were severed at the base and mounted on stickers affixed to glass slides and were scanned at 1,200 dpi on an Hewlett-Packard Scanjet 4890. Measurements of FL, ML, and WL were made from the scans, using image analysis software (ImageJ 1.34s, National Institutes of Health). Measurement repeatability was >0.99 for all traits in both sexes (Bonduriansky 2006).

Analysis

All analyses were performed using both raw and log-transformed data, but only the former are shown (see "Results"). For each trait, the degree of condition dependence was calculated in two ways: (1) as the percent difference

Table 1: ANOVA for effects of family (F), sex (S), and diet (D) on body size (thorax length) in *Telostylinus angusticollis* adults

Factor	Effect	SS[a]	df[b]	MS[c]	Error df	Error MS	*F*	*P*
F	Random	40,356	13	3,104	11.22	1,156.33	2.68	.0532
S	Fixed	3,278	1	3,278	13.36	314.68	10.42	**.0064**
D	Fixed	20,082	1	200,821	13.10	1,147.82	174.96	**<.0001**
F × S	Random	4,096	13	315	13.00	319.83	.99	.51
F × D	Random	15,093	13	1,161	13.00	319.83	3.63	**.0136**
S × D	Fixed	6,141	1	6,141	13.36	319.31	19.24	**.0007**
F × S × D	Random	4,157	13	319	206.00	285.00	1.12	.34

Note: Family was considered a random effect, whereas sex and diet were considered fixed effects, and error degrees of freedom and mean squares were obtained using the denominator synthesis method. Probabilities <.05 are highlighted in bold.

[a] Error = 58,709.

[b] Error = 206.

[c] Error = 285.

Figure 2: Condition and sexual size dimorphism: mean thorax lengths (mm) of females (*squares*) and males (*circles*) reared on rich and poor diets (bars show 95% confidence limits).

between mean trait sizes of same-sex siblings reared on different larval diets and (2) as the percent difference between reduced major axis (RMA) allometric slopes of same-sex siblings reared on different larval diets. Likewise, the degree of sexual dimorphism was calculated as the percent difference between mean trait sizes or allometric slopes of males and females. Thorax length was used as an index of body size in the calculation of allometric slopes because it loads most strongly on the first principal component (see "Results"; also see Bonduriansky and Rowe 2005*b*; Bonduriansky 2006).

I tested for diet, sex, and family effects and their interactions on body size (TL) using a factorial ANOVA. To test for diet and sex effects on allometric slope and intercept, ANCOVAs were performed for each trait, with TL as covariate (see "Results"). When all traits are measured with similar error, allometries are properly examined using RMA regression rather than least squares regression. Nonetheless, I tested for condition effects on allometric slope and intercept using least squares–based ANOVA tools because these tools afford maximum power and because the difference between RMA and least squares regressions is negligible when there is little scatter about the regression line ($r \approx 1$), as is the case for these traits in *T. angusticollis* (see Bonduriansky 2006). Statistical analysis was performed using Statistica 7.0 (StatSoft, Tulsa, OK).

Results

Development time from egg to adult was 6% shorter in larvae reared on rich food than in their siblings reared on poor food (rich food: mean = 33 ± 3.4 days; poor food: mean = 35 ± 3.5 days; *t*-test for dependent samples: $N = 14$ broods, $t = 5.61$, $P < .0001$). Mean larval survival rate was nearly equal in the two diet treatments (rich food: mean = 76% ± 15%; poor food: mean = 74% ± 16%; *t*-test for dependent samples: $N = 14$ broods, $t = 0.51$, $P > .6$). Thus, the treatment effects reported reflect developmental reaction norms rather than sampling effects of differential selection under rich and poor larval diets.

Diet quality had significant effects on adult body size and shape (fig. 1). Flies reared on rich food were larger than their siblings reared on poor food, and this effect was more pronounced in males than in females, as indicated by a significant sex × diet interaction in the ANOVA (table 1). Interestingly, diet quality reversed the sign of sexual size dimorphism from (nonsignificantly) female biased on poor food to (significantly) male biased on rich food (fig. 2). I also observed a significant family × diet interaction (table 1), suggesting that the magnitude of the diet effect on body size varied across genotypes.

Principal component (PC) analysis of the correlation matrix revealed a major axis of body shape variation (PC2) contrasting elongated head (HL), antenna (AL), and legs (FL, ML) at one extreme (positive loadings) with wide

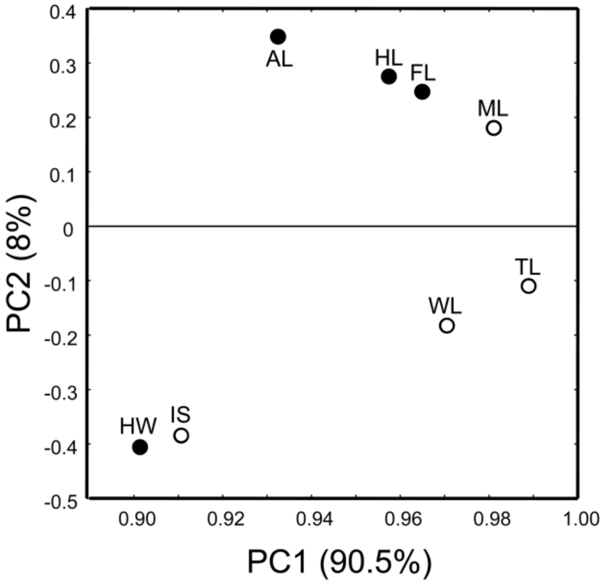

Figure 3: Ordination plot for sexual (*filled circles*) and nonsexual (*open circles*) traits in *Telostylinus angusticollis*, with both sexes and diet treatments pooled (see "Material and Methods").

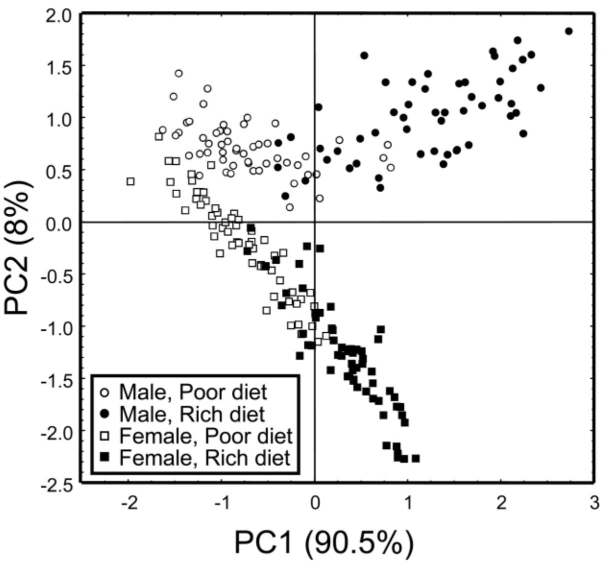

Figure 4: Condition effects on body shape: body shape (PC2 factor score) of males (*circles*) and females (*squares*) diverges with increasing body size (PC1 factor score), with flies reared on rich larval diets (*filled symbols*) exhibiting greater sex differences and a greater rate of divergence with increasing body size than flies reared on poor larval diets (*open symbols*).

head (HW), wide space between the posterior dorsocentral bristles (IS), and long wings (WL) at the other extreme (negative loadings; fig. 3). This pattern is consistent with body shape variation observed in wild-collected *Telostylinus angusticollis* specimens (Bonduriansky 2006). A plot of individual factor scores on PC1 and PC2 revealed increasing divergence in body shape between the sexes with increasing body size and condition (fig. 4): the smallest individuals reared on poor food exhibited little or no sexual shape dimorphism, whereas large individuals reared on rich food exhibited considerable dimorphism.

I used ANCOVA with TL as covariate to test for diet effects on each of seven components of body shape (i.e., trait sizes relative to body size). First, a separate ANCOVA was performed for each of the seven traits to test for effects of diet, sex, and family and their interactions on allometric slope, indicated by a significant interaction with TL (table 2). Of the seven traits, only HL and AL exhibited sex differences in the effect of diet on allometric slope (sex × diet × TL interaction). The traits FL, ML, and HL exhibited only sex effects on allometric slope (sex × TL interaction), whereas the allometries of WL and IS were not affected significantly by either sex or diet. Log transformation of variables produced qualitatively similar results (not shown), except that two interactions became nonsignificant: sex × TL for HW and sex × diet × TL for AL.

I then performed a separate ANCOVA for each treatment combination with a distinct slope to test for diet effects on the allometric intercept (i.e., mean relative trait size). Because HL and AL exhibited significant sex and diet effects on allometric slope, I performed separate ANCOVAs for each sex × diet combination to test for family effects (table 3). Family effects were significant in each case, except for HL in poor diet males. Because HW, FL, and ML exhibited significant sex effects on allometric slope, I performed separate ANCOVAs for each sex to test for family and diet effects (table 4). For HW, I found a significant family × diet interaction in females only. For FL, I found significant family effects in both sexes, as well as a significant diet effect in females. For ML, I found a significant family effect in males and a significant family × diet interaction in females. The traits WL and IS did not exhibit significant heterogeneity in allometric slope, so I performed a single ANCOVA for family, sex, and diet effects (table 5). For WL, I found significant family and sex effects and a sex × diet interaction. For IS, I found a significant sex effect and a significant sex × diet interaction.

I examined the relation between sexual dimorphism and condition dependence in two ways (fig. 5). Based on differences in mean trait size, the degree of sexual dimorphism was not correlated with the strength of condition dependence among female traits (Pearson correlation: $N = 8$, $r^2 = 0.03$; $r = 0.18$, $P = .66$; $y = 0.18 + 0.02x$), whereas male traits exhibited a strong positive correlation (Pearson correlation: $N = 8$, $r^2 = 0.94$; $r = 0.97$, $P < .0001$; $y = -0.46 + 0.71x$). The correlation for male traits was significantly stronger than the correlation for female traits ($N = 8$, $Z = 3.02$, $P = .0013$). Similarly, based on differences in allometric slope, there was no significant correlation for females (Pearson correlation: $N = 7$, $r^2 = 0.21$; $r = -0.46$, $P = .30$; $y = 4.88 - 3.51x$) but a strong positive correlation for males (Pearson correlation: $N = 7$, $r^2 = 0.90$; $r = 0.95$, $P = .0011$; $y = -1.37 + 2.40x$) and a significant difference between male and female correlation coefficients ($N = 7$, $Z = 3.29$, $P = .0005$). Log-transformed data yielded qualitatively and quantitatively similar results (not shown). Thus, sexual dimorphism and condition dependence are strongly positively correlated among male (but not female) traits.

Discussion

An analysis of variation among morphological traits in *Telostylinus angusticollis* revealed a striking congruence between sexual dimorphism and condition dependence in males but not in females. This finding supports the hypothesis that sexual selection drives the coevolution of sexual dimorphism and condition dependence. The

Table 2: ANCOVA results for effects of family (F), sex (S), diet (D), and their interactions on allometric slope for each of seven traits, with thorax length (TL) as a covariate

		Trait						
Factor	df[a]	HL	HW	AL	FL	ML	WL	IS
TL	1	2,880.61	254.76	436.38	1,564.19	3,484.79	661.89	436.79
		(**<.0001**)	(**<.0001**)	(**<.0001**)	(**<.0001**)	(**<.0001**)	(**<.0001**)	(**<.0001**)
F × TL	13	.24	.61	.69	1.01	.50	2.74	.84
		(.99)	(.78)	(.73)	(.52)	(.85)	(.53)	(.62)
S × TL	1	64.86	5.19	37.58	29.56	43.93	.00	1.31
		(**<.0001**)	(**.0269**)	(**<.0001**)	(**<.0001**)	(**<.0001**)	(1.00)	(.27)
D × TL	1	30.30	.45	22.09	2.09	3.45	.00	.00
		(**<.0001**)	(.50)	(**<.0001**)	(.16)	(.0789)	(1.00)	(1.00)
F × S × TL	13	2.22	1.10	1.25	1.75	.64	.61	1.70
		(.0816)	(.43)	(.35)	(.16)	(.78)	(.81)	(.18)
F × D × TL	13	1.26	1.17	1.28	.83	1.45	.70	1.17
		(.34)	(.39)	(.33)	(.63)	(.26)	(.73)	(.39)
S × D × TL	1	36.98	.06	4.49	1.96	1.01	.00	.00
		(**<.0001**)	(.81)	(**.0426**)	(.17)	(.33)	(1.00)	(1.00)
F × S × D × TL	13	.65	.82	1.27	.74	.84	1.53	.65
		(.81)	(.64)	(.24)	(.72)	(.61)	(.11)	(.81)

Note: *F* ratios are shown, with corresponding *P* values below in parentheses. Family was considered a random effect, whereas sex and diet were fixed effects, and separate slopes were fitted to each treatment combination. Probabilities <.05 are highlighted in bold.

[a] Error df = 128–149.

strength of this covariation suggests that within-sex and between-sex variation in body shape is largely controlled by the same genetic mechanisms. Although the largest condition effects were observed in male sexual traits, condition had remarkably complex effects on body shape in both sexes, affecting the allometric slopes and/or intercepts of all sexual and nonsexual traits.

Coevolution of Condition Dependence and Sexual Dimorphism

A trait that is more directly targeted by directional sexual selection in males should (all else being equal) attain a greater displacement from the viability-selected phenotype expressed by females, resulting in more pronounced sexual dimorphism. The degree of displacement from the viability optimum will, on average, reflect the magnitude of viability costs of trait expression incurred by males and, consequently, the degree of condition dependence favored by selection (Rowe and Houle 1996). Thus, theory predicts that sexual dimorphism and condition dependence will coevolve, and an analysis of multiple traits subject to varying intensities of sexual selection should reveal a strong positive covariation between the degree of sexual dimorphism and the strength of condition dependence. This study provides strong support for this prediction, showing that the expression of sexual size and shape dimorphism is almost entirely conditional in *T. angusticollis*: small low-condition males and females are nearly indistinguishable in multivariate body shape, whereas large high-condition individuals exhibit unmistakably sex-specific phenotypes. The strength of the covariation between condition dependence and sexual dimorphism in this species (fig. 5) suggests that these two complex traits share a common genetic and developmental basis. Sexual dimorphism may reflect a pleiotropic effect of conditionally expressed sex-linked genes that determine the degree of allocation to a trait in males relative to females (fig. 6). Variation in the degree of plasticity exhibited by these sex-linked allocation genes will thus determine the strength of the genetic correlation between sexual dimorphism and condition dependence.

The pattern of sexual dimorphism observed in *T. angusticollis* may be called incomplete because low-condition flies approach sexual monomorphism in body size and shape (fig. 4). In contrast, some species exhibit a complete sexual dimorphism, where a sex-specific phenotype is expressed in all individuals. For example, in *Prochyliza xanthostoma*, sexual shape dimorphism is maintained even at the lowest level of condition observed in viable individuals (Bonduriansky and Rowe 2005*b*; Bonduriansky 2006). This may reflect a difference in the shape of the sex-dependent allocation function (fig. 7). Nonetheless, *P. xanthostoma* males with severe developmental abnormalities tend to exhibit unusually femalelike secondary sexual traits (R. Bonduriansky, unpublished data), suggesting that sexual shape dimorphism may be incomplete in *P. xanthostoma* as well, if subviable adults are included in the sample. Incomplete sexual dimorphism also occurs in *Onthophagus* dung beetles, where low-condition males are hornless, as are females (Emlen 1994, 1997; Emlen and Nijhout 1999;

Table 3: ANCOVA results for effects of family (F) on head length (HL) and antenna length (AL), with thorax length (TL) as covariate, performed separately within each sex × diet combination for each trait

		Males				Females			
		Rich diet		Poor diet		Rich diet		Poor diet	
	df[a]	*F*	*P*	*F*	*P*	*F*	*P*	*F*	*P*
HL:									
TL	1	1,114.41	**<.0001**	1,395.33	**<.0001**	459.88	**<.0001**	981.65	**<.0001**
F	14	19.23	**<.0001**	1.46	.16	6.50	**<.0001**	10.58	**<.0001**
AL:									
TL	1	533.80	**<.0001**	491.55	**<.0001**	186.69	**<.0001**	357.87	**<.0001**
F	14	12.19	**<.0001**	3.50	**.0005**	2.22	**.0201**	2.40	**.0131**

Note: Probabilities <.05 are highlighted in bold.

[a] Error df = 46–52.

Moczek and Emlen 1999), whereas complete sexual dimorphism appears to occur in the diopsid (stalk-eyed) fly *Cyrtodiopsis dalmanni* (Cotton et al. 2004*a*). Moreover, in general, secondary sexual traits tend to exhibit condition-dependent sexual dimorphism (either complete or incomplete), whereas primary sexual traits exhibit complete and largely condition-independent sexual dimorphism.

Several previous studies have reported phenotypic plasticity of sexual dimorphism in relation to environmental parameters such as food abundance and temperature (David et al. 1994; Post et al. 1999; Karan et al. 2000; Weladji et al. 2005). This study shows that environmental effects on sexual dimorphism can vary in nature and magnitude among both sexual and nonsexual traits within a species. Strong covariation between the magnitude of phenotypic plasticity (i.e., condition dependence) and the degree of sexual dimorphism among male traits in *T. angusticollis* shows that sexual dimorphism and condition dependence are biologically and conceptually inseparable because dimorphism results almost entirely from condition dependence. This highlights the need to understand the role of condition dependence in the genetic architecture of sexually dimorphic traits. Considerable thought has been given to the genetic architecture of sexual dimorphism, including the role of X-linked segregation (Rice 1984), sex-linked modifiers (e.g., Rice and Chippindale 2002), sex-limited gene expression (Rhen 2000), and parent-of-origin effects (Day and Bonduriansky 2004; Bonduriansky and Rowe 2005*a*), but none of these models can account for the condition dependence of sexual dimorphism (see Bonduriansky 2007). Conversely, theory on the genetic architecture of condition dependence (Rowe and Houle 1996; Tomkins et al. 2004) fails to address its sex-specific nature (Bonduriansky and Rowe 2005*b*). It is necessary to integrate these disparate bodies of theory. Genetic models of sexual dimorphism should account not only for sex-dependent trait expression but also for the complex epistasis thought to engender the heightened condition dependence of secondary sexual traits (see Rowe and Houle 1996). Recent advances in our understanding of the genetic and hormonal control of secondary sexual trait expression in insects (Stern and Emlen 1999; Emlen et al. 2006) may ultimately shed light on the developmental mechanisms responsible for the complex sex- and condition-dependent reaction norms often exhibited by such traits.

In light of these findings, it is interesting to consider how condition dependence might affect the rate of evolution of sexual dimorphism. Rowe and Houle (1996) hypothesized that condition dependence results from genic capture, a form of male-limited epistasis linking trait ex-

Table 4: ANCOVA results for effects of family (F) and diet (D) on head width (HW), foretibia length (FL), and midtibia length (ML), with thorax length (TL) as covariate, performed separately within each sex and trait

		Males		Females	
	df[a]	*F*	*P*	*F*	*P*
HW:					
TL	1	274.83	**<.0001**	558.69	**<.0001**
F	13	2.12	.0921	1.42	.27
D	1	3.57	.0620	1.24	.27
F × D	13	1.26	.25	2.40	**.0077**
FL:					
TL	1	2,384.70	**<.0001**	1,517.36	**<.0001**
F	13	5.64	**.0017**	5.31	**.0022**
D	1	.04	.84	4.92	**.0290**
F × D	13	.95	.51	1.06	.40
ML:					
TL	1	2,672.04	**<.0001**	1,802.31	**<.0001**
F	13	6.41	**.0009**	2.39	.0638
D	1	.06	.80	.00	1.00
F × D	13	1.19	.30	2.59	**.0040**

Note: Probabilities <.05 are highlighted in bold.

[a] Error df = 87–103.

Table 5: ANCOVA results for effects of family (F), sex (S), and diet (D) on wing-vein length (WL) and intersetal width (IS), with thorax length (TL) as covariate, performed separately within each trait

		WL		IS	
Factor	df[a]	*F*	*P*	*F*	*P*
TL	1	2,503.06	**<.0001**	991.60	**<.0001**
F	13	5.92	**.0059**	1.80	.15
S	1	12.59	**.0033**	97.59	**<.0001**
D	1	1.02	.32	3.95	.0503
F × S	13	1.50	.24	2.35	.0684
F × D	13	1.35	.30	1.69	.17
S × D	1	8.65	**.0098**	43.92	**<.0001**
F × S × D	13	1.38	.17	1.18	.29

Note: Probabilities <.05 are highlighted in bold.

[a] Error df = 192–202.

pression with variation at numerous loci affecting the efficiency of resource acquisition and allocation. If genic capture can evolve in males without correlated effects on females, then the evolution of heightened condition dependence in male sexual traits may reduce intersexual genetic correlations for those traits (Bonduriansky 2007). In other words, captured resource acquisition genes will reduce the overlap between the sets of genetic factors modulating trait expression in males and females, permitting the divergence of male and female phenotypes. Thus, condition dependence may be expected to facilitate the evolution of sexual dimorphism in secondary sexual traits (but see Bonduriansky and Rowe 2005*b*).

Sources of Variation among Traits

The largest condition effects on mean trait size and allometric slope were observed in two male sexual traits (HL and AL). However, other sexual traits (HW and FL) did not exhibit sex differences in condition effects on allometric slope, and, curiously, only females exhibited significant condition effects on allometric intercept for these traits (table 4). Condition effects on allometric intercept were also observed in the nonsexual traits (tables 4, 5). Thus, male weapon and signal traits, as well as nonsexual traits in both sexes, vary considerably in the nature and strength of condition dependence in *T. angusticollis*. Similar conclusions can be drawn from studies on the piophilid fly *P. xanthostoma* (Bonduriansky and Rowe 2005*b*), stalk-eyed flies (David et al. 1998, 2000; Cotton et al. 2004*a*, 2004*c*), and peacocks (Møller and Petrie 2002). These findings are relevant to the hypothesis that sexual selection drives the evolution of heightened condition dependence. Although such studies have generally found that sexual traits exhibit stronger condition dependence than nonsexual traits, the intertrait variation suggests that the role of sexual selection in the evolution of condition dependence is complex and that other factors probably play important roles.

As with any trait, variation in the strength of condition dependence (and degree of sexual dimorphism) is likely to reflect an interplay between selection and genes. Different sexual traits are probably subject to differing regimes of sexual and viability selection. Many signal and weapon

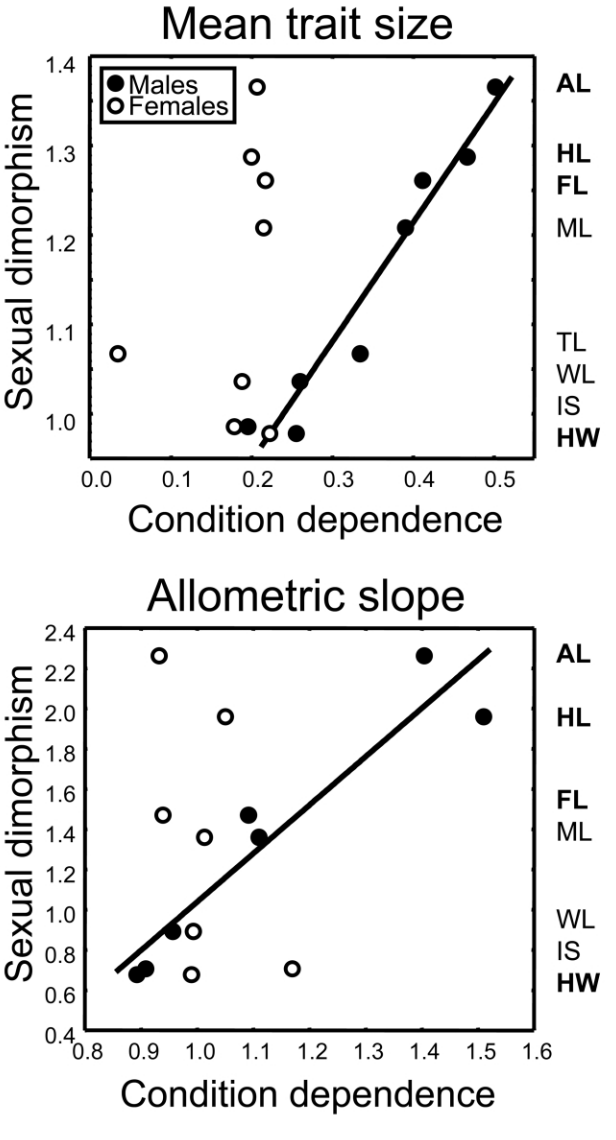

Figure 5: Covariation of degree of sexual dimorphism (mean difference between sexes) and strength of condition dependence (mean difference between diet treatments within sexes) for eight traits, quantified in terms of mean trait size (*top*) and allometric slope (*bottom*). Filled circles show male data (with fitted reduced major axis), and open circles show female data. The traits are identified at the right of each panel, with sexual traits shown in bold (see “Material and Methods”).

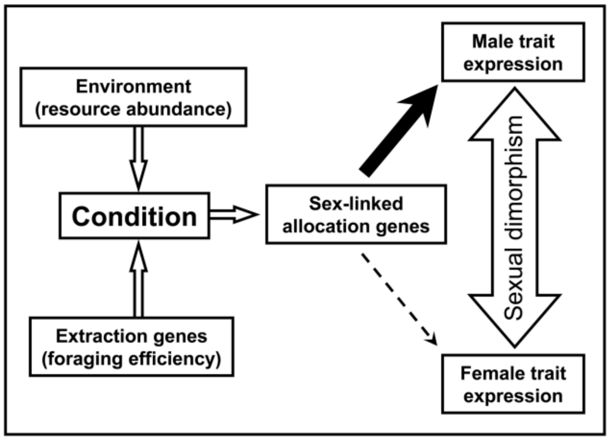

Figure 6: Model of condition-dependent sexual dimorphism: condition (determined both by resource abundance in the environment and by genes affecting the efficiency with which those resources are extracted and converted to metabolic resources) affects the relative rates of resource allocation to a trait in males and females, based on a sex-dependent resource allocation function determined by sex-linked genes. The sex-dependent resource allocation function determines how relative allocation to each sex changes with increasing condition (see fig. 7). Sexual dimorphism is thus a pleiotropic effect of sex-linked genes with condition-dependent expression.

traits appear to be subject to directional sexual selection, whereby fitness is maximized by expressing such traits at the greatest size that viability costs permit (e.g., see Green 1992). The need to optimize this trade-off between mating success benefits and viability costs generates selection for strong condition dependence. But bigger is not always better. Because performance in sexual competition may reflect an interaction between the size of the trait and the ability to use it in combat or courtship, a weapon or signal trait that is too large to wield with agility may reduce mating success. Moreover, trait size may have conflicting effects on performance in combat and courtship (Moore and Moore 1999; Bonduriansky and Rowe 2003). Thus, some sexual traits may be subject to stabilizing sexual selection, favoring relatively weak condition dependence. Condition dependence strength is also expected to reflect the viability costs of secondary sexual trait expression, which may vary among traits.

The evolution of condition dependence may also be constrained by genetic covariances with other traits, mediated by ontogenetic resource allocation trade-offs (Nijhout and Emlen 1998; Emlen 2001; but see Preziosi and Roff 1998; Tomkins et al. 2005) or pleiotropy (Conner 2002). Selection for heightened condition dependence of sexual traits may result in correlated evolution of condition dependence in some nonsexual traits. This would counteract selection for developmental stability and displace those nonsexual traits from their viability optima (de Visser et al. 2003). The resulting viability costs may impede the evolution of condition dependence in sexual traits. Positive genetic covariance may account for the similarity of responses to condition of foreleg and midleg traits (tables 2, 4; fig. 5), despite the foreleg's more direct role in sexual competition (Bonduriansky 2006). For similar reasons, the evolution of condition dependence in male traits may also be constrained by an intersexual genetic correlation for condition dependence, resulting in correlated evolution of condition dependence in homologous female traits (Bonduriansky and Rowe 2005*b*). The intra- and intersexual genetic covariance structure may thus contribute to variation in condition dependence among sexual and nonsexual traits.

Heightened condition dependence also appears to evolve only along some phenotypic dimensions of signal and weapon traits, probably reflecting the direction of the strongest vector of sexual selection. In *T. angusticollis*, sexual selection appears to have driven the elongation and heightened condition dependence of the male head and antennae. In contrast, a perpendicular dimension—head width—exhibits much weaker condition dependence (fig. 5). Thus, high-condition males appear to use their extra resources primarily to elongate rather than widen the head. This allocation effect may be amplified by a negative gensetic correlation between HL and HW (Bonduriansky and

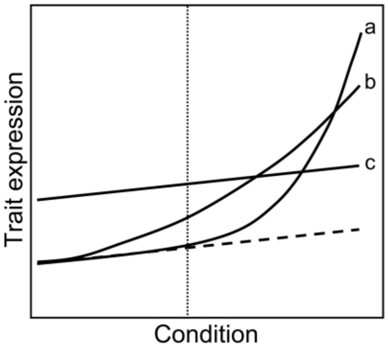

Figure 7: Hypothetical sex-dependent resource allocation functions: the dashed line shows the increase in trait expression with condition in females, and the solid lines show different patterns of trait expression in relation to condition for males. The vertical dotted line indicates the minimum condition that can support viable adults. The pattern observed in *Telostylinus angusticollis* males is represented by curve *a*, where male trait expression begins to diverge from female trait expression as condition increases above the minimum (incomplete sexual dimorphism). The pattern observed in *Prochyliza xanthostoma* is represented by curve *b*, where male trait expression differs from female trait expression throughout the observed range of condition (complete sexual dimorphism) but may converge with female trait expression in subviable individuals. Curve *c* represents a hypothetical condition-independent sexual dimorphism.

Rowe 2005*b*) and by trait geometry: given the elongated and tapered shape of the head, a unit of resources allocated to length will have a greater effect than a unit of resources allocated to width.

Further empirical and theoretical research is required to understand intertrait variation in condition dependence. Sexual selection functions have been estimated for very few species and traits, and even fewer examples of sex-specific patterns of selection are available (but see Preziosi and Fairbairn 2000; Chenoweth and Blows 2003). The viability costs of sexual traits also remain poorly known (Kotiaho 2001). Likewise, almost nothing is known about genetic constraints on the evolution of condition dependence. Alongside empirical evidence, new theory is required to understand how selection functions and genetic architectures shape the evolution of condition dependence.

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