JOURNAL OF Evolutionary Biology

doi: 10.1111/jeb.12713

Intralocus sexual conflict, adaptive sex allocation, and the heritability of fitness

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Keywords:

animal model;
genetic variance;
lizard;
reproductive success;
sexual selection;
survival.

Abstract

Intralocus sexual conflict arises when selection favours alternative fitness optima in males and females. Unresolved conflict can create negative between-sex genetic correlations for fitness, such that high-fitness parents produce high-fitness progeny of their same sex, but low-fitness progeny of the opposite sex. This cost of sexual conflict could be mitigated if high-fitness parents bias sex allocation to produce more offspring of their same sex. Previous studies of the brown anole lizard (*Anolis sagrei*) show that viability selection on body size is sexually antagonistic, favouring large males and smaller females. However, sexual conflict over body size may be partially mitigated by adaptive sex allocation: large males sire more sons than daughters, whereas small males sire more daughters than sons. We explored the evolutionary implications of these phenomena by assessing the additive genetic (co)variance of fitness within and between sexes in a wild population. We measured two components of fitness: viability of adults over the breeding season, and the number of their progeny that survived to sexual maturity, which includes components of parental reproductive success and offspring viability (RS^V). Viability of parents was not correlated with adult viability of their sons or daughters. RS^V was positively correlated between sires and their offspring, but not between dams and their offspring. Neither component of fitness was significantly heritable, and neither exhibited negative between-sex genetic correlations that would indicate unresolved sexual conflict. Rather, our results are more consistent with predictions regarding adaptive sex allocation in that, as the number of sons produced by a sire increased, the adult viability of his male progeny increased.

Introduction

Males and females often experience very different forms of natural selection acting on the same phenotypic traits, yet they share most of the same genes for these traits. This shared genome is predicted to impose a constraint on the degree to which the sexes can evolve towards their respective phenotypic optima (Lande, 1980; Roff, 1997). If traits subject to sexually antagonistic selection are positively genetically correlated between the sexes, then high-fitness parents should produce high-fitness progeny of their sex but low-fitness progeny of the opposite sex, a phenomenon known as intralocus sexual conflict (Bonduriansky & Chenoweth, 2009; van Doorn, 2009). Positive genetic correlations are commonly observed for traits with different fitness optima in males and females (Poissant *et al.*, 2010), and recent reviews suggest that sexual conflict may be widespread (Bonduriansky & Chenoweth, 2009; Cox & Calsbeek, 2009).

Whereas intralocus sexual conflict arises due to positive intersexual genetic correlations for pheno-

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typic traits, it imposes its costs by creating negative intersexual genetic correlations for fitness (Chippindale *et al.*, 2001; Qvarnstrom *et al.*, 2006; Foerster *et al.*, 2007; Delcourt *et al.*, 2010). This can erode the fitness benefits of mate choice if high-fitness parents produce low-fitness progeny of the opposite sex (Fedorka & Mousseau, 2004; Pischedda & Chippindale, 2006). In light of this potential cost, sexual conflict theory predicts that selection should favour the adaptive allocation of progeny sex as a function of parental quality [e.g. high-fitness sires producing more sons (Fawcett *et al.*, 2007, 2011; Pryke & Griffith, 2009; Blackburn *et al.*, 2010; Katsuki *et al.*, 2012; Booksmythe *et al.*, 2013)]. Sexual conflict, and the mechanisms for its potential resolution, should therefore have important implications for genetic variation in fitness. Understanding these implications is important because genetic variation in fitness underlies the capacity for adaptive evolution (Fisher, 1958; Falconer & MacKay, 1996; Kruuk *et al.*, 2000).

Previous studies of the brown anole (*Anolis sagrei*) suggest the potential for a high degree of intralocus sexual conflict (Calsbeek & Bonneaud, 2008; Cox & Calsbeek, 2010a). Adult male brown anoles are, on average, 25–35% larger and 2–3 times more massive than adult females. This sexual dimorphism is reinforced by natural selection, which tends to be positive and directional in males, but stabilizing in females (Cox & Calsbeek, 2010b). Brown anoles produce disproportionately more sons with the sperm from large sires, and this cryptic fertilization bias appears to be adaptive in that sons of large sires have high juvenile survival in the wild, whereas those of small sires have low juvenile survival (Cox & Calsbeek, 2010a). By contrast, the survival of daughters is independent of sire size. These data are at least partially consistent with the presence of intralocus sexual conflict (males and females have alternative fitness optima for body size) and a sex-specific good-genes effect in which large sires carry genes for the production of high-quality sons.

Here, we used a multiyear study of a pedigreed wild population to test whether patterns of heritability in fitness are consistent with the presence of unresolved intralocus sexual conflict, and whether patterns of progeny sex allocation are adaptive with respect to progeny fitness (Table 1). First, if genetic variation in fitness is present and sexual conflict is unresolved, then fitness should be heritable within sexes (i.e. from sires to sons or dams to daughters), but negatively genetically correlated between sexes [i.e. from sires to daughters or dams to sons; Foerster *et al.*, 2007]. Collectively, these relationships should manifest as a negative between-sex genetic correlation for fitness or components of fitness (e.g. survival, reproductive success). Second, if previously established patterns of biased sex allocation (Calsbeek & Bonneaud, 2008; Cox & Calsbeek, 2010a) are based on sex-specific good-genes effects, then sires that produce relatively more sons should also produce sons with higher survival and reproductive success. Support for both predictions would indicate that biased sex allocation mitigates some of the fitness costs associated with unresolved sexual conflict. However, even in the absence of a negative between-sex genetic correlation for fitness, biased sex allocation could still confer adaptive benefits if the genetic correlation for fitness is < 1.

We tested these predictions in a wild population by estimating two important components of fitness. First, we measured breeding-season survival of adult males and females and, in the subsequent generation, of their progeny as adults. Second, for each adult male and female, we used genetic parentage assignments to estimate the number of their progeny that survived to reach sexual maturity the following generation. This measure of fitness integrates both the reproductive success of adults and the subsequent viability of their progeny as juveniles. As such, we refer to this component of fitness as reproductive success after accounting for offspring viability ('RS^V'). We report parent–offspring correlations for each measure, and we then used generalized linear mixed models (i.e. animal models; Wilson, 2008) to estimate heritability and between-sex genetic correlations for these two components of fitness.

Materials and methods

Study species

This study took place on Kidd Cay, near Georgetown on Great Exuma, The Bahamas (23°31′N 75°49.5′W), in a population of *A. sagrei* that we have monitored continuously since 2002. Data in this study are from 2005 to 2008, years in which we performed mark–recapture studies on all males and females on the island and also collected tissue samples from all adults and their progeny (parentage analysis was conducted from

Table 1 Intralocus sexual conflict theory predicts that unresolved sexual conflict should result in positive heritability for fitness (e.g. viability and/or RS^V) between parents and their same-sex progeny but negative between-sex genetic correlations between parents and their opposite-sex progeny. The adaptive sex allocation hypothesis predicts that sires that produce more sons should produce sons with higher fitness, whereas sires that produce more daughters should produce daughters with higher fitness.

	Response variable	
Predictor variable	Fitness of sons	Fitness of daughters
Intralocus sexual conflict theory		
Fitness of sire	+	−
Fitness of dam	−	+
Adaptive sex allocation theory		
Number of sons by sire	+	−
Number of daughters by sire	−	+

2005 to 2007). Most females (~80%) produce offspring with multiple males (Calsbeek *et al.*, 2007), and breeding usually begins in March. Females lay one or two eggs at approximately 10-day intervals throughout the breeding season, which typically ends by September (Calsbeek *et al.*, 2007). Hatchlings emerge approximately 35–50 days after eggs are laid and receive no parental care. Although hatchlings emerge over an extended period ranging from May through October, most do not attain sexual maturity until the following breeding season. Hence, the number of surviving progeny present at the beginning of the breeding season provides an index of the extent to which parents contribute to the subsequent breeding cohort. Most adults (> 85%) in this population die before reaching a second breeding season, and the bulk of mortality occurs during the summer months (May–October; Calsbeek & Smith, 2007). Although an imperfect measure, we use the numbers of surviving progeny captured each spring as our estimate of the sex ratio produced by each sire or dam. We do this with the caveat that previous studies have documented sex-specific differences in juvenile survival (Urbach *et al.*, 2013), such that adult sex ratios are often female-biased in natural populations.

Each year in May, we captured (with a hand-held noose) and measured the mass (to the nearest 0.1 g) and snout–vent length (SVL; to the nearest mm) of all individuals on the island. We marked each lizard with a unique combination of coloured elastomer dyes injected subdermally into the ventral side of their hind- and forelimbs (Nauwelaerts *et al.*, 2000). Dyes are invisible to predators and other lizards and so do not affect survival or social interactions. We removed a 2-mm sample of tail tissue for genetic analyses and returned all lizards to their original point of capture within 4–6 h. Each year in fall (late August–early September), we performed a recapture census of the entire island to assess survivorship. Each measured lizard received a small spot of white paint on its hindlimb to prevent immediate recapture during a census.

Microsatellite genotyping

We extracted whole-genomic DNA using a DNeasy Blood and Tissue Kit (Qiagen, Valencia, CA, USA), according to the manufacturer's protocols for tissue extraction, except that we eluted samples into a final volume of 30 μL AE buffer to maximize DNA yield. We conducted PCR on each sample using a library of 10 microsatellite markers. Markers AAAG-38, AAAG-61, AAAG-68, AAAG-70, AAAG-76, AAAG-77, AAAG-91 and AAAG-94 were previously designed for *A. sagrei* (Bardelbeen *et al.* 2004). Markers Acar11 and Acar23 were developed by Wordley *et al.* (2011) using the *A. carolinensis* genome, and we verified amplification quality and polymorphism in our study population of *A. sagrei* prior to this study. We grouped markers into two pool sets of five markers each and individually labelled forward primers with a series of unique fluorescent tags (Life Technology, Waltham, MA USA) that we assigned according to fragment sizes for each pool. We amplified microsatellite markers in multiplex PCR of each pool using Type-it kits (Qiagen). We conducted each multiplex PCR in a 10-μL volume using 1 μL DNA template, 5 μL Master Mix (Qiagen), 1 μL primer mix and 3 μL molecular-grade water. We optimized primer concentrations to marker-specific amplification rates based on preliminary genotyping runs and diluted PCR products for genotyping in 18.85 μL Hi-Di Formamide (Life Technology) and 0.15 μL LIZ sizing standard (Life Technology). Finally, we genotyped the diluted products on an ABI3730 Genetic Analyzer (Life Technology) and then binned and verified fragment sizes by eye using GENEMAPPER software (Life Technology).

Parentage analysis

We assigned parentage for each individual by considering the previous year's cohort of adults as candidate parents (e.g. offspring from the 2006 cohort of lizards were assigned to candidate parents from the 2005 data set and so on for 2007 and 2008). Because marked individuals captured during spring censuses were known to have been alive in the previous spring, these individuals were excluded from the offspring pool for parent assignment. We assigned parentage using the software package COLONY (Owen & Wang, 2009). We allowed COLONY to individually assign maternity and paternity using the pairwise approach (Owen & Wang, 2009) and accepted candidate sires and dams as parents in cases where the probability of parentage was 0.8 or higher. Although not all progeny could be assigned to parents in each year, we assumed that variation in assignment was random with respect to family and that this effect therefore did not bias our analyses.

Statistical analysis

We used generalized linear models (GzLM) to test our predictions regarding the sex-specific nature of correlations in fitness components between parents and progeny. To account for the Poisson distribution of RS^V, we performed significance tests using GzLM with log link functions. Similarly, to account for the binomial distribution of survival (live/die), we used GzLM with logit links. We used separate mixed models that included parent ID as a random effect to account for statistical nonindependence of siblings, and we report regression coefficients ($\beta \pm$ SE) from these models as estimates of the correlations between parents and offspring. We tested for sex-specific relationships among fitness components by first including interactions between a factor for 'sex' and the other independent variables. Nonsignificant interaction terms were removed from models, but we included factors for sex

and year in all analyses. These correlations were used as a straightforward means to interpret each combination of parent–offspring relationships. To complement the linear models, and to estimate heritability and genetic correlations, we next partitioned the variances from our pedigree using a series of generalized linear mixed models (i.e. animal models; Wilson, 2008).

Animal models

We ran separate animal models (restricting data to only the first observation per animal as there were very few cases in which an individual survived multiple years) to estimate the heritability and between-sex genetic correlation of the two fitness components: adult viability and reproductive success (RS^V). We used the Bayesian framework implemented in the package MCMCGLMM v. 2.21 (Hadfield, 2010) in R v. 3.0.1 (Team, 2013). Models for viability and RS^V were assigned categorical (logit link) and Poisson distributions, respectively.

All models were run for ≥5 000 000 iterations, with a burn-in of ≥50 000 iterations, saving samples every 1000 iterations. This resulted in a sample size of ≥4950. Convergence and mixing properties were initially verified by visual inspection of the chains, as well as by ensuring that the autocorrelation did not frequently exceed 0.1, as suggested by Hadfield (2014, p. 22), to ensure a sufficient effective sample size (i.e. > 1000). We ran Heidelberger and Welch's convergence diagnostic as implemented in the package coda (Plummer *et al.*, 2012) to verify that the number of iterations was adequate for chains to achieve convergence. For the binomial model, the latent variables were saved to verify that they did not exceed 20 (Hadfield, 2014, p. 132). Finally, for the selected models, we ran a second chain and compared the outputs by checking for differences between the variance of the first and second chain in relation to the variances within chains (i.e. Gelman–Rubin diagnostic; Gelman & Rubin, 1992), also implemented in package coda (Plummer *et al.*, 2012).

We conducted model selection based on the deviance information criteria (DIC; Spiegelhalter *et al.*, 2002) to determine the inclusion or removal of sex and year as fixed effects, and dam (i.e. maternal effects) as a random effect. To estimate the between-sex genetic correlations (r_{mf}) for fitness components, we fit a random-slope model, including sex as a fixed effect and the interaction of sex and animal as a random term. Significance of the random slope was also tested by model selection. For the models with the random slope, we fit a residual covariance structure that allowed for each of the sexes to have different residual variances, with covariances set as 0 [i.e. rcov = < idh(sex):units], as suggested by Hadfield (2014, p. 71).

Narrow-sense heritability is defined as the proportion of variance assigned to the random effect 'animal' (i.e. additive genetic variance), over the sum of this variance and the residual variance (i.e. total phenotypic variance) (Falconer & MacKay, 1996). However, as the residual variance estimates for non-Gaussian models are correlated with the mean of the population and, therefore, are not comparable to other estimates of variance, heritability estimates are presented in the latent scale (Nakagawa & Schielzeth, 2010; Morrissey *et al.*, 2014). Latent-scale heritability estimates are obtained by adding the variance component related to the link function: for the binomial model, heritability of viability ($h^2_{\text{V-latent}}$) was estimated as: $\sigma^2_A/(\sigma^2_P + \pi^2/3)$; for the Poisson model, heritability for RS^V ($h^2_{\text{RS-latent}}$) was estimated as: $\sigma^2_A/\{\sigma^2_P + \log[1/(\exp(\beta_0)+1)]\}$, where σ^2_A is the additive genetic variance, σ^2_P is the total phenotypic variance, and β_0 is the intercept of the Poisson model. The mode and credible interval of the posterior distribution of heritability estimates are presented for the best model selected through DIC for each of the variables. We calculated between-sex genetic correlations as the covariance between the sexes divided by the product of the standard deviations for each of the sexes (Bonduriansky & Chenoweth, 2009).

For the categorical models, the prior for the residual variance was fixed at 1, so the posterior distribution is proper (Hadfield, 2014). In addition, we used chi-square distributions with 1 degree of freedom as priors for the random effects, as this resulted in a cumulative distribution that was close to a uniform distribution for the estimates of heritability (de Villemereuil *et al.*, 2013). For the prior of fixed effects, we used the default diffuse normal distribution (mean of 0 and variance of 10^8).

We ran models testing for between-sex genetic correlation in viability twice: first, using the same chi-square priors as described before for the random-intercept model; and second, with an inverse-Wishart prior with variance of 0.02 and the belief parameter set to 3 (i.e. dimension of the variance–covariance matrix plus 1), which is considered an uninformative (i.e. uniform) prior for correlation (Hadfield, 2014), as it results in an uniform distribution between the intervals −1 and 1 (Hadfield, 2014).

For the Poisson model, the prior for the residual variance was the default inverse-Wishart with variance 1 and a belief parameter (nu) of 0.002. For the random-effects prior, we used a parameter-extended prior for the 'animal' term, with prior mean (alpha.mu) of 0 and prior covariance matrix (alpha.V) of I*1000, where I is the identity matrix. A parameter-extended prior takes the form of a noncentral F-distribution, as described by Gelman (2006), and is recommended to account for near-zero variances (Hadfield, 2014). For the dam term, we ran models both with the parameter-extended prior and with an inverse-Wishart prior, as the initial run with the parameter-extended prior indicated that the variance associated with dam was much larger than the variance associated with the animal term. The

fixed-effects priors were a diffuse normal distribution as described for binomial models.

For models testing for a between-sex genetic correlation in RS^V, we used the inverse-Wishart distribution for the prior of the residual variance of both sexes, and a diffuse normal distribution with mean 0 and variance of 10^8 for each sex. We ran each model twice, with two different priors for the interaction between the sex and animal terms in the random structure: first, with a parameter-extended prior as described for the random-intercept models, and second, with an inverse-Wishart prior with variance of 0.02 and the belief parameter set to 3, as described for the random-slope model of viability. These models had comparable results, and we present the results for the second set of models, as the mixing properties of the chain were better. As random-intercept models indicate a large proportion of variance associated with maternal effects, the random-slope models were also run with dam identity included in the random structure. For the models that included the dam identity, we also ran the models with two different priors: an inverse-Wishart prior with variance of 0.02 and the belief parameter set to 1, and a parameter-extended prior.

Adaptive sex allocation

We tested for adaptive sex allocation by asking whether the mean adult fitness of sons and daughters produced by each sire or dam (using both viability and RS^V) was correlated with the sex ratio of progeny (measured at sexual maturity) produced by each sire or dam. To complement this analysis of sex ratio with a related measure of sex allocation, we also used the total numbers of sons or daughters that survived to maturity as independent variables in analogous models. We regressed progeny fitness components against either sex ratio, number of sons, or number of daughters to test the specific prediction that sires who produced more sons (or daughters) also produced sons (or daughters) with greater adult viability (i.e. a good-genes effect). Similar to models described above, we used GzLM with log link and logit links functions to account for the distributions of RS^V and survival, respectively. We used separate mixed models that included parent ID as a random effect to account for statistical nonindependence of siblings, and we report regression coefficients ($\beta \pm$ SE) from these models as estimates of the correlations between parents and offspring. These models also included a covariate for body mass, to account for variation in offspring quality that might arise due to size. Finally, to compare with previous studies (Calsbeek & Bonneaud, 2008; Cox & Calsbeek, 2010a), we estimated the relationship between fitness components of progeny and sire body size using GzLM (as above) with sire body mass as the independent variable. We also included year as a factor in these models.

Results

Parent–offspring correlations in fitness components

We assigned parents to a total of 581 progeny over the course of this study. Of these, 300 were assigned to sires and 281 to dams, and 162 were assigned both parents with at least 80% confidence. We assigned parents to 294 progeny in 2005 (~62% of 474 total progeny), 121 progeny in 2006 (~41%) and 166 progeny in 2007 (~35%). RS^V was not significantly different between males (mean $RS^V \pm$ SE = 1.66 ± 0.09) and females (1.67 ± 0.1; GzLM: $\chi^2 = 0.004$, $P = 0.95$), but RS^V varied by year ($\chi^2 = 125.72$, $P < 0.0001$).

RS^V was not correlated between sires and sons ($\beta = 0.08 \pm 0.08$; $\chi^2 = 1.24$, $P = 0.42$; Fig. 1a) or between dams and sons ($\beta = -0.02 \pm 0.08$; $\chi^2 = 0.09$, $P = 0.79$; Fig. 1b). Although RS^V was positively correlated between sires and their daughters ($\beta = 0.22 \pm 0.08$; GzLM: $\chi^2 = 10.71$, $P = 0.01$; Fig. 1c), it showed a nonsignificant negative correlation between dams and daughters ($\beta = -0.09 \pm 0.07$; $\chi^2 = 3.12$, $P = 0.22$; Fig. 1d). When pooling progeny of both sexes, RS^V was positively correlated between sires and their offspring ($\beta = 0.12 \pm 0.04$; $\chi^2 = 9.68$, $P = 0.02$; Fig. 1e) but was not correlated between dams and their offspring ($\beta = -0.06 \pm 0.05$; $\chi^2 = 2.61$, $P = 0.14$; Fig. 1f).

Adult viability, measured as survival over the breeding season, was not correlated between sires and sons ($\beta = 0.02 \pm 0.04$: $\chi^2 = 0.77$, $P = 0.65$; effect of year $P = 0.005$) or between dams and sons ($\beta = -0.02 \pm 0.05$: $\chi^2 = 0.32$, $P = 0.67$; effect of year $P = 0.20$). Nor was adult viability correlated between sires and daughters ($\beta = 0.04 \pm 0.04$: χ^2=0.30, $P = 0.38$; effect of year $P = 0.63$) or dams and daughters ($\beta = 0.02 \pm 0.05$, χ^2=0.38, $P = 0.65$; effect of year $P = 0.35$). Patterns remained similarly nonsignificant when we pooled progeny of both sexes ($\beta = 0.02 \pm 0.03$, $P = 0.44$, and $\beta = -0.005 \pm 0.03$, $P = 0.87$ for sires and dams, respectively).

Heritability and between-sex genetic correlations for fitness

Using adult viability as a fitness component, model selection based on DIC indicated moderate evidence favouring a model with year as a fixed effect, in comparison with a model with only the global intercept (ΔDIC ~ 10; Table 2). However, because the inclusion of fixed effects will cause heritability estimates to increase due to a reduction in σ^2_P (Wilson, 2008), we report heritability for the model excluding fixed effects. Heritability of adult viability was estimated as 0.0020 (mean of posterior = 0.1427), with a 95% credible interval of 6.629e-10 to 0.392. The mean of the additive genetic variance posterior distribution was estimated as 0.8588 (95% CI: $2.844e^{-09}$ to 2.773), and

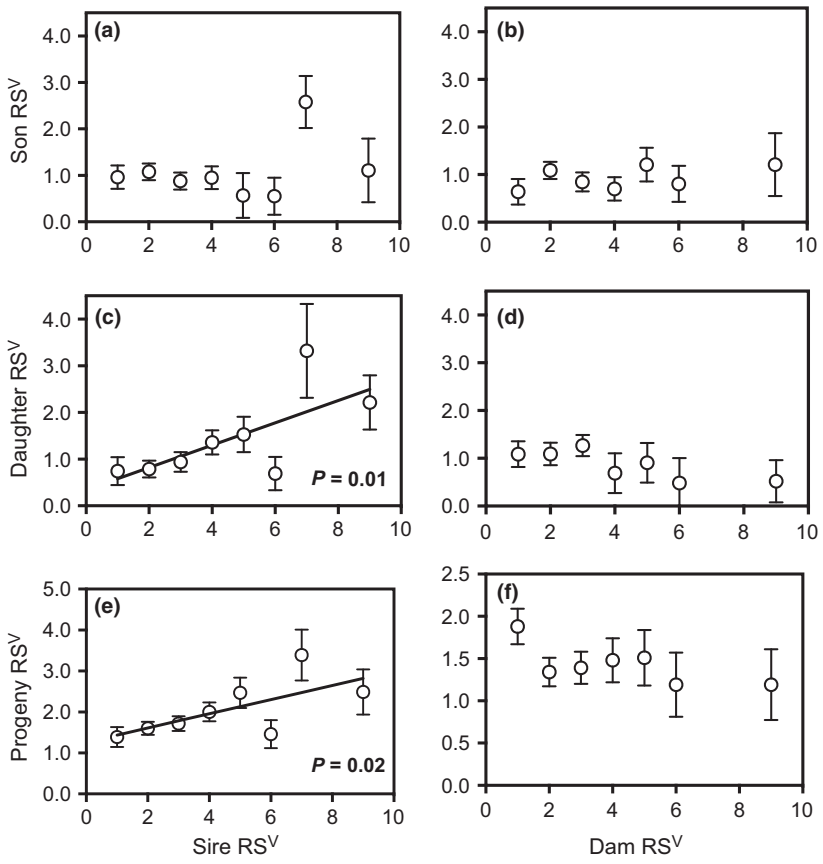

Fig. 1 Heritability of fitness, measured as the reproductive success of offspring that survived to maturity (RS^V). RS^V was not heritable between sires and sons (a) or between dams and sons (b). RS^V was heritable father to daughter (c) and was not correalted between daughters and dams (d). For illustrative purposes, panels show simple linear regressions vs. means (plus/minus some estimate of error, not sure what) from each reproductive size interval. Correlation-based models used individuals or family means as data points and included a factor for year and a covariate for offspring mass. *P*-values are from generalized linear models with log link function to account for Poisson distributions of reproductive success.

the between-sex genetic correlation (r_{mf}) for adult viability was 0.006 (95% CI: −0.060 to 0.062).

Using RS^V as a fitness component, model selection based on DIC favoured a model with only the global intercept, and a random structure including dam (Table 2). Based on this model, heritability of RS^V was estimated as 0.000145 (mean of posterior is 0.006), with a 95% credible interval of $8.026e^{-10}$ to 0.023. The mean of the variance posterior distribution associated with dam identity was estimated as 0.442 (95% CI: 0.160 to 0.745), whereas the mean variances for the animal and residual components were estimated as 0.005 (95% CI: $6.105e^{-11}$ to 0.020) and 0.011 (95% CI: 0.0002 to 0.038), respectively. This indicates that a large proportion of the variance for RS^V was associated with maternal effects. The results reported are from the model with the extended parameter prior for dam (as variance is still small), but the results were robust to the prior specification (i.e. the estimate from the model with an inverse-Wishart distribution as the prior for the dam was similar: $h^2_{RS^V\text{-latent}} = 4.137e^{-05}$ (95% CI: $2.316e^{-11}$ to 0.022).

Model comparison indicated that the random-slope model was no better than the simple, random-intercept model ($\Delta DIC \gg 10$; Table 3). As model selection for the random-intercept model indicated large variance associated with dam identity, we conducted model selection among random-slope models. This also favoured a model including dam in the random structure (Table 3). The between-sex genetic correlation (r_{mf}) for RS^V from the model with the extended parameter prior for dam was estimated as -0.045 (mode of the posterior) with a 95% credible interval ranging from −0.720 to 0.622. The results are robust to the prior specification for the dam component (i.e. the estimate from the model with an inverse-Wishart distribution as the prior for the dam was similar: $r_{mf} = 0.051$, 95% CI −0.718 to 0.626). None of the random-slope models, however, passed the Heidelberger and Welch's diagnostic for the estimation of between-sex covariance, even with an increased sample size of 7425, suggesting that there is not enough information in the pedigree for reliable estimation of the between-sex correlation.

Adaptive sex allocation

The sex ratio of progeny produced by a sire was not associated with the adult viability of his sons (GzLM $\chi^2 = 0.12$, $P = 0.74$) or daughters (GzLM $\chi^2 = 0.71$, $P = 0.38$). However, the adult viability of male offspring

Table 2 The animal model revealed no evidence for heritability of survival (h^2 = 0.0020, 95% credible interval of 6.629e-10 to 0.392; see text). Table entries show model subsets, indicating the fixed and random effects included in each model, and the respective DIC for each models. Bold text indicates the model used for final analyses, which was chosen based on consideration of both ΔDIC scores and a preference for models with relatively few parameters.

Fixed effects	Random effects	DIC	ΔDIC
Heritability			
Year + sex	animal	747.520	*
Year	animal + dam	747.741	0.221
Year	animal	748.706	1.186
Sex	animal + dam	757.487	9.967
	animal + dam	757.648	10.128
	animal	**758.227**	**10.707**
Sex	animal	758.301	10.781
Between-Sex Correlation Models*			
Sex	us(sex):animal	760.321	12.801

*Lowest DIC model.

Table 3 The animal model revealed no evidence for heritability of reproductive success (RS^V). Table entries show model subsets indicating the fixed and random effects included in each model, and the respective DIC for each of the models. Bold text indicates the model used for final analyses, which was chosen based on consideration of both ΔDIC scores and a preference for models with relatively few parameters.

Fixed effects	Random effects	DIC	ΔDIC
Heritability			
	animal + dam	**2460.720**	*
Sex	animal + dam	2461.717	0.997
Year	animal + dam	2466.974	6.254
Year	animal	2506.119	45.399
Year + Sex	animal	2507.617	46.897
	animal	2514.823	54.103
Sex	animal	2516.206	55.486
Between-Sex Correlation Models			
Sex	**us(sex):animal + dam**	**2466.112**	**5.392**
Sex	us(sex):animal	2519.636	58.916

*Lowest DIC model.

was positively correlated with the number of a sire's sons that reached maturity ($\beta = 0.11 \pm 0.04$; GzLM $\chi^2 = 8.01$, $P = 0.01$; Fig. 2a), whereas the adult viability of female offspring was not significantly correlated with the number of a sire's sons that reached maturity ($\beta = -0.01 \pm 0.04$; $\chi^2 = 0.12$ $P = 0.79$) (Table 4). These sex-specific slopes were significantly different from one another (number of sons × offspring sex: $\chi^2 = 4.41$ $P = 0.04$). Thus, sires that recruited more sons into the next breeding generation also produced male offspring with higher adult viability, but they did not produce female offspring with disproportionately high or low adult viability. The number of mature daughters that a sire produced was not correlated with the adult viability of his male offspring ($\beta = 0.001 \pm 0.04$; $\chi^2 = 0.03$, $P = 0.99$, Fig. 2b) or that of his female offspring ($\beta = 0.06 \pm 0.04$; $\chi^2 = 4.03$, $P = 0.06$).

The sex ratio of progeny produced by a sire was not associated with the RS^V of his sons (GzLM $\chi^2 = 0.12$, $P = 0.74$), although it was positively correlated with the RS^V of his daughters (GzLM $\chi^2 = 9.41$, $P = 0.009$). Moreover, sires that recruited more sons into the next breeding generation also produced progeny with disproportionately higher RS^V. The number of mature sons produced by a sire was positively correlated with the RS^V of his male offspring ($\beta = 0.28 \pm 0.12$; $\chi^2 = 5.62$, $P = 0.04$ Fig. 2c) and with the RS^V of his female offspring ($\beta = 0.36 \pm 0.11$; $\chi^2 = 13.01$, $P = 0.003$). The number of mature daughters produced by a sire was not correlated with the RS^V of his offspring of either sex ($P = 0.84$ and 0.28 for male and female offspring, respectively; Fig. 2d), nor were the numbers of mature progeny of either sex produced by dams correlated with RS^V of either male or female offspring (all $P > 0.3$).

Sire body mass was unrelated to the adult viability of either sons ($\chi^2 = 0.62$, $P = 0.62$) or daughters ($\chi^2 = 2.12$, $P = 0.14$). Sire body mass was also unrelated to the RS^V of either sons ($\chi^2 = 0.16$, $P = 0.68$) or daughters ($\chi^2 = 2.33$, $P = 0.13$). Results were similar when we used sire SVL instead of body mass (not shown).

Discussion

Negative between-sex genetic correlations for fitness (or components of fitness) are one key indicator of intralocus sexual conflict (Bonduriansky, 2007). In the absence of biased sex allocation, this genomic architecture can constrain adaptive evolution because selection for increased fitness within each sex will be counteracted by a reduction in fitness due to the production of low-fitness progeny of the opposite sex (Fedorka & Mousseau, 2004; Pischedda & Chippindale, 2006). Still, evidence for negative between-sex genetic correlations remains relatively rare, and the mechanisms that form these genetic correlations are poorly understood (Punzalan *et al.*, 2014). Despite substantial indirect evidence for sexual conflict in *A. sagrei* (Calsbeek & Bonneaud, 2008; Cox & Calsbeek, 2010a,b), we did not find any positive or negative genetic correlations for adult viability in within- or between-sex comparisons of progeny and parents, and the heritability of this fitness component was effectively zero.

Our measure of reproductive fitness, RS^V, which includes elements of progeny viability, was positively correlated between sires and their daughters, but not between sires and their sons. The correlation between dams and daughters was negative for RS^V (Fig. 1d). Animal models revealed that the variance in these effects likely arises due to strong maternal effects,

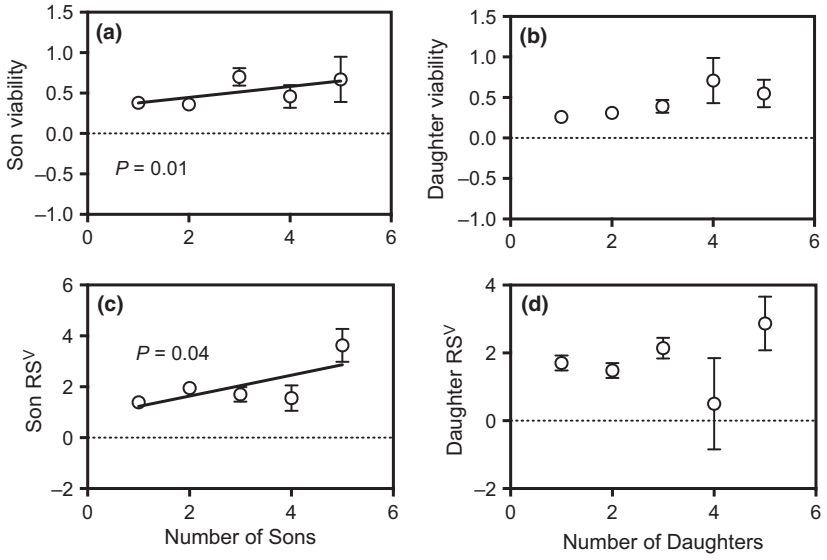

Fig. 2 Adult viability of sons (solid line) was positively correlated with the number of mature sons produced by a sire (a). Adult viability of daughters was not significantly correlated with the number of mature daughters produced by a sire (b). The RS^V of sons, measured as the number of their own offspring that reached maturity, was positively correlated with the number of mature sons produced by sires (c), but there was no relationship between the RS^V of daughters and the number of mature daughters produced by sires (d). For illustrative purposes, panels show simple linear regressions and mean values (±SE) from each reproductive size interval. Statistical models used individuals and included a factor for year and a covariate for offspring mass. *P*-values are from generalized linear models with logit link functions to account for binomial distributions of survival (live/die) in panels a and b, or log link function to account for Poisson distributions of reproductive success in panels c and d (see text and Table 4 for details.).

rather than additive genetic variance *per se*. Strong maternal effects in *A. sagrei* are known to reduce heritability by inflating total phenotypic variance (Warner & Lovern, 2014). In sum, we found no evidence that current intralocus sexual conflict in *A. sagrei* is of sufficient strength to generate negative between-sex genetic correlations for viability and reproductive success, nor did we find strong evidence that these components of fitness are heritable within either sex.

Although comparisons of the same components of fitness between parents and offspring were not in line with predictions from sexual conflict theory, comparisons of offspring viability as a function of the number of sons produced by sires were generally consistent with adaptive sex allocation (Fawcett *et al.*, 2007, 2011; Booksmythe *et al.*, 2013). Sires that recruited more mature male offspring into the next generation produced sons with higher adult viability. Similarly, sires that recruited more mature female offspring into the next generation produced daughters with high adult viability. One explanation for this pattern, and one that is consistent with our previous work on sexual conflict in this system (Cox & Calsbeek, 2010a), is that sires pass on sex-specific genes for progeny viability. Large sires are more likely to produce sons, and these sons tend to have high viability in the wild relative to offspring from smaller sires (Cox & Calsbeek, 2010a). Previous work has also demonstrated a tendency for smaller sires to produce more daughters (Calsbeek & Bonneaud, 2008), although that study did not include data on juvenile viability to test whether daughter production by small sires is likewise adaptive. If the biased secondary sex ratios produced by sires at hatching are still evident as biased tertiary sex ratios (measured as the sex ratio of their progeny that survived to adulthood), then the relative production of adult sons and daughters may indeed reflect a good-genes effect, and predict viability of adult offspring (Moore, 1994; Jennions & Petrie, 2000). If true, it remains unclear why viability *per se* was not heritable in this study, although one likely contributing factor is that dichotomizing survival as 0/1 over a particular interval may fail to capture much of the subtle variation in the genetic basis of viability. In contrast to our previous studies (Calsbeek & Bonneaud, 2008; Cox & Calsbeek, 2010a), we did not find any evidence that sire size influences components of progeny fitness; neither RS^V nor survival of progeny was related to sire body size. This may be in part due to the fact that our previous measures of progeny survival were conducted at the juvenile stage, whereas the present study focused on adult survival and reproductive success. Another possibility is that the error associated with our measures of viability, at both the individual level (live/die) and the family level (family size range 1–9 progeny; average family size = 3.1 progeny), complicated our estimates of heritability and

Table 4 Progeny viability varied as a function of the number of male and female offspring produced by sires but not dams. Viability of male offspring increased with the number of sons produced by sires, whereas viability of female offspring increased with the number of daughters produced by sires. By contrast, progeny viability was not correlated with the number of sons or daughters produced by dams. RS^V for progeny of both sexes increased with the number of sons produced by sires but was not correlated with any other measure of parental sex allocation. Results are from generalized linear models with logit link functions to account for the binominal distribution of survival data (live/die) and log links to account for the Poisson distribution of fitness. Models include year as a factor and offspring mass as a covariate. Significant results are highlighted in bold.

	Progeny viability		Progeny RS^V	
	χ^2	*P*-value	χ^2	*P*-value
Daughters from sires				
# of sons	0.03	0.99	0.01	0.84
# of daughters	4.03	0.06	0.03	0.28
Offspring mass	1.12	0.29	1.41	0.23
Year	1.89	0.38	42.19	0.0001
Sons from sires				
# of sons	**8.01**	**0.01**	**5.62**	**0.04**
# of daughters	0.12	0.79	**13.01**	**0.003**
Offspring mass	4.2	0.04	**7.87**	**0.005**
Year	13.25	0.001	**21.37**	**0.0001**
Daughters from dams				
# of sons	0	0.98	0.8	0.37
# of daughters	0.25	0.62	0.38	0.54
Offspring mass	0.9	0.34	**3.87**	**0.05**
Year	2.55	0.27	**18.19**	**0.0001**
Sons from dams				
# of sons	0.13	0.72	0.85	0.36
# of daughters	0.64	0.42	0.19	0.66
Offspring mass	0.49	0.48	**4.12**	**0.04**
Year	3.53	0.17	**22.01**	**0.0001**

between-sex genetic correlations. Future studies should aim to resolve whether genetic variation in viability may also be partitioned between juvenile and adult stages (Chippindale *et al.*, 2001).

Additional evidence that the production of adult sons is driven by a good-genes process comes from the positive correlation between number of mature sons produced by sires and the RS^V of his male and female progeny. That is, sires that produced more adult sons also produced progeny who themselves produced more offspring that survived to sexual maturity. The lack of sex-specificity in this result is consistent with the interpretation that fitness accounted for by RS^V is at least partially independent of adult viability *per se* (Promislow *et al.*, 1998; Mallet *et al.*, 2012). For example, sires that produced more sons also produced daughters with high RS^V, despite those daughters also having low adult survival. In other words, whereas the genetic architecture underlying viability appears to reflect an adaptive response to sexually antagonistic selection (Bonduriansky, 2007), that of RS^V does not, highlighting the likelihood that sexual conflict has been resolved for much of the fitness variation in this population (Kokko *et al.*, 2002).

All else being equal, consistent directional selection is expected to deplete genetic variance by eliminating low-quality phenotypes and genotypes (Fisher, 1958; Charlesworth, 1987). It is therefore paradoxical that natural populations should continue to harbour genetic variation in fitness, as fitness should always be under positive directional selection (Mousseau & Roff, 1987). Although several studies (Chippindale *et al.*, 2001; Brommer *et al.*, 2007; Foerster *et al.*, 2007) have shown that components of fitness (i.e. survival, mating success, fecundity) have relatively lower heritability than, for example, morphological traits, it remains unclear what processes maintain variation in fitness in the wild (Kruuk *et al.*, 2001). It is well understood that genetic variation in fitness is maintained by mutation (Kimura, 1983), gene flow (Slatkin, 1985; Gomulkiewicz *et al.*, 2000), fluctuating environments (Grant & Grant, 2002) and changing selection pressures (Trotter & Spencer, 2008). Sexual conflict has recently been proposed as having an important role as well (Arnqvist & Rowe, 2002; Patten *et al.*, 2010). If sexually antagonistic selection favours different alleles in males and females, then it effectively acts as a form of balancing selection that can maintain genetic variation in phenotypic traits and in fitness itself. Despite the previous suggestion of sexual conflict in our study system, our direct test found no evidence for negative genetic correlations in components of fitness between males and females, which would be required to maintain overall genetic variation in fitness. However, even in the absence of a negative between-sex genetic correlation for fitness, biased sex allocation could still be adaptive if the genetic correlation for fitness is < 1. In such a case, the adaptive benefits of sex-ratio bias could be viewed as accruing from the preferential production of the 'better' sex for a given parental genotype, rather than as a solution to unresolved sexual conflict *per se*.

Acknowledgments

We thank Nancy Bottomley for permission to work on her land. The US National Science Foundation (DEB 0816862), the National Geographic Society (#7739-04), a CAPES fellowship to DG (SwB, 13442/13-9) and Dartmouth College provided financial support. This research was approved by Animal Care and Use Committees at the University of California, Los Angeles (protocol 2004-47-04), and Dartmouth College (protocol 07-02-03) and conducted under permits from the Bahamas Ministry of Agriculture and BEST Commission. The authors have no conflict of interests to declare.

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Received 13 September 2014; revised 25 June 2015; accepted 29 July 2015