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Sexually antagonistic genetic variance for fitness in an ancestral and a novel environment

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The intersex genetic correlation for fitness (r_w^{fm}), a standardized measure of the degree to which male and female fitness covary genetically, has consequences for important evolutionary processes, but few estimates are available and none have explored how it changes with environment. Using a half-sibling breeding design, we estimated the genetic (co)variance matrix (**G**) for male and female fitness, and the resulting r_w^{fm}, in *Drosophila serrata*. Our estimates were performed in two environments: the laboratory yeast food to which the population was well adapted and a novel corn food. The major axis of genetic variation for fitness in the two environments, accounting for 51.3 per cent of the total genetic variation, was significant and revealed a strong signal of sexual antagonism, loading negatively in both environments on males but positively on females. Consequently, estimates of r_w^{fm} were negative in both environments (-0.34 and -0.73, respectively), indicating that the majority of genetic variance segregating in this population has contrasting effects on male and female fitness. The possible strengthening of the negative r_w^{fm} in this novel environment may be a consequence of no history of selection for amelioration of sexual conflict. Additional studies from a diverse range of novel environments will be needed to determine the generality of this finding.

Keywords: adaptation; *Drosophila serrata*; intersex genetic correlation; fitness; sexual conflict

1. INTRODUCTION

Sexually antagonistic genetic variation for fitness exists when alleles segregating within a population tend to have opposite effects on the fitness of the two sexes. Such variation arises when different values of a shared trait maximize male and female fitness, generating intralocus sexual conflict in which the adaptive evolution of one sex is impeded by the other (Lande 1980; Rice 1984; Parker & Partridge 1998; Rice & Chippindale 2001; Bonduriansky & Chenoweth in press). The extent of this evolutionary constraint is indicated by the intersex genetic correlation for fitness (r_w^{fm}), a standardized measure of the genetic covariance of male and female fitness. In the absence of sexual conflict, r_w^{fm} is expected to be large and positive (Lynch & Walsh 1998). Sexually antagonistic fitness variation will cause a negative covariance between male and female fitness, thereby lowering r_w^{fm}. Negative correlations can severely constrain adaptive evolution, even in the presence of substantial genetic variation for lifetime fitness (Lande 1982) and, if common, could explain why genetic variation in fitness appears to be higher than expected under mutation–selection balance (Turelli & Barton 2004). Negative correlations may also affect the evolution of mate choice, impeding the evolution of female preferences for males of high breeding value for fitness (i.e. 'good genes' theories of sexual selection; Chippindale *et al.* 2001; Pischedda & Chippindale 2006; Kirkpatrick in press).

A number of studies have demonstrated the existence of sexually antagonistic genetic variation for specific traits or components of fitness (Meagher 1992; Rice 1992; Gibson *et al.* 2002; Calsbeek & Sinervo 2004; Fedorka & Mousseau 2004; Pischedda & Chippindale 2006; Prasad *et al.* 2007). Fewer studies have reported estimates of the intersex genetic correlation for lifetime fitness: -0.85 in collared flycatchers (Qvarnström *et al.* 2006); -0.48 in red deer (Foerster *et al.* 2007); and -0.16 in a laboratory population of *Drosophila melanogaster* (Chippindale *et al.* 2001). However, general conclusions are tentative because direct estimates of r_w^{fm} are few, standard errors on them are large and measures of fitness vary (Kirkpatrick in press). Nevertheless, the available data suggest that sexually antagonistic fitness variation may be more common than previously thought.

An important aspect of the nature of sexually antagonistic genetic variation that has yet to be investigated is the extent to which r_w^{fm} may vary among environments. Genotype-by-environment interactions are common for many traits (Lynch & Walsh 1998), including measures of performance (Hunt *et al.* 2004), and it is therefore likely that the genetic basis of fitness will vary with environment. Theory suggests that there is little reason to expect genetic variances and covariances to remain constant when environments change (Via & Lande 1987), and, consistent with this, genetic correlations for numerous traits, including morphology and life history, have been shown to vary across environments (Sgrò & Hoffmann 2004). How r_w^{fm} in particular may change in novel environments is difficult to predict, and alternative scenarios are possible depending on

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changes in sex-specific selection and the nature of any genotype-by-environment interactions for the traits determining fitness.

One possible scenario for changes in r_w^{fm} builds on the idea that sustained sexually antagonistic selection in an environment is ultimately expected to favour mutations that allow males and females to achieve their sex-specific optima, thereby resolving sexual conflict (Lande 1980, 1987). Over the long term, any (partial) resolution of sexual conflict via the evolution of sexual dimorphism will cause r_w^{fm} to become less negative (reviewed in Bonduriansky & Chenoweth in press). The time scale over which this occurs may be exceedingly slow, however, if the intersex genetic correlations for the phenotypic traits experiencing sexually antagonistic selection are high (Lande 1987). In a novel environment with unique sex-specific optima for the traits determining fitness and no history of selection to accommodate this, r_w^{fm} may therefore be expected to be strongly negative. This is of particular relevance to the recent observation that sexual selection may often fail to promote adaptation to novel environments in evolution experiments (Holland 2002; Rundle *et al.* 2006). One possible explanation for why sexual selection fails in this regard is that a strong negative r_w^{fm} in the novel environments hampers a good genes process, impeding or even preventing sexual selection from contributing to adaptation.

Here, we estimate genetic variances for male and female fitness, and the resulting r_w^{fm}, using a laboratory population of the Australian fruitfly, *Drosophila serrata*. We perform these measures in each of two environments, one to which the population is long adapted ('ancestral' yeast food environment) and a novel environment (corn food) to which the population is not well adapted. The addition of the novel environment permits insight into how the genetic covariance of male and female fitness varies across environments in response to any changes in the genetic basis of fitness, and is of direct relevance to the question of whether sexual selection promotes adaptation to novel environments as predicted by good genes models (Lorch *et al.* 2003). The inclusion of a novel environment also allows us to directly address the possibility that the importance of sexually antagonistic fitness variation is exaggerated in laboratory populations due to long-term adaptation to unusually consistent environments (Chapman *et al.* 2003).

We estimated genetic variances and covariances for male and female fitness in both environments within the context of a large quantitative genetic paternal half-sibling breeding design that involved 81 sires mated to 326 dams. Fitness assays were conducted in the ancestral and novel environments on a total of 2173 female and 2005 male offspring from the breeding design. *Drosophila serrata* is an ideal candidate for such experiments because fitness measures can integrate the major components relevant for the population in the environment to which it is adapted, including mating success of males within a competitive context. In addition, good genes mate choice has been well studied in this species (Hine *et al.* 2004), including a manipulative evolution experiment testing the contribution of sexual selection during adaptation of this same population to the same novel corn food environment (Rundle *et al.* 2006).

2. MATERIAL AND METHODS

(a) *Half-sibling breeding design*

A paternal half-sibling breeding design was conducted using a previously described laboratory population of *D. serrata* (Rundle *et al.* 2006; Chenoweth *et al.* 2008). Eighty-one sires were each mated to four virgin females (dams) and these females were subsequently allowed to oviposit for 20 hours in one environment (yeast or corn) and then 48 hours in the other. The order of oviposition environments was alternated among females. The breeding design was conducted in three blocks consisting of 22, 30 and 29 sires that spanned five generations of the laboratory population. In all cases, fitness assays were performed on 3-day-old virgin sons and daughters from each family collected at emergence and separated by sex using light CO_2 anaesthesia.

(b) *Male fitness assays*

A competitive measure of male fitness was conducted by placing together in a vial one virgin son from the breeding design along with two virgin competitor males of similar age randomly chosen from a stock population into which a recessive orange-eye mutation had been introgressed. These males competed to fertilize a randomly chosen virgin orange-eye female that was free to oviposit in the same vial. After 72 hours, all flies were discarded and vials were retained for offspring development. Vials contained food that matched the environment in which the son was raised (i.e. yeast or corn). Five replicate trials using five separate sons were conducted using the offspring of each dam in each environment. Male fitness was calculated as the ratio of wild-type : orange-eye individuals in the adult offspring produced by the female. This measure of male fitness included his mating success, the subsequent productivity of the female he mated and the survival to emergence of his male and female offspring in competition with half-sibling offspring sired by the orange-eye competitors. Sons that did not produce any offspring were excluded from the analysis. Prior to analyses, data were ln-transformed (after the addition of a constant to both mutant and wild-type offspring counts) and standardized to a mean of zero and unit variance.

(c) *Female fitness assays*

Female fitness was measured by placing together in a vial one virgin daughter from the breeding design along with one randomly chosen virgin male of similar age from the stock population. The pair was allowed to mate freely and the female could oviposit on the food. After 48 hours, both flies were discarded and the vial was retained for offspring development. Vials contained food that matched the environment in which the daughter was raised (i.e. yeast or corn). Five replicate trials using five separate daughters were conducted using the offspring of each dam in each environment. Female fitness was calculated as the total number of adult offspring emerging in each vial. Daughters that did not produce any offspring were not included in the analysis. Data were square-root transformed and standardized to a mean of zero and unit variance prior to analysis.

This measure of female fitness included her fecundity along with the survival to emergence of her male and female offspring. This differed from our measure of male fitness in that it lacked any contribution of mating success arising from mate choice by the opposite sex (i.e. male choice), and competition from half-sibling offspring was absent during larval development. Since male and female fitness are gained

through different traits, such a difference in measurement is difficult to avoid. For example, variance in female mating success due to male mate choice is probably a small component of female fitness because it is likely that a single male would have mated all three females within the time provided had we mirrored the design used to measure male fitness. The strength of larval competition overall was probably similar in both fitness assays as well because larval rearing densities did not differ drastically in the two assays (mean total number of adults emerging per vial $\pm$s.e. for females and males, respectively: yeast 62.9 ± 1.2 and 79.9 ± 1.3; corn 40.8 ± 0.7 and 45.2 ± 0.7).

(d) *Statistical analysis*

The additive genetic variance–covariance (i.e. **G**) matrix for male and female fitness in the two environments was constructed at the sire level by REML using the factor-analytic modelling approach implemented in the mixed procedure of SAS v. 9.1 (Hine & Blows 2006; McGuigan & Blows 2007). The model was

$$Y_{ijklm} = \mu + S_j + D_{k(j)} + B_l + O_m + B_l O_m + \varepsilon_{ijklm}, \qquad (2.1)$$

where Y_{ijklm} is the observed fitness of the ith offspring of the kth dam (D) nested within the jth sire (S) and ε is unexplained error. Fixed effects include the intercept (μ), experimental block (B), the order of oviposition environments (O) and their interaction.

The factor-analytic approach has several advantages including that, by directly estimating the principal components of **G**, the resulting covariance matrix is guaranteed to be positive semi-definite (all eigenvalues greater than or equal to zero; Kirkpatrick & Meyer 2004). This approach also provides a powerful method for directly testing the dimensionality of **G**, or, in other words, the number of genetically independent traits underlying male and female fitness in these two environments (Hine & Blows 2006). For this analysis, the covariance matrix was constrained to be from four to zero dimensions, and a series of nested likelihood ratio tests were used to determine whether excluding each dimension significantly worsened the fit of the model (Hine & Blows 2006). Significance of specific covariances (correlations) from an unconstrained covariance matrix were determined using likelihood ratio tests in which the parameters of interest were fixed at specific values (either 0 or 1) and the fit of the model was compared with its unconstrained counterpart (Shaw 1991; Fry 2004).

To test whether the genetic basis of fitness differed overall between the two environments, male and female fitness were each pooled across the environments and a single unconstrained 2×2 covariance matrix was constructed at the sire level using equation (2.1). A fixed effect of environment was included in this model because fitness of each sex was standardized to a mean of zero and a unit variance only in this analysis. The fit of this model was compared with one in which separate unconstrained 2×2 covariance matrices were constructed in each environment by employing the 'group' statement at the sire level in the SAS mixed procedure, with significance determined using a likelihood ratio test.

3. RESULTS

The genetic basis of male and female fitness varied significantly between the two environments ($\chi^2_3=117.5$, $p<10^{-5}$), so the additive genetic (co)variances matrix (**G**) for male and female fitness was estimated by treating the fitness of each sex in each environment as separate traits (table 1). Factor-analytic modelling revealed support for one significant genetic dimension underlying this suite of traits ($\chi^2_2=10.60$; $p=0.031$; table 2), which explained 51.3 per cent of the total genetic variance in fitness. This major axis of genetic variance loaded negatively in both environments in males and positively in females (table 1), revealing a strong signal of sexual antagonism for the majority of genetic variance in fitness across these two environments.

Table 1. Full-rank additive genetic (co)variance matrix (**G** matrix) for male and female fitness in the ancestral (yeast) and novel (corn) environments. (Genetic variances are given in italic along the diagonal and covariances below. The corresponding genetic correlations are given above the diagonal.)

	♀ yeast	♂ yeast	♀ corn	♂ corn	PC1[a]
♀ yeast	*0.1704*	−0.3429	0.5581	−0.0701	0.7902
♂ yeast	−0.0518	*0.1339*	0.0556	0.2734	−0.4493
♀ corn	0.0460	0.0041	*0.0399*	−0.7260	0.2892
♂ corn	−0.0090	0.0313	−0.0453	*0.0978*	−0.3001

[a]The first eigenvector of **G**, accounting for 51.3% of the genetic variance.

Table 2. Model fit statistics of the number of effective dimensions of the sire-level covariance matrix, **G**.

no. of dimensions	no. of parameters	−2 log likelihood	AIC[a]	p-value[b]
fa0(4)	10	11801.69472	11845.70	1.000
fa0(3)	9	11801.69471	11845.70	0.248
fa0(2)	7	11804.48596	11844.50	0.269
fa0(1)	4	11808.41856	11844.40	0.031
fa0(0)	0	11819.01405	11847.00	—

[a]Akaike's information criterion.
[b]Results of a likelihood ratio test of whether the fit of a model that excludes one factor (i.e. the model in the row below) is significantly worse than the fit of the current model.

Within each sex, the genetic correlation in fitness between the two environments was positive (males: $r=0.2734$; females: $r=0.5581$; table 1). Thus, sires producing high-fitness daughters (sons) in one environment tended also to produce high-fitness daughters (sons) in the other environment, indicating that the genetic basis of fitness in the two environments is at least partially shared within each sex. By contrast, intersex genetic correlations for fitness were negative in both environments (yeast: $r_w^{fm}=-0.34$; corn: $r_w^{fm}=-0.73$; table 1), indicating that, in the ancestral and the novel environments, sires producing high fitness sons tended to produce low fitness daughters, and *vice versa* (figure 1). In both environments, r_w^{fm} was significantly less than 1 (yeast: $\chi^2_1=5.87$, $p=0.015$; corn: $\chi^2_1=5.63$, $p=0.018$) and the associated intersex covariance was significantly less than zero in corn ($\chi^2_1=4.43$, $p=0.035$), although not in yeast ($\chi^2_1=1.22$, $p=0.270$).

The extent to which the negative r_w^{fm} would constrain the evolution of fitness in these environments can be illustrated by applying the multivariate breeders equation using the 2×2 **G** matrices for each environment (table 1) and a unit vector of linear selection gradients that seek

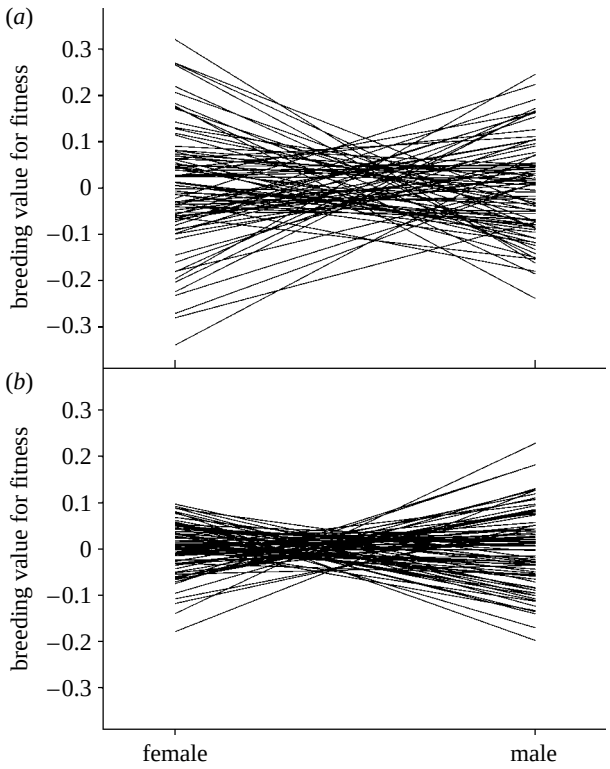

Figure 1. Interaction plot of the breeding values of female and male fitness in (*a*) yeast and (*b*) corn. Lines join breeding values for the same sire, estimated as best linear unbiased predictors from equation (2.1) using the factor-analytic approach with four dimensions for the sire-level covariance matrix.

to increase fitness in both sexes to the same extent (i.e. $\boldsymbol{\beta}^{\mathrm{T}} = [0.5,\ 0.5]$). In yeast, there remains a small amount of genetic variance available to increase fitness in both sexes, with fitness predicted to increase by 6 and 4 per cent of a phenotypic standard deviation in females and males, respectively. In corn, by contrast, female fitness is predicted to decrease by 0.2 per cent while male fitness would increase by 3 per cent.

4. DISCUSSION

Sexually antagonistic genetic variance arises when the fitness optima for shared traits differ between males and females, generating intralocus sexual conflict in which the adaptive evolution of each sex impedes the other (Lande 1980; Rice 1984; Parker & Partridge 1998; Rice & Chippindale 2001; Chapman *et al.* 2003; Bonduriansky & Chenoweth in press). Sexually antagonistic genetic variance will result in a negative $r_{\mathrm{w}}^{\mathrm{fm}}$, which can have important consequences for the maintenance of genetic variation, the evolution of recombination and rates of adaptation. It may also reduce or even eliminate the genetic benefits that accrue to females from mating with high-fitness males (Chippindale *et al.* 2001; Pischedda & Chippindale 2006; Kirkpatrick in press).

Using a laboratory population of *D. serrata*, we estimated a negative $r_{\mathrm{w}}^{\mathrm{fm}}$ in a yeast food environment to which the population was well adapted, as well as in a novel corn food environment ($r_{\mathrm{w}}^{\mathrm{fm}} = -0.34$ and -0.73, respectively). This novel environment displaced the population from its adaptive peak: female fitness was initially reduced by 35 per cent on average, yet was shown previously to rapidly increase (52% in 16 generations) during adaptation to this environment (Rundle *et al.* 2006). Determining whether the persistence and apparent strengthening of the negative $r_{\mathrm{w}}^{\mathrm{fm}}$ is a general outcome of colonization of a novel environment, as opposed to an effect specific to the novel environment we used, will require additional studies across a range of environments. Nevertheless, our results indicate that the importance of sexually antagonistic fitness variation is not necessarily an artefact of an unusually consistent laboratory environment, as has been previously suggested for laboratory populations (Chapman *et al.* 2003).

Our estimates of negative $r_{\mathrm{w}}^{\mathrm{fm}}$ in both environments may be conservative. Because the fitness of both sexes was measured by counting the number of adult offspring produced, egg-to-adult survival of male and female offspring was included in the fitness estimates of both sexes. These shared fitness components will covary positively, biasing $r_{\mathrm{w}}^{\mathrm{fm}}$ to be more positive than it would otherwise be. This bias could be substantial if sexually antagonistic genetic variance was prevalent for larval fitness. Estimates from *D. melanogaster* suggest that this is not the case (Chippindale *et al.* 2001), although this has not been determined for *D. serrata*.

Our current quantitative genetic analysis of fitness is consistent with a recent evolution experiment that independently manipulated the opportunities for both natural and sexual selection during adaptation of the same *D. serrata* population to the same novel corn food environment. After 16 generations in the novel environment, female fitness increased by 52 per cent on average due to natural selection alone. By contrast, and despite strong sexual selection on display traits (Chenoweth *et al.* 2008), sexual selection failed to promote adaptation either on its own or in combination with natural selection (Rundle *et al.* 2006). The strong negative intersex genetic correlation for fitness estimated here in the corn environment provides a potential explanation for this failure: genetic variance for fitness in corn is predominately sexually antagonistic in this population and is therefore not conducive to a good genes process. The negative intersex correlation for fitness also makes a clear prediction: given the dramatic increase in female fitness that occurred over 16 generations in the corn environment, male fitness should have decreased substantially as a correlated response. Unfortunately, male fitness was not tracked during that experiment, but this will be an important focus of future studies.

A negative $r_{\mathrm{w}}^{\mathrm{fm}}$ in yeast is perhaps not unexpected: given a constant environment with moderate population size, mutations that are unconditionally beneficial (deleterious) in both sexes should be fixed (eliminated) fairly efficiently. The majority of genetic variation left segregating may therefore arise from alleles that persist at intermediate frequencies for longer because they are under conflicting selection. Sexual antagonism, in which an allele is favoured in one sex but selected against in the other, is a primary source of such conflict because other processes (e.g. spatial and temporal variation in selection) are minimized under laboratory conditions. That $r_{\mathrm{w}}^{\mathrm{fm}}$ remained negative—potentially even strengthening—in the novel environment, is perhaps less intuitive, although it is consistent with one possible scenario concerning the resolution of sexual conflict via the evolution of sexual

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dimorphism after long-term adaptation to consistent selection (Lande 1980, 1987). There are at least two potential processes that could be contributing.

First, the average strength of sexual antagonism may vary between novel mutations overall and the small subset of these that are left segregating after long-term adaptation. Standing genetic variation for fitness in yeast has never been subjected to selection in the novel corn environment, so in the majority of cases its effects should be deleterious in both sexes, resembling that expected for novel mutations in this environment. The strong negative r_w^{fm} in corn implies that the majority of this variation has a much greater fitness cost in one sex than the other. This could occur, for example, due to variation in the sex chromosomes (i.e. in males all mutations on the X would be expressed, whereas in females they would be largely masked).

Second, long-term evolution under consistent sexual conflict could favour the evolution of mechanisms that allow males and females to express their sex-specific optima for the traits determining fitness, thereby generating sexual dimorphism that ameliorates sexual conflict and makes r_w^{fm} less negative (Lande 1980, 1987). Such mechanisms include (i) the physical relocation of loci to the sex chromosomes (Fisher 1930; Rice 1984), (ii) the evolution of modifiers that permit the sex-limited expression of loci (Chenoweth *et al.* 2008), (iii) the duplication of autosomal loci followed by their sex-limited expression (Rhen 2000; Rice & Chippindale 2002), and (iv) the evolution of parent-of-origin effects via genomic imprinting (Day & Bonduriansky 2004). If any of these mechanisms are environment-dependent in their action, they could break down in the novel corn environment, causing sexual conflict to increase. Environment-dependent expression levels of a modifier permitting sex-limited expression are plausible, and recent experimental work suggests the possibility of environment-dependent genomic imprinting (Bonduriansky & Head 2007).

The above two processes are not mutually exclusive, and determining their contributions to changes in r_w^{fm} in novel environments will be a difficult task. An important first step will be to study how r_w^{fm} changes on colonization of a range of novel environments and its subsequent evolution as populations adapt to these environments.

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