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The consequences of sexual selection in well-adapted and maladapted populations of bean beetles†

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Whether sexual selection generally promotes or impedes population persistence remains an open question. Intralocus sexual conflict (IaSC) can render sexual selection in males detrimental to the population by increasing the frequency of alleles with positive effects on male reproductive success but negative effects on female fecundity. Recent modeling based on fitness landscape theory, however, indicates that the relative impact of IaSC may be reduced in maladapted populations and that sexual selection therefore might promote adaptation when it is most needed. Here, we test this prediction using bean beetles that had undergone 80 generations of experimental evolution on two alternative host plants. We isolated and assessed the effect of maladaptation on sex-specific strengths of selection and IaSC by cross-rearing the two experimental evolution regimes on the alternative hosts and estimating within-population genetic (co)variance for fitness in males and females. Two key predictions were upheld: males generally experienced stronger selection compared to females and maladaptation increased selection in females. However, maladaptation consistently decreased male-bias in the strength of selection and IaSC was not reduced in maladapted populations. These findings imply that sexual selection can be disrupted in stressful environmental conditions, thus reducing one of the potential benefits of sexual reproduction in maladapted populations.

KEY WORDS: **Adaptation, environmental change, fitness landscape, genetic variance, sexual conflict, sexual selection.**

Predicting the fate of populations exposed to changing environmental conditions is a major contemporary challenge facing biologists (Chevin et al. 2010; Hoffmann and Sgrò 2011; Garcia-Gonzalez et al. 2012; Walters et al. 2012). Among the many factors that can contribute to evolutionary rescue, the role of sexual selection is hotly debated (reviewed by: Candolin and Heuschele 2008; Whitlock and Agrawal 2009; Miller and Svensson 2014). Theory predicts that sexual selection can provide population-level benefits if it weeds out males of low genetic quality, and by doing so, removes mutations with generally deleterious effects from the population (Zahavi 1975; Rowe and Houle 1996; Lorch et al. 2003; Tomkins and Radwan 2004). Consequently, sexual selection could increase the rate of adaptation at a low demographic cost (Manning 1984; Agrawal 2001; Siller 2001), because females–whose reproductive rate ultimately limits population growth–would experience weaker selection and not suffer the costs of adaptation (*sensu* Haldane 1957).

Sexual selection is generally expected to be stronger in males than in females in polygamous species (Bateman 1948; Robert 1972; Clutton-Brock and Parker 1992; Andersson 1994; Arnqvist

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and Rowe 2005; Janicke et al. 2016), suggesting that this benefit of sexual selection could indeed be significant in natural populations. However, the necessary assumption that sexual selection in males is aligned with natural selection has been challenged by a number of recent studies. These show that sexual selection often can cause the evolutionary interests of males and females to diverge to a point where selection in males favors alleles that are detrimental when expressed in females (reviewed in: Bonduriansky and Chenoweth 2009; Rice and Gavrilets 2014). Such Intralocus Sexual Conflict (**IaSC**: Chippindale and Rice 2001) may effectively cancel out, or even reverse, any potential population-level benefits of sexual selection.

This negative impact could be further magnified by sexual selection for male strategies that directly inflict physical harm on females during mating interactions (Arnqvist and Rowe 2005; Rankin et al. 2011; Takahashi et al. 2014; Chenoweth et al. 2015; Berger et al. 2016). These diverse effects of sexual selection are reflected in the many idiosyncratic results reported in experimental evolution studies that typically investigate the net outcome of all these components (Whitlock and Agrawal 2009). In this article, we suggest that deeper insights into the impact of mating system variation on rates of adaptation can be gained by studying these components in isolation. Our approach is motivated by recent theoretical (Connallon and Clark 2012, 2014; Connallon 2015) and empirical (Long et al. 2012; Plesnar-Bielak et al. 2012; Berger et al. 2014, but see: Delcourt 2009; Punzalan et al. 2014) evidence suggesting that the relative impediment on adaptation imposed by IaSC may be reduced in environments to which the populations are not adapted. This theory predicts that selection in the sexes should align, and IaSC should be reduced, if environmental change causes a similar displacement of male and female phenotypes from their new phenotypic optima (Lande 1980; Connallon and Clark 2014). In Figure 1 we illustrate how this displacement from the fitness peak is expected to elevate and redistribute the standing genetic variation for fitness in maladapted populations, increasing the potential for sexual selection in males to aid adaptation from standing genetic variation.

We tested these predictions by estimating and comparing three quantities across populations of bean beetle that were experimentally manipulated to be well-adapted or maladapted to their environment. First, we assessed the upper limit for the strength of selection in each sex by estimating the mean standardized variance in fitness, $\boldsymbol{I}$, known as "the opportunity for selection" (Crow 1958):

$$I = \frac{V\omega}{\bar{\omega}^2} \quad (1)$$

where $V\omega$ is the variance in fitness and $\bar{\omega}$ mean fitness. $\boldsymbol{I}$'s additive genetic component $\boldsymbol{I}_A$ (Houle 1992) predicts the response to selection of fitness itself (Fisher 1930; Price 1972). Sex-specific estimates of $\boldsymbol{I}$ and its subsequent partitioning into genetic and environmental components therefore represent an empirically tractable means to explore the relative strength of, and response to, selection in males and females (Shuster and Wade 2003; Krakauer and Webster 2011). Thus, we compared whether (i) $\boldsymbol{I}$ was generally greater in males than in females (as often predicted for polygamous species), (ii) $\boldsymbol{I}$ was greater in maladapted compared to well-adapted populations (as predicted by a simple Gaussian fitness landscape; Fig. 1), and (iii) the relative strength of selection in the sexes ($\boldsymbol{I}_M/\boldsymbol{I}_F$) was contingent upon the level of maladaptation.

Second, the intensity of IaSC in a given population can be quantified by the intersexual genetic correlation (r_{MF}) for fitness:

$$rMF = \frac{\mathrm{COV}(M,F)}{\sqrt{I_{A^M} * I_{A^F}}} \quad (2)$$

where I_{AM} and I_{AF} are the genetic variances in relative fitness in males and females, respectively, and COV(M,F) is the intersexual genetic covariance. A positive r_{MF} indicates that a genotype with high male fitness also shows high female fitness and that most of the genetic variation in the population has sexually concordant fitness effects. On the other hand, a negative r_{MF} is the hallmark of pronounced IaSC and indicates that a genotype encoding high male fitness yields females with low fitness (and vice versa). Hence, we tested if the r_{MF} for fitness was more positive (or less negative) in maladapted compared to well-adapted populations (Fig. 1).

Finally, under the assumption that males do not provide parental care and contribute nothing more to females than unlimited numbers of sperm, population fitness will be limited by female fecundity. By further assuming a constant "environment" and negligible density regulation, (i.e., neglecting the second term in Fisher's theorem (Fisher 1930)), we can predict the rate of increase in population fitness using the Robertson-Price identity (Robertson 1966; Price 1970):

$$R_{Wpop} = \frac{1}{2}[I_{A^F} + \mathrm{COV}(M,F)] \quad (3)$$

where the covariance term accounts for the genetic response in relative fitness of females due to selection on shared allelic variation in males. This can be compared to the case for an asexual population where all individuals are females and the increase in population fitness is predicted simply by the genetic variance in relative fitness, I_{AF}. Thus, sexual selection in males will increase population fitness more than selection in females when $\mathrm{COV}(M,F) > I_{A^F}$.

To estimate these quantities across well-adapted and maladapted populations, we used replicated experimentally evolved populations of the bean beetle *Acanthoscelides obtectus* (see Fig. 2 for a graphical depiction of the experimental design). The

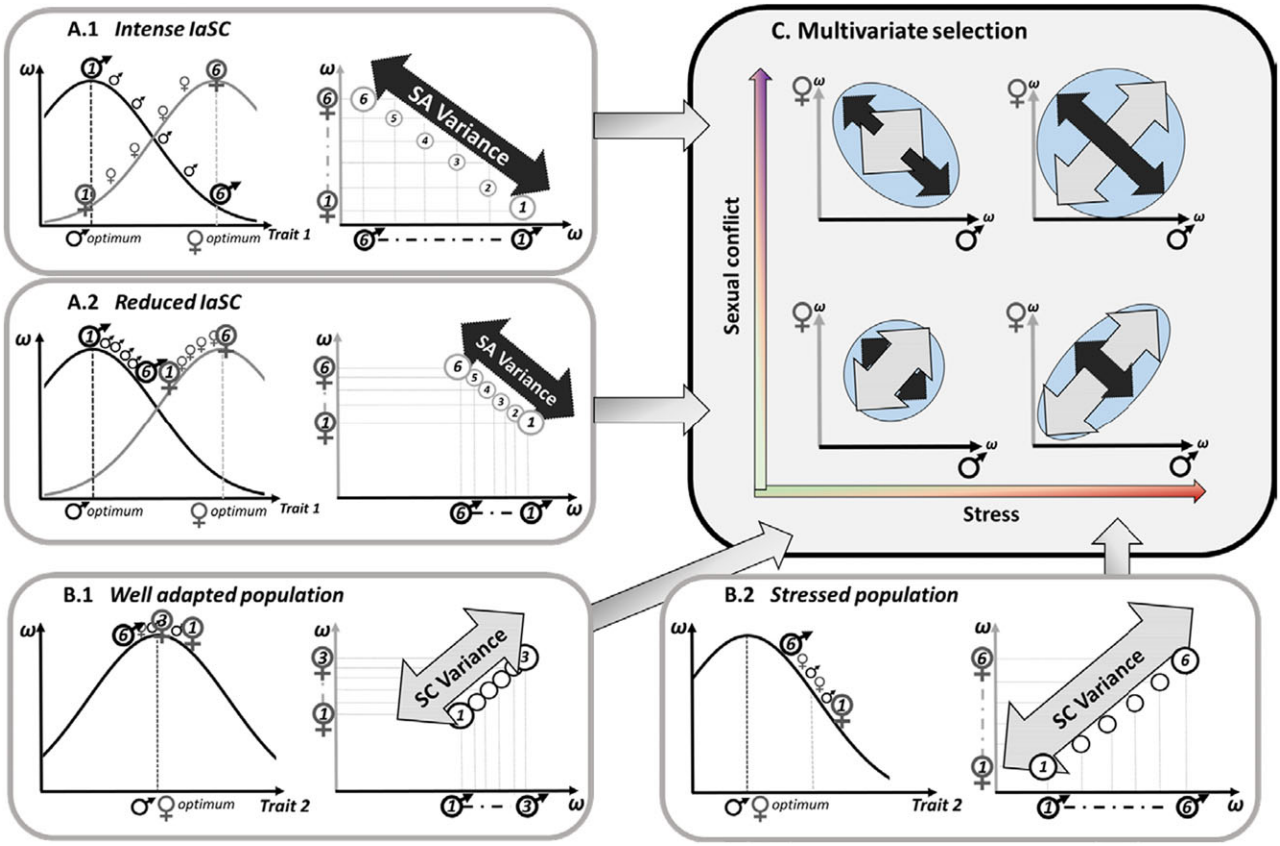

Figure 1. The effects of intralocus sexual conflict (IaSC) and population maladaptation on the r_{MF} for fitness.
In (A), males and females have distinct phenotypic optima for Trait 1 and selection acts in opposing directions in the sexes. Additive genetic variation for Trait 1 generates sexually antagonistic genetic variance in fitness (SA Variance) and the r_{MF} for fitness is negative. When trait expression is perfectly correlated between the sexes (i.e., a given genotype is expressed as the exact same phenotype in both sexes), conflict is maximal (A.1). However, by the evolution of increased sexual dimorphism in Trait 1, the intensity of sexual conflict can be reduced (A.2).
In (B), males and females share the same phenotypic optimum for Trait 2. As a result, additive genetic variation for Trait 2 has equal fitness consequences in males and females (sexually concordant, SC variance), contributing to a positive r_{MF}. An environmental change, causing a similar displacement of the sexes away from their new phenotypic optimum for Trait 2, will align selection in the sexes and increase the amount of SC variance (compare B.1 and B.2). Thus, the population-level benefit of sexual selection in males is predicted to scale inversely with how well the population is adapted to its environment.
In (C), the multivariate case including both traits is presented. Four extreme scenarios resulting in different intersexual genetic correlations and (co)variances are depicted, depending on the amount of IaSC (A.1 to A.2) and level of maladaptation (B.1 to B.2). For a given level of IaSC, environmental maladaptation will make the r_{MF} more positive by increasing sexually concordant genetic variation. Conversely, for a given level of environmental adaptation, increasing IaSC will make the r_{MF} more negative by increasing sexually antagonistic genetic variation.

populations used have been adapting for more than 80 generations to either of two different host plants; *Phaseouls vulgaris* (white bean) or *Cicer arietinum* (chickpea), henceforth referred to as the P and C evolution regime. Previous studies on effects of environmental stress on IaSC are inconclusive, finding significant reductions in IaSC (Long et al. 2012; Berger et al. 2014) as well as nonsignificant changes in IaSC (Delcourt et al. 2009; Punzalan et al. 2014; Holman and Jacomb 2017). However, these results are difficult to interpret, not only due to the inherently low statistical power of quantitative genetic experiments, but also because none of these studies controlled for the possibility that the amount of genetic (co)variation in fitness may differ across environments, irrespective of the level of adaptation to the environment (Hoffmann and Merilä 1999; Agrawal and Whitlock 2010). Here, in an attempt to remedy this predicament, we compare the sex-specific genetic variation for fitness in the two evolution regimes when cross-reared on the alternative hosts. This reciprocal experimental design rendered the benign host treatment of one evolution regime stressful to the other (Fig. 2), allowing us to isolate the effects of population maladaptation on genetic (co)variances and IaSC.

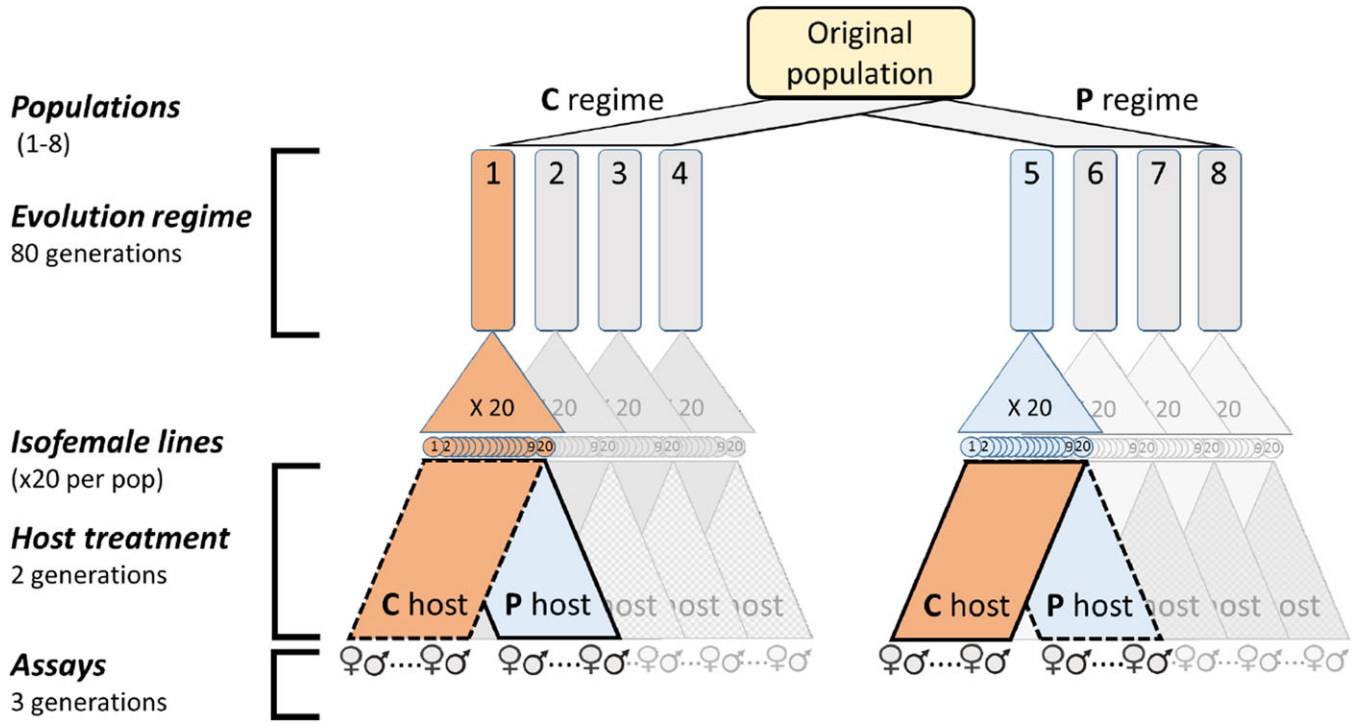

Figure 2. Experimental design including evolution regimes, host treatment and fitness assays.

Methods

STUDY POPULATION

The bean beetle *Acanthoscelides obtectus* is a pest of seed storages and uses the common bean (*P. vulgaris*, henceforth: the P-host) as a preferred host but can also develop on other host plants (Savković et al. 2016), including chickpea (*Cicer arietinum*, henceforth: the C-host). The development from egg to adult occurs inside the bean within approximatively 30 days. Adult beetles are facultatively aphagous; when they emerge out of the beans they are mature within a few hours and are able to mate and reproduce successfully without requiring water or other resources. Males usually chase females and attempt mounting; male aggressiveness and female choice are two factors that influence mating success (Stojković et al. 2014). The laboratory environment is in many aspects reflecting the seed storage environment that is colonized by natural populations of this species. Indeed, the populations used in this study originate from a founder population established from three grain storages in the region of Belgrade (Serbia) in 1983, and have been maintained on the P-host without adult food and water supply under the laboratory conditions of 30°C and 30–40% RH ever since (see Stojković et al. 2014, 2016).

EVOLUTION REGIMES AND ISOFEMALE LINES

Each evolution regime was replicated four times, using a founding population size of $N = 1000$ individuals for each replicate, eight years prior to the start of the experiments. The eight populations were maintained for 80 generations on their respective host inside climate chambers at 30°C and 30–40% RH, at a population size of 300 adults (further details can be found in and Savković et al. 2016). Importantly, previous studies have confirmed substantial local adaptation of these evolution regimes to their respective hosts; life histories are significantly diverged and estimates of fitness and population growth rates are highest for the P-regime when the two regimes are reared on the P-host, but highest for the C-regime when reared on the C-host (Savković et al. 2016). This local adaptation is also accompanied by differences in mating behaviors and the composition of cuticular hydrocarbons used in chemical communication during mating interactions (Stojković et al. 2014).

Three generations prior to the start of the quantitative genetic experiment, 50 isofemale lines were created from each of the eight populations by mating a single virgin female to a single virgin male. Out of these original lines, the 10 lines with what seemed to be lowest productivity were discarded before 20 lines of the remaining 40 per population (80 per evolution regime) were randomly selected to be used in the quantitative genetic breeding design (see Fig. 2). The lines were maintained at a population size of 50 individuals (excluding the individuals used in the assays) throughout the experiment. The isofemale line method allows the capture of genetic variation of the original population, while offering a means to rear genetic strains across

different environments in a replicated manner (David et al. 2005). Additive genetic variance is then estimated as twice the variance between isofemale lines (Hoffman and Parsons 1988), although this estimate is likely to include also some dominance and epistasis, which makes it broad-sense genetic variance (David et al. 2005). We note that our estimates of female LRS (Fig. S1) are very similar to previously published estimates of female fecundity in the outbred replicate populations (Savković et al. 2016), suggesting that inbreeding depression among the lines used in our experiment cannot have contributed to our results.

In addition, five generations prior to the quantitative genetic experiment, a reference population from each regime (henceforth, the C and P reference) was created by mixing more than 3000 individuals from the four replicate populations. The reference populations were maintained at a population size of approximately 1000 individuals on 600 grams of their respective host seed.

EXPERIMENTAL DESIGN

A graphical depiction of the experimental design can be found in Figure 2. Two generations before the start of the experiment, isofemale lines and the two reference populations were split into two copies that were moved to either the C- or P-host and maintained there for the rest of the experiment. Hence, P- and C-populations were assayed on both hosts. To estimate genetic (co)variances, males and females of each isofemale line and host treatment were assayed for their adult reproductive success (see detailed description below) by competing and mating them with same- and opposite sex individuals deriving from the reference population of the same origin. For each isofemale line, we performed at least 10 replicate assays on each host and for each sex. To acquire a sufficient sample size, the experiment was carried out during three consecutive generations. In total, 7212 assays were performed, evenly distributed across the two sexes and the four evolution regime × host treatment combinations.

ASSAYS OF ADULT LIFETIME REPRODUCTIVE SUCCESS

All beetles were virgin and 0–24 h old when used in the assays. An assay was set up by placing a focal male or female from an isofemale line inside a petri dish measuring 90 mm in diameter, together with two reference individuals of the opposite sex and a reference competitor of the same sex. The same-sex competitor was sterilized with gamma radiation (100Gy) prior to its introduction in the assay. The radiation causes lifelong sterility, which ensures that all adult offspring emerging from an assay can be attributed to the focal individual (confirmed in *A. obtectus* by a pilot experiment). Sterilized males compete actively for mating opportunities and their sperm should be able to fertilize eggs (while the resulting zygote is nonviable), such that both pre- and postcopulatory sexual selection was included in the male assays (as demonstrated repeatedly in the close relative *Callosobruchus maculatus*: e.g., Eady 1991; Maklakov and Arnqvist 2009; Grieshop et al. 2016; Martinossi-Allibert et al. 2017). Sterilized females are able to compete with the focal females over egg laying substrate (i.e., seeds) and potential mating opportunities (personal observations). Thus, even though males typically have a higher propensity to mate and will compete more over fertilization than females in this polygamous species, our design itself did not bias the opportunity for sexual selection in male and female assays. We note that the rearing density in our assays (four individuals in 90 mm petri dishes) is likely to be somewhat lower and sets a lower limit to the maximum number of mating partners compared to the standard culturing conditions (300 beetles in 1 liter bottles), but otherwise includes the same abiotic conditions. The four individuals were left in the petri dish with ad libitum supply of common beans or chickpeas (depending on host treatment) to mate and lay eggs for their entire lifetime at standard temperature and humidity inside climate chambers. Once all resulting offspring had emerged, the petri dish was frozen at –20°C for at least two days before the offspring were counted.

Statistical Analysis

MEAN LRS ACROSS HOSTS AND EVOLUTION REGIMES

To confirm that the alternative host represented a more stressful environment relative to the native host for each evolution regime, we analyzed differences in mean LRS across the male and female assays. In contrast to previous studies showing evidence of local adaptation in these populations (Savković et al. 2016), our estimates of LRS contain a component of soft selection in form of intraspecific competition, which could obscure the signal of local adaptation. This component of soft selection is likely to be pronounced in male assays because focal males compete with conspecific reference males, masking superior male performance on the native host. Hence, differences in mean LRS in the male assays are likely driven by differences in fecundity of the reference females. Our analyses used maximum likelihood estimates from linear-mixed effects models implemented in the lme4 package (Bates et al. 2011) for R (R Core Team 2013). The absolute number of offspring produced in the assays was analyzed in a general linear-mixed effects model assuming a Poisson distribution. The fixed effects of the model were sex, evolution regime, and host treatment, as well as their interactions. Population identity crossed by host treatment and sex, and date of the assay, were added as random effects. We also added a host- and sex-specific individual (observation)—level effect to correct for overdispersion of the data.

VARIANCES IN RELATIVE FITNESS, COVARIANCES, AND CORRELATIONS

We estimated sex- and environment-specific opportunities for selection (***I***) and genetic (co)variances in LRS in mixed effects models using both ML estimation in the lme4 package and Bayesian estimation utilizing Markov Chain Monte Carlo (MCMC) simulations implemented in the MCMCglmm package (Hadfield 2010) for R. Separate models were run for each experimental evolution regime (i.e., for C and P- regimes separately). The response variable was relative fitness (LRS_i / $\overline{LRS}$), calculated separately for each combination of sex, evolution regime, and host treatment, assuming normally distributed data. First, using ML estimation, we tested for differences between the replicate populations in sex- and host-specific genetic architecture by comparing the log-likelihood of models estimating these (co)variances globally across populations, or per population. We found that populations were not different and therefore continued by pooling populations in subsequent analyses (see *Results*). Similarly, we also used ML estimation to test for significant sex- and environment specificity in genetic architecture comparing models with and without these effects incorporated (Table S3).

ML and Bayesian models had the same specification and built except that the MCMCglmm package allowed increased flexibility in specifying the structure of the residual variances. This was important as estimating sex- and host-specific opportunities for selection was central to our study. Moreover, the MCMC resampling allowed us to calculate 95% credible intervals for all comparisons and estimates based on the posterior distributions. As standardizing the data by the mean prior to running statistical analysis could lead to the underestimation of confidence intervals for variance estimates, we also analyzed nonmean-standardized data and found no difference in the results that we describe in this study. Isofemale line (co)variance was partitioned by sex and host treatment and a full variance-covariance matrix was estimated for the isofemale line effect with MCMCglmm using the "us" structure and an unbiased parameter expanded prior for the covariance matrices (Hadfield 2012). Residual variance was estimated per sex, evolution regime, and host treatment using the "idh" structure. In addition, we included date as a random effect. The main effect of population and its interactions with sex and host treatment was added as a fixed effect, certifying that population differences did not contribute to our estimates of within-population genetic (co)variance and allowing mean LRS to vary between populations and across the different treatments. MCMCglmm estimates were also used to calculate log-ratios of variances (phenotypic and genetic) across host treatments and sexes, for each evolution regime. This allowed us to compare the change in selection imposed by a change of host across sexes and evolution regimes.

Results

RESPONSE TO EXPERIMENTAL EVOLUTION

The interaction between evolution regime and host treatment significantly affected LRS ($\chi^2 = 15.8$, df $= 1$, $P < 0.001$), confirming that populations had on average higher LRS if assayed on their native host (Fig. S1). This result was consistent across male and female assays (all interactions including sex: $P > 0.74$). Indeed, relative to when reared on their respective native hosts, fitness on the novel host decreased by 24% in the P-regime and 21% in the C-regime, averaged across sexes. There were also overall differences between sexes and evolution regimes in mean LRS. The C-regime had overall higher LRS than the P-regime, and females had higher LRS than males, on both hosts (Fig. S1). This was unexpected and we elaborate on this result in the discussion. The full summary of statistics is available in Table S2.

THE OPPORTUNITY FOR SELECTION, SEX-SPECIFIC GENETIC VARIANCES, AND THE LEVEL OF ADAPTATION

We first tested whether the four replicate populations within each evolution regime displayed significant differences in their sex- and host-specific genetic architecture. We did not find any such evidence (comparing models with estimation of sex- and environment-specific genetic (co)variance done globally or per populations; C-regime: $\chi^2 = 4.57$, df $= 12$, $P = 0.97$, P-regime: $\chi^2 = 8.20$, df $= 12$, $P = 0.77$). Admittedly, the power of these analyses was very low. However, population estimates of sex- and host-specific genetic variances and opportunities for selection were very consistent (Figs. S4 and S5), justifying pooling the populations in subsequent analyses.

Overall, significant sex-specific genetic variance in LRS was found in all evolution regime by assay environment combinations (Table S3). Males had significantly higher genetic and phenotypic variances across all combinations of evolution regimes and treatments (Fig. 3, Tables S6 and S7), suggesting that the strength of selection generally was greater in males compared to females. In females, there was more variance in fitness on the C-host compared to the P-host. However, this difference was greater for the P-regime (Fig. S8, Table S9). Thus, the increase in variance on the C-host was higher when populations were maladapted, consistent with predictions from the applied Gaussian fitness landscape (Fig. 1). Males however, showed the opposite response, with only the well-adapted C-regime having increased variance on the C-host. As a consequence of these sex-specific responses, the male bias in the opportunity for selection (quantified as the log-ratio of I_M/I_F) was significantly reduced in maladapted populations ($P_{MCMC} < 0.001$, Fig. 4), suggesting that sexual selection was weakened under host stress. Log-ratios of genetic variances behaved in a similar way (Table S9 and S10).

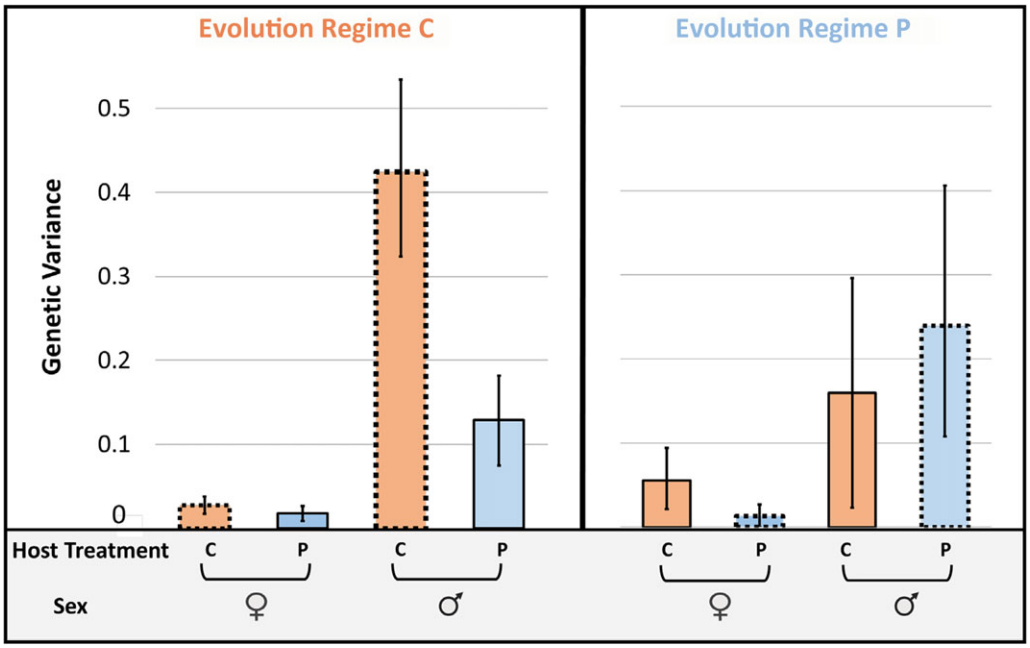

Figure 3. Genetic variance in relative LRS (I_A) for each sex, evolution regime and host treatment. Error bars represent 95% credible intervals from the Bayesian posterior distributions. Bars for well-adapted populations are delineated by dashed lines, and color represents the host treatment.

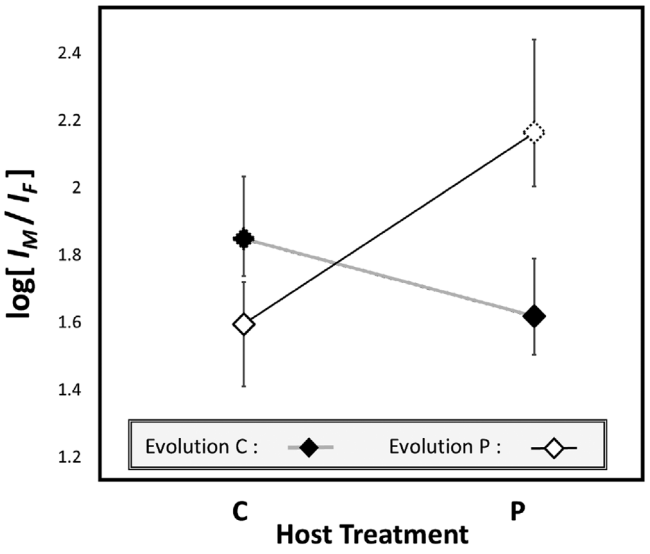

Figure 4. The opportunity for selection in the sexes in well-adapted and maladapted populations.
The relative difference in the opportunity for selection is quantified by a log-ratio of male over female variance in LRS (log[I_M / I_F]). A log-ratio of zero indicates no sex-difference in the opportunity for selection. Values above zero indicate greater opportunity for selection in males. The results imply that maladaptation consistently reduced the male-bias in the strength of selection.

r_{MF}s And The Level Of Adaptation

Contrary to predictions, the r_{MF} did not become more positive when populations were exposed to a novel host (Fig. 5). Limited statistical power may be an issue here, although we confirm that significant levels of sex-specific genetic variation were detected in all four host treatment/evolution regime combinations (all P-values < 0.022, Table S3). Moreover, we detected significant cross-host correlations in females from the C-regime (Fig. 5, left panel) and in males from the P-regime (Fig. 5, right panel). Moreover, the r_{MF} estimated across (well-adapted) C-populations raised on the C-host was positive and closest to achieving statistical significance of all four estimated r_{MF}s, opposite to theoretical predictions.

RELATIVE MAGNITUDE OF FEMALE GENETIC VARIANCE AND INTERSEXUAL GENETIC COVARIANCE FOR FITNESS

To assess the potential for selection on males to promote adaptation in females, we compared female genetic variance to the intersexual genetic covariance in LRS (eq. (3)). Intersexual genetic covariances were never significantly larger than female genetic variances (Fig. 6). In well-adapted populations, intersexual genetic covariances and female genetic variances were of similar magnitude. Moreover, in maladapted populations, female genetic variances even tended to be higher than intersexual genetic covariances (Fig. 6). This again was opposite to our predictions and suggests that the efficacy of sexual selection was reduced under environmental stress.

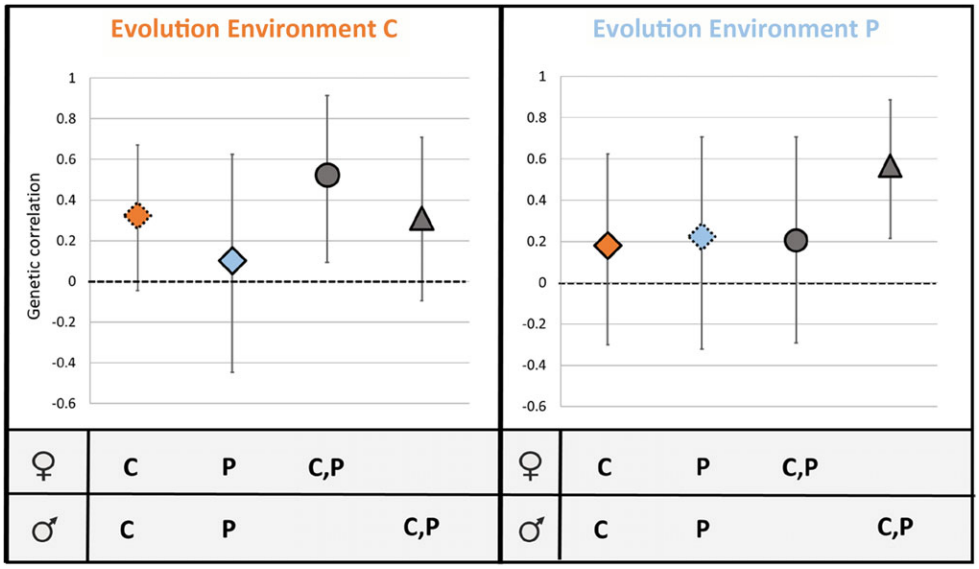

Figure 5. r_{MFS} and cross-environment genetic correlations for LRS.
r_{MFS} are given for host C (filled diamonds) and host P (open diamonds). Diamonds depicting r_{MFS} of adapted populations (i.e., when the host treatment matches the evolution regime) are designated by a dashed outline. Cross-host genetic correlations are depicted for males (triangles) and females (circles). Error bars represent a 95% credible intervals based on the Bayesian posterior distributions.

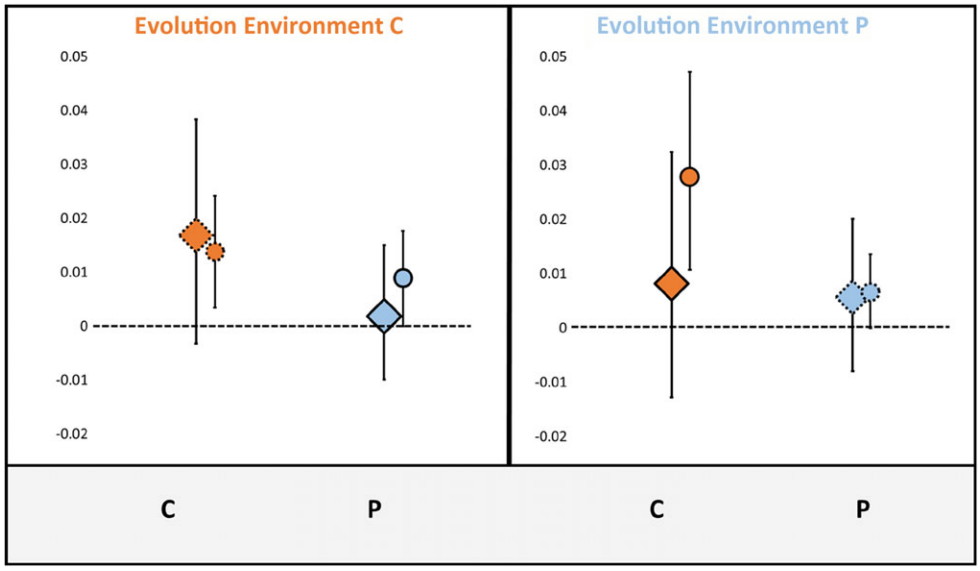

Figure 6. Female genetic variance compared to intersexual genetic covariance in relative LRS.
Genetic variances for female LRS are presented (circles), along with corresponding intersexual genetic covariances (diamonds) for each combination of evolution regime and host treatment. Symbols with dashed outlines indicate estimates in adapted populations (i.e., when the host treatment matches the evolution environment). Error bars represent a 95% credible intervals from the Bayesian posterior distributions.

Discussion

Sexual selection may promote adaptation by purging generally deleterious alleles through strong selection in males (Manning 1984; Agrawal 2001; Siller 2001), but sexual selection can also result in IaSC, rendering selection in one sex detrimental to the other (Bonduriansky and Chenoweth 2009; Cox and Calsbeek 2009). The negative impact of IaSC, however, is expected to be reduced in maladapted populations (Long et al. 2012; Berger et al. 2014; Connallon and Clark 2014, 2015; Connallon and Hall 2016). Here, we assessed these predictions in populations of bean beetles that had been experimentally manipulated to be either well-adapted or maladapted to their environment. We partitioned the effects of sexual selection on population fitness by quantifying

(i) sex-bias in the strengths of selection, and (ii) the general alignment of selection in the sexes. As predicted, we found that the opportunity for selection (***I***) and its genetic component ($\boldsymbol{I}_A$) was generally greater in males as compared to females. However, environmental stress affected variation in reproductive success very differently in the sexes, resulting in a general reduction of the male-bias in selection in maladapted populations. Moreover, we found no evidence suggesting that the alignment of selection between the sexes increased (and IaSC decreased) in maladapted populations. Below we discuss these results in more detail and further elaborate on the assumptions and application of fitness landscape theory, used here and previously, to predict adaptation in sexually reproducing populations.

Females consistently exhibited greater ***I*** and $\boldsymbol{I}_A$ when assayed on the C-host relative to the P-host, regardless of the evolution regime, which demonstrates a strong host-specific effect. However, the use of a reciprocal experimental design allowed us to disentangle and partition the effects of host and level of adaptation, and as predicted, maladaptation resulted in a relatively higher ***I*** and $\boldsymbol{I}_A$ in females. This result is consistent with predictions from a simple Gaussian fitness landscape. We further note that, while the overall higher variance in fitness on the C-host was not an explicit prediction from the Gaussian fitness landscape, this result suggests that the two host environments differed in the strength of phenotypic selection on females. Such dynamics can readily be incorporated in more complex models by relaxing the common assumption that the shape of the fitness surface (and hence the strength of stabilizing selection) remains constant across environments (reviewed in: Agrawal and Whitlock 2010).

Males, on the other hand, did not show patterns consistent with predictions; P-regime males showed no difference in ***I*** across hosts, and C-regime males showed greater ***I*** and $\boldsymbol{I}_A$ on their native C-host. Strikingly, these sex-specific responses to host stress resulted in the general male-bias in selection being significantly reduced in maladapted populations. The unexpected responses in males may ultimately be rooted in that, contrary to fitness components under hard selection such as female fecundity, sexual selection is frequency dependent by nature. The fertilization success of a given genotype is determined not only by abiotic environmental conditions but also by the frequencies of other male genotypes in the population (Ayala and Campbell 1974; Wolf et al. 2014). If genotype-environment interactions redistribute and change the relative frequencies of male breeding values for reproductive success (e.g., Wolf et al. 2014), the simple fitness landscape model presented in Figure 1 could thus be rendered inapplicable (Jones et al. 2012; Calsbeek et al. 2013). Frequency dependent sexual selection can, and has been, incorporated in more sophisticated landscape models (e.g., Lande 1980; Wolf et al. 2014), but it remains difficult to predict with certainty how such dynamics would respond to a change in the abiotic environment. In the present study, pre- and postcopulatory forms of male–male competition and female choice would all be plausible sources of frequency-dependent selection in males.

Additionally, an overall difference in the prevalence of female mate choice across environments may be partly responsible for the reduced strength of selection in males in maladapted populations, as suggested by previous empirical observations (Chaine and Lyon 2008; Gosden and Svensson 2008). For example, if male signals vary unpredictably with the environment, female choice for "good genes" may be disrupted in novel environments (Holman and Kokko 2014). As a consequence, both the direction and magnitude of sexual selection in males could change (Candolin and Heuschele 2008; Ingleby et al. 2010; Kolluru 2014). Another possibility is that female choice itself is condition dependent (Hunt et al. 2005; Cotton et al. 2006), resulting in reduced female choosiness and relaxed intersexual selection on males in maladapted populations. The bean beetle populations used in the present study have previously been assessed for mate preferences across the two hosts in a similar experimental set-up as employed here, and modifications of both female and male mating behavior were observed during the host shifts (Stojković et al. 2014). For example, males and females from the P-regime raised on the (to them) stressful C-host were less discriminant in mate choice (Stojković et al. 2014). This relaxation of precopulatory female choice may thus partly explain the observed weaker selection in maladapted P-regime males.

All r_{MF}s were low but positive, indicating that selection in one sex should, if anything, bring net benefits to the other. However, only one estimate was close to being significantly different from zero; that for the C-regime reared on the C-host, that is, in well-adapted populations. This was opposite to our expectation that r_{MF}s should be less positive in adapted populations (Long et al. 2012; Berger et al. 2014; Connallon and Clark 2014a; Connallon and Hall 2016). The prediction that environmental stress should reduce IaSC by aligning selection in the sexes relies on the assumption that the environmental change displaces males and females from their respective phenotypic optima in similar fashion (e.g., Lande 1980). While it seems likely that substantial environmental shifts will enforce sexually concordant selection on traits underlying local adaptation, picking up such effects in quantitative genetic designs relies on there being enough standing variation to leave a detectable signal on genetic covariances (see Fig. 1). We did, however, expect to find such a signal based on the previous work on these lines, showing that a range of life history (Savković et al. 2016), behavioral (Stojković et al. 2014), and chemical (Savković et al. 2012) traits are differentiated between the evolution regimes, implying a polygenic basis of local adaptation. Another possible explanation for the discrepancy between predictions and data is that the forces of sexually antagonistic selection also may be affected by environmental

change, as previously demonstrated in the closely related seed beetle *C. maculatus* (Berger et al. 2014). In fact, loci under sexually antagonistic selection could play an underappreciated role in adaptation to novel environments as they are predicted to contain high levels of polymorphism in functional phenotypes such as life-history traits (Bonduriansky and Chenoweth 2009; Berger et al. 2016; Radwan et al. 2016). Such scenarios have been treated implicitly in applications of fitness landscapes describing sex-specific selection (e.g., Connallon and Clark 2014; Connallon and Hall 2016). While these models generally predict that the alignment between male and female selection should decrease (and IaSC increase) over evolutionary time in the environment as the population approaches its new fitness peak (Lande 1980; Connallon and Clark 2014a,b), it is more difficult to predict how this alignment compares across environments when these vary in the prevalence of sex-specific selection. Indeed, this was a major motivation for the use of a reciprocal design that allowed us to separate the effect of evolutionary history and (mal)adaptation from environment-specific effects. Yet, our experiment did not detect any signal on r_{MF}s from either the host environment or the level of maladaptation.

We further tested the potential for selection on males to purge the fitness load on females by estimating the intersexual genetic covariances for LRS and comparing these to the variance in LRS measured in females. Although the genetic variance for LRS was greater in males, intersexual genetic covariances were never significantly greater than the genetic variance in females. Unsurprisingly given the previous discussion, the intersexual genetic covariances did not scale predictably with the level of (mal)adaptation. In fact, the intersexual genetic covariance instead tended to be greater than the female genetic variance in well-adapted populations. These findings echo the previously discussed discrepancies between the sexes in the effect of maladaptation on $\boldsymbol{I}_{\mathrm{A}}$, underlining that the net efficacy of sexual selection in purging mutation load on female fecundity seemed to be reduced in maladapted populations.

Our approximations of fitness variance utilizing measures of LRS do not account for the missing fraction of focal adults that were raised on the respective hosts but did not survive to be included in the assays of LRS. High juvenile mortality can reduce phenotypic variation in the surviving population, limiting the opportunity for sexual selection in adults. Indeed, the studied host-shifts are known to be associated with potentially substantial reductions in juvenile survival of *A. obtectus* (Savković et al. 2016). This effect of juvenile survival is perhaps an underappreciated factor determining the opportunity for sexual selection in stressful environments and can generally act to limit the purging of mutation load via sexual selection on males (Whitlock and Agrawal 2009; Martinossi-Allibert et al. 2017). Such an affect could have reduced the opportunity for adult selection in maladapted population in our experiment but it cannot have caused the observed sex-specific responses to host environment (unless juvenile survival itself is strongly sex and host specific).

A somewhat unexpected result in our study was the overall higher mean LRS of the C-regime (Fig. S1). Evolution on the C-host involves relaxed host discrimination during oviposition and increased fecundity (via increased body size) in C-regime females on both host plants. This also translates to increased fecundity on the P-host (Savković et al. 2016). P-regime females, on the other hand, lay fewer eggs in general, which may reflect a different life-history strategy under greater intraspecific larval competition on the P-host (Savković et al. 2016). Our assays provided host seeds ad libitum, which may have reduced larval competition and mortality relative to rearing conditions during experimental evolution. This in turn may have resulted in similar LRS of the C- and P-regime when assayed on the P-host. Additionally, our estimates of mean LRS will to some extent reflect soft selection as they contained an element of intraspecific adult competition between the focal individuals and the conspecific references. This element of the assays could have further obscured the signal of local adaptation between regimes. Nevertheless, the >20% drop in mean LRS on the novel host, which was evident in both regimes in our reciprocal design, reflects well the previous results showing evidence for local adaptation in form of crossing reaction norms for fitness components under hard selection in these lines (Savković et al. 2016).

Another unexpected result was that mean LRS in female assays was greater than that of male assays (Figure S1). Mean LRS in male assays must reflect the mean fecundity of the reference females they mate with. Hence, if reference females were of lower condition compared to focal females, this could have contributed to the sex-difference in mean LRS. Such a situation may have resulted from the somewhat higher larval rearing densities of the reference cultures compared to focal-rearing conditions, in combination with an overall greater resource requirement of the larger bodied females relative to their conspecific males. Another factor contributing to the sex-difference in mean LRS could be the consequences of using irradiated male competitors in the male assays. The seminal fluid of *A. obtectus* (as well as other seed beetles) contains gonadotropins that are potent female fecundity stimulants (Huignard 1983). Irradiation adversely affects these male accessory gland proteins in some fruit flies (Abraham et al. 2012) and it is possible that this also happened in our experiment. Given multiple mating by all individuals in the assays, this means that focal females in the female assays only mated with fertile (unirradiated) males and got on average one equivalent of male provided gonadotropins, while reference females in the male assays got on average less than one male equivalent by also mating with the irradiated male. This would have depressed mean female (and hence also mean male) LRS in the male assays.

Our study suggests that the effect of host maladaptation is to increase the strength of selection in female bean beetles, in line with predictions from a simple Gaussian fitness landscape. However, contrary to our expectations, the strength of selection on males did not increase in general, and IaSC was not reduced, under host stress. In sum, this resulted in an apparent reduction of the benefits of sexual selection in maladapted populations. Results from previous experimental evolution studies have been idiosyncratic regarding the role of sexual selection in adaptation (reviewed in: Whitlock and Agrawal 2009). This has led to the understanding that the net outcome of sexual selection is to a large extent determined by the prevalence of both intra- and interlocus sexual conflict (Arnqvist and Rowe 2005; Rice and Gavrilets 2014). Our study adds to the growing knowledge provided by these previous studies by presenting evidence suggesting that the efficacy of sexual selection can be reduced in maladapted populations, possibly even without changes in the extent of sexual conflict. The demonstrated sex-specific responses in the strength of selection may, if general, have hitherto underappreciated effects on the rate of adaptation in changing environments.

AUTHOR CONTRIBUTIONS

B.S., U.S., and M.Đ. created and maintained the long-term evolution regimes. I.M.A., U.S., B.S., G.A., and D.B. together conceived and planned the study. I.M.A. and U.S. collected the data. I.M.A. and D.B. analyzed the data and wrote the first draft of the manuscript. All authors contributed substantially to the final version.

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DATA ARCHIVING

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

S1. Mean LRS for each sex, host treatment and evolution regime.
S2. Anova table for a generalized linear mixed-effect model assuming a Poisson distribution showing the effect of sex, host treatment, evolution environment and their interactions on the absolute number of offspring (LRS).
S3. Test of the significance of sex-specific genetic variance for each evolution regime by host treatment combination.
S4. Genetic variance in relative LRS for each replicate population, given for each sex, evolution regime and host treatment.
S5. Phenotypic variance in relative LRS for each replicate population, given for each sex, evolution regime and host treatment.
S6. MCMCglmm estimates and 95% confidence intervals for genetic and phenotypic variance in LRS, as well as broad sense heritabilities for each sex, host treatment and evolution regime combinations.
S7. P-values for pairwise comparisons of MCMCglmm estimates of genetic and phenotypic variance in LRS.
S8. Log-ratio comparing the opportunity for selection across host treatment for each sex and evolution regime (C host over P host).
S9. Log-ratios of genetic and phenotypic variance in LRS comparing Host C/Host P.
S10. Log-ratios of genetic and phenotypic variance in LRS comparing Male/Female.