Sexual selection improves population fitness: a meta-analysis

Abstract

Sexual selection has manifold ecological and evolutionary consequences, making its net effect on population fitness difficult to predict from first principles. Instead, one can empirically determine whether sexual selection increases or decreases population fitness by manipulating the opportunity for sexual selection in replicate populations, allow evolution to proceed, and then comparing the evolved average fitness between the treatment groups. Here, we synthesise 459 effect sizes from 65 pertinent experimental evolution studies using meta-analysis. We find that sexual selection tends to elevate the mean and reduce the variance for many fitness traits, especially in females, and for populations evolving under stressful conditions. We conclude that the beneficial population-level consequences of sexual selection typically outweigh the harmful ones, and that the effects of sexual selection on total selection, genetic variance, and/or evolvability can differ markedly between sexes and environments. We discuss the implications of these results for conservation and evolutionary biology.

Keywords: Adaptation, Gender load, Monogamy, Mutation load, Sexual conflict.

Word count: XXXX in main text.

Sexual selection, defined as selection resulting from competition for mates or their gametes[1], is a ubiquitous evolutionary force that has profoundly shaped the natural world. As far back as Darwin[2], researchers have theorised that sexual selection can change the average absolute fitness of individuals in a population (henceforth termed 'population fitness'[3,4]). However, opinion is divided over whether the net effect on population fitness is positive or negative[5–8]. *Prima facie*, one might predict that sexual selection would have no effect on population fitness, since it does not matter which individuals of the faster-reproducing sex (typically males) succeed in mating, so long as some do[9]. However, when genotypes with high mating or fertilisation success also have superior breeding values for traits that affect population fitness (e.g. survival, parental care, female fecundity, or success in interspecific competition), sexual selection is predicted to elevate population fitness by causing a correlated response in these other traits[8]. In essence, the demographically-limiting sex (typically females) benefits from a gene pool that has been purged of harmful alleles through sexual selection on the non-limiting sex (typically males). Theoretically, the benefit to population fitness might could be large[10–12].

Conversely, sexual selection can decrease population fitness if male sexually-selected traits are negatively genetically correlated with female fitness, producing intralocus sexual conflict[13–16]. Additionally, sexual selection frequently favours phenotypes that reduce population fitness but benefit the individuals expressing them, such as harassment or infanticide by mate-seeking males[17–19], as well as investment in costly sexual signals and weaponry at the expense of parental care (*inter*locus sexual conflict)[20–22]. Given these conflicting theoretical expectations and empirical results, it remains unclear whether sexual selection tends to have a net benefit or cost to population fitness[5–8].

Researchers have investigated the population-level consequences of sexual selection using a range of approaches including macro-evolutionary studies[23–25], analysis of the fossil record[26], quantitative genetics[13,27–30] and especially experimental evolution. In particular, many experimental evolution studies have manipulated the intensity of sexual selection in captive populations, allowed evolution to proceed, and then measured population fitness components such as lifespan, fecundity, population extinction rate and mutation load. This approach facilitates direct measurement of the net effect of sexual selection on population fitness, at least in the specific populations and ecological conditions under study.

A number of factors might influence the strength and sign of the correlation between sexual selection and population fitness. First, the genetic correlation between female fecundity and male mating/fertilisation success varies in sign and magnitude between species[31] and even between conspecific populations[32], implying that sexual selection on males increases mean female fitness in some species and populations but not others. These inconsistencies could derive from differences in allele frequencies, or environmental differences that alter how genotype relates to phenotype and fitness. Second, it has been hypothesised that populations should display a more positive genetic correlation between male and female fitness – and thus potentially between mating/fertilisation success and population fitness – in novel or fluctuating environments, relative to stable environments[13,30,33,34]. This is because stable environments create consistent selection, preferentially eroding genetic variation at sexually-concordant loci (i.e. loci where the fittest genotype is the same in both sexes) and leaving behind variation at sexually antagonistic loci. We know of no systematic reviews testing this latter theory, though it is has motivated several recent empirical tests (e.g.[13,27,29]), and is relevant to conservation genetics.

Here, we synthesise the empirical literature on sexual selection and population fitness using formal meta-analysis. We focus exclusively on experimental evolution studies that manipulated the presence or strength of sexual selection, and then measured some fitness component, since these provide a particularly strong test

of the hypothesis that sexual selection affects the average fitness of populations. Our principle aims were to measure the average net effect of sexual selection on the population-level mean and variance for various fitness components, to test whether this effect varies between stressful and benign environments, and identify key moderators of the effect size of sexual selection treatment.

Results

The effect size dataset

We retrieved 459 effect sizes from 65 studies. 94 effect sizes were collected from populations evolving under stressful conditions, while 335 were measured for those evolving in benign conditions. 189 of the effect sizes came from measurements made on males, 219 on females, and the remaining 51 from measurements of a mixed-sex sample of individuals. Most effect sizes in our dataset came from studies that manipulated sexual selection by completely removing it in one treatment via enforced random monogamy ($n = 241$); other effect sizes ($n = 218$) derived from alternative manipulations, such as changing the adult sex ratio. In total, we obtained effect sizes for 20 different fitness traits, with reproductive success ($n = 156$) and offspring viability ($n = 56$) being the most commonly-measured traits. We classified 216 effect sizes as direct measures of fitness, 147 as indirect, and the remaining 96 effect sizes as ambiguous (see Methods, Fig. 1 and Table S1). Specifically, direct measures were components of reproduction or long-term viability (*e.g.*, reproductive success, offspring viability and extinction rate), indirect measures were traits often used as a proxy of fitness but that do not directly measure aspects of success in reproduction or population viability (*e.g.*, lifespan, mating success and ejaculate quality/production); and ambiguous measures were those reported to have an unclear or variable association with fitness (*e.g.*, body size, mating duration and early fecundity). Tables S4-S5 give a detailed description of our dataset.

Sexual selection is associated with higher mean values for most fitness components

The grand mean for all effect sizes was positive (REML $\beta = 0.23$, 95% CIs: 0.01 to 0.44, $p = 0.038$; Bayesian $\beta = 0.23$, 95% CIs: -0.06 to 0.51, $BF_{>0} = 18$), indicating that sexual selection typically had a net positive effect on the majority of populations and fitness components so far studied. Moreover, the effect sizes associated with the manipulation of sexual selection varied between different fitness traits. Sexual selection had a beneficial effect on most fitness traits, including all of the fitness traits that we classified as being 'direct' measurements of population fitness: extinction rate (REML $\beta = 0.37$, 95% CIs: 0.01 to 0.73; Bayesian $\beta = 0.37$, 95% CIs: -0.02 to 0.79, $BF_{>0} = 30.13$; $n = 4$), offspring viability (REML $\beta = 0.20$, 95% CIs: 0.042 to 0.36; Bayesian $\beta = 0.21$, 95% CIs: -0.04 to 0.47, $BF_{>0} = 24.89$; $n = 56$) and reproductive success (REML $\beta = 0.17$, 95% CIs: 0.013 to 0.32; Bayesian $\beta = 0.18$, 95% CIs: -0.06 to 0.44, $BF_{>0} = 15.53$; $n = 156$; Fig. 1; Table S6). Sexual selection significantly reduced two fitness components, namely immunity (REML $\beta =$ -0.49, 95% CIs: -0.72 to -0.26; Bayesian $\beta = -0.49$, 95% CIs: -0.78 to -0.19, $BF_{>0} = 0.0054$; $n = 35$) and body condition (REML $\beta = -1.3$, 95% CIs: -1.9 to -0.67; Bayesian $\beta = -1.3$, 95% CIs: -1.9 to -0.63, $BF_{>0} =$ 0.00025, $n = 1$). Fig. 1 presents Bayesian estimates for each fitness component and Table S6 contains both Bayesian and REML estimates, with sample sizes and test statistics.

The roles of environmental stress and sex

We found that the sex of the individuals measured (male, female, or a mixture), and the conditions under which the population evolved (stressful or benign) interacted to affect fitness (Table 1, 2). Sexual selection significantly improved female fitness, and the beneficial effect of sexual selection was significantly stronger for females from populations evolving under stressful conditions (e.g. a food source to which they were not well-adapted) than under benign conditions (Fig. 2a, Table 2). Sexual selection had a positive but non-significant effect on male fitness, and in contrast to females, fitness benefits were significantly weaker in stressful than benign environments (Fig. 2a, Table 2). Consistent with the different consequences of sexual selection for female and male fitness, the mean effect size in mixed-sex samples was non-significantly positive, and there was no significant difference between benign versus stressful conditions (Fig. 2a, Table 2). Overall, our results indicate that the positive effect of sexual selection on fitness is greater for females than males, and the difference between the sexes is magnified in stressful environments.

Other moderator variables that we examined had minimal impacts on effect size (Fig. S2 and Table S7). Specifically, effect size did not depend on whether or not the study was conducted blind (Fig. S9), nor on the number of generations for which the experimental evolution study was run (Fig. S10 and Fig. S11).

The effect size estimates we recovered were highly heterogeneous (I^2 = 94.9%, 95% CIs: 93-96%), reflecting the large differences in experimental procedures, study species, and fitness components included in our meta-analysis[35]. Most of the observed heterogeneity stemmed from between-study differences (I^2_{study} = 61.5%, 95% CIs: 42.9-78.9%), with variation between fitness components accounting for comparatively less heterogeneity ($I^2_{fitness\ components}$ = 33.5%, 95% CIs: 15.8-52.7%). The effect of taxon varied between REML and Bayesian models: the REML model suggested no effect (I^2_{taxon} = 0), while the Bayesian model implied a non-zero effect (σ^2 = 0.145 [0.006-0.517]; Table S10). Variation among taxa is explored further in the Supplementary Material, (Figure S3 and Table S8).

Sexual selection reduces phenotypic variance, for female traits in stressful environments

By applying meta-analysis to log coefficient of variation ratios (*lnCVRs*)[36], we found evidence that sexual selection reduces phenotypic variation under certain conditions (Fig. 2b). Specifically, phenotypic variance was significantly reduced by sexual selection for fitness components measured in females under stressful conditions ($lnCVR$ = -1.0, 95% CIs: -1.4 to -0.69). By contrast, we found no significant effect of sexual selection on phenotypic variance in males, or for either sex under benign conditions (Fig. 2b; Tables S13-S16). However, as expected from the results in females, there was a non-significant trend for a reduction in phenotypic variance in mixed-sex samples measured under stressful conditions ($lnCVR$ = -0.44, 95% CIs: -0.80 to -0.081; Fig. 2b).

As in the meta-analysis of trait means, there was high heterogeneity in the estimates of $lnCVR$ (I^2 = 95.4%, 95% CIs: 93-97%). Heterogeneity in the dataset was due to variability between studies (I^2_{study} = 40.1 %, 95% CIs: 23.5 % to 60.2 %), fitness components ($I^2_{fitness\ components}$ = 38.7%, 95% CIs: 17.1-60.7%) and taxon (I^2_{taxon} = 16.6, 95% CIs: 3.1-37.2%).

Publication bias

The funnel plot of effect sizes was asymmetrical, suggesting that some publication bias might be present (Fig. 3a; Egger's test: $z = 6.2$, $p < 0.0001$). Specifically, there was a moderate excess of low-powered studies in which sexual selection had a more positive effect on the fitness component than average, implying that low-powered studies are more likely to be published if they report statistically significant fitness benefits of sexual selection. There was no significant relationship between effect size and journal impact factor (Fig. 3b; $t_{437} = 1.2$, $p = 0.25$) or year of publication (Fig. 3c; $t_{437} = -1.0$, $p = 0.31$); thus, we found no evidence that effect size dictates the likelihood of publication in high-profile journals, or that effect sizes have diminished as the field has matured[37,38].

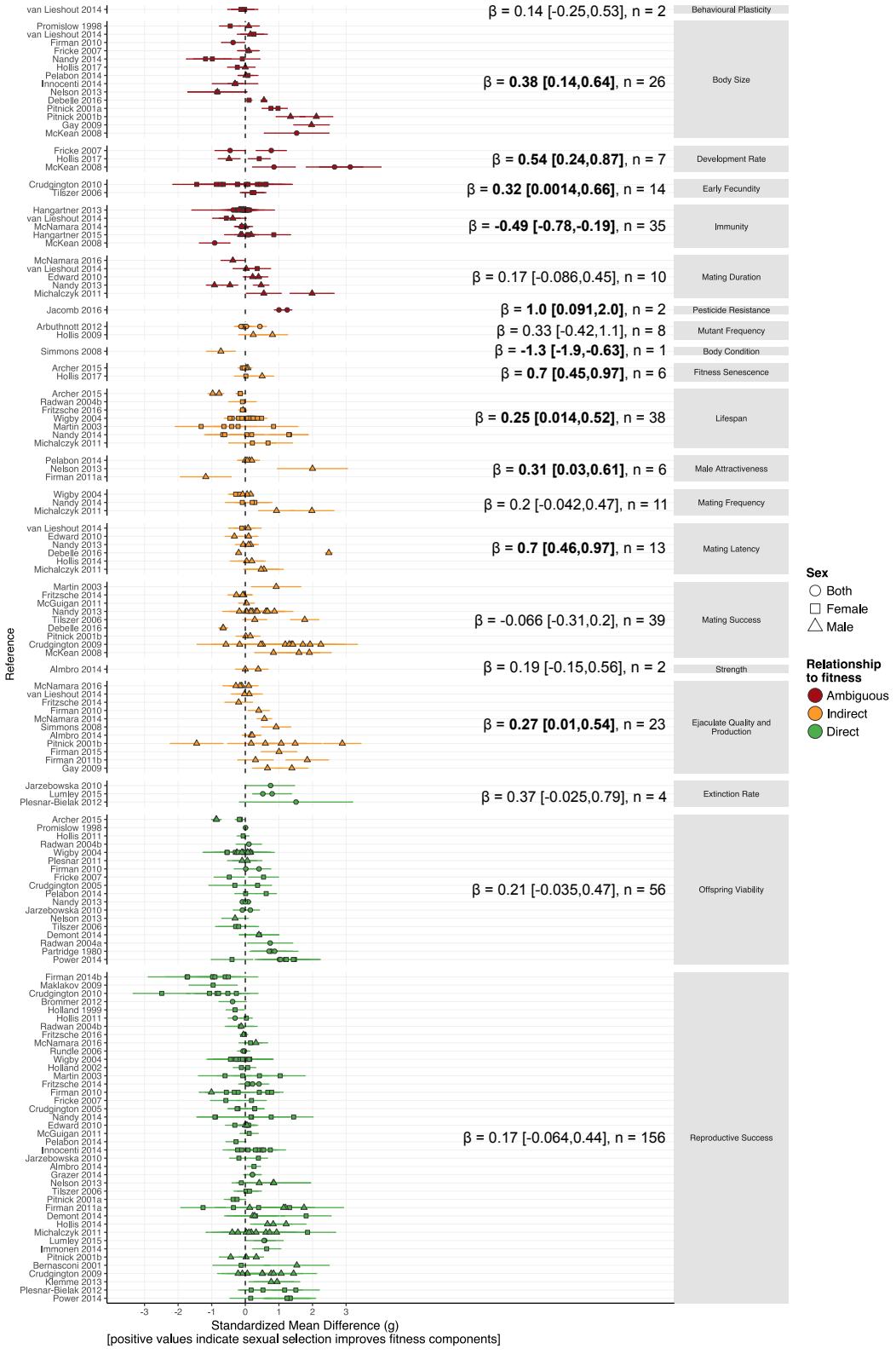

Figure 1: Forest plot showing the effect of manipulation of sexual selection, for 20 fitness components from 65 studies. Effect sizes (n = 459) varied between fitness components, and were more often positive than negative. Predicted average values are presented alongside each fitness component; those in bold have 95% confidence intervals that do not cross zero. The estimates presented here are Bayesian predictions, which were slightly more conservative than the REML predictions (Table S6).

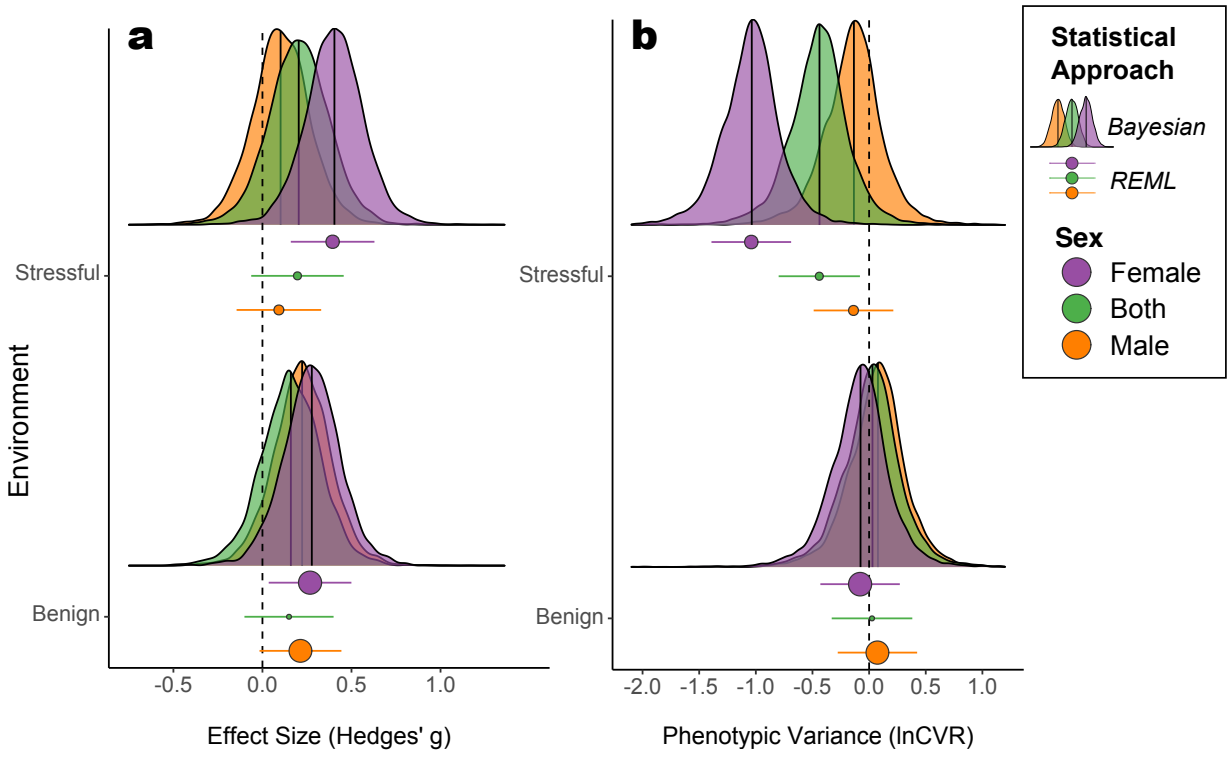

Figure 2: (**a**) Sexual selection tends to increase the population mean values of fitness traits, especially for female traits and for populations living under stressful conditions. (**b**) Under stressful conditions, sexual selection tends to reduce the phenotypic variance in fitness traits, especially for traits measured in females or mixed-sex individuals. The points with error bars show the mean effect sizes and their 95% CIs, determined from a meta-regression fit using restricted maximum likelihood (REML); the point sizes are proportional to the number of effect sizes (see Tables S12 and S14). Results from Bayesian meta-regression are shown as posterior prediction density curves, with vertical lines indicating the median.

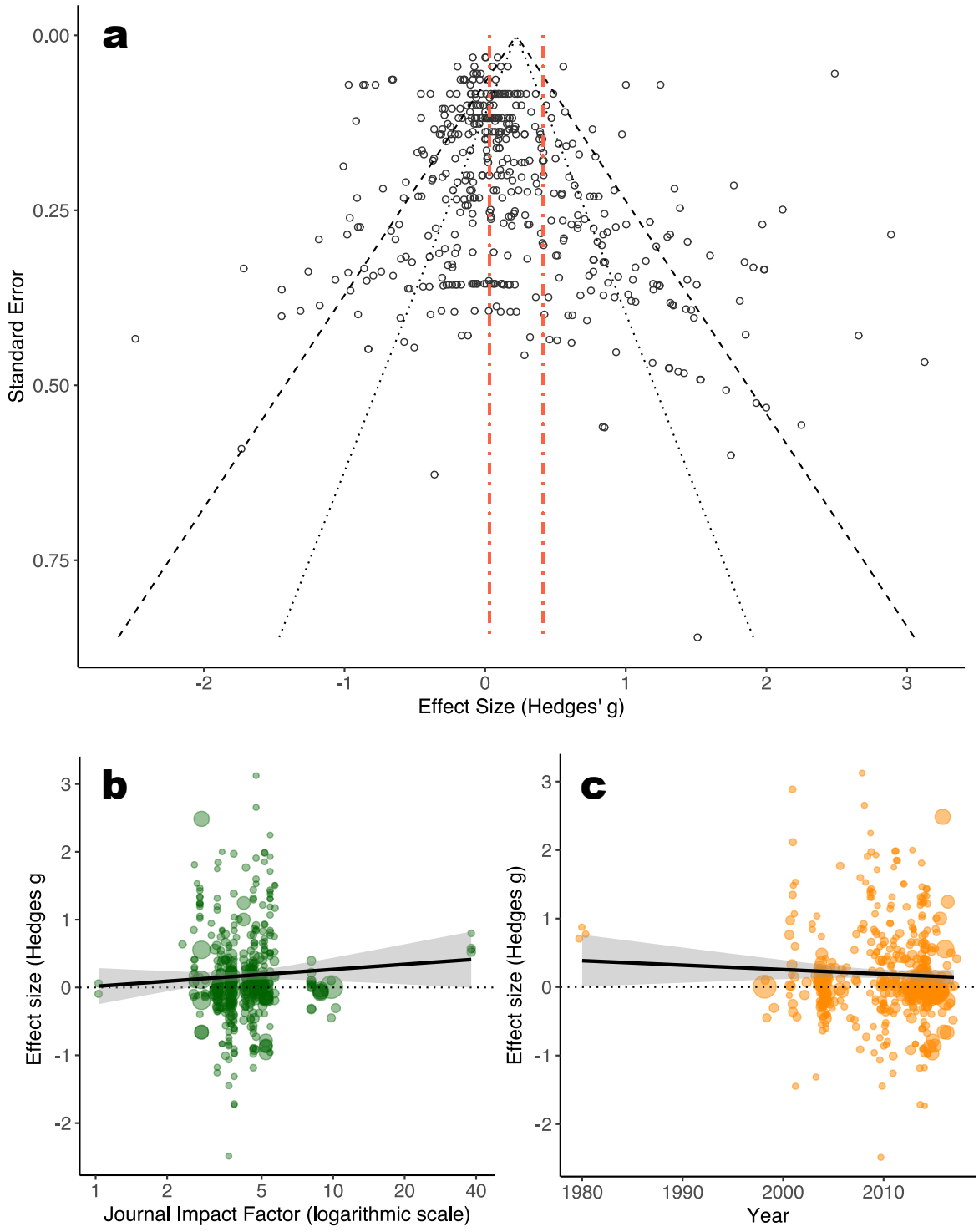

Figure 3: Tests of publication bias are mixed and suggest publication bias may be present. (**a**) Inspection and statistical tests of the funnel plot reveal large amounts of heterogeneity in the dataset with asymmetry from increased low-powered, large effect studies. (**b**) No significant correlation exists between journal impact factor and effect size, (**c**) additionally there is no significant correlation between effect size and year of publication when testing for the time-lag bias. For (**b**) and (**c**) point size is proportional to the precision of the effect size (i.e. the inverse of its variance).

Table 1: Results for the multilevel meta-analysis model testing the effects of sexual selection in different environments and between sexes. Moderator variables whose 95% confidence intervals do not cross zero are shown in bold.

Parameters	**Estimate**	**SE**	**LCI**	**UCI**	**z**	**p**
REML Model						
Intercept	0.21	0.12	-0.02	0.44	1.8	0.069
Both Sexes	-0.064	0.06	-0.19	0.06	-1.0	0.3
Female Sex	**0.054**	0.03	0.00	0.11	1.9	0.057
Stressed Environment	**-0.12**	0.04	-0.20	-0.04	-2.5	0.0033
Both Sexes x Stressed Environment	**0.17**	0.08	0.08	0.33	2.1	0.039
Female Sex x Stressed Environment	**0.25**	0.05	0.15	0.35	4.9	<0.001
Bayesian Model						
Intercept	0.22	0.16	-0.10	0.55		
Both Sexes	-0.063	0.06	-0.19	0.06		
Female Sex	0.054	0.03	0.00	0.11		
Stressed Environment	**-0.12**	0.04	-0.20	-0.04		
Both Sexes x Stressed Environment	**0.16**	0.08	0.00	0.32		
Female Sex x Stressed Environment	**0.25**	0.05	0.15	0.34		

Table 2: Hypothesis tests using restricted maximum likelihood (REML) or Bayesian methods, illustrating how sex and environmental stress interact to modulate effect size. Tests whose 95% confidence intervals do not cross zero are shown in bold.

Condition	**Test**	**Statistical Approach**	**Estimate**	**SE**	**LCI**	**UCI**
In benign environments	Female > Male	REML	-0.054	0.03	-0.11	0.00
		Bayesian	**-0.054**	0.03	-0.11	0.00
In stressful environments		**REML**	**-0.3**	0.04	-0.39	-0.22
		Bayesian	**-0.3**	0.04	-0.38	-0.22
For Females	Stressful > Benign	**REML**	**-0.13**	0.04	-0.20	-0.06
		Bayesian	**-0.13**	0.04	-0.20	-0.06
For Males	Benign > Stressful	**REML**	**0.12**	0.04	0.04	0.20
		Bayesian	**0.12**	0.04	0.04	0.20
For Both	Stressful > Benign	REML	-0.047	0.07	-0.19	0.10
		Bayesian	-0.044	0.07	-0.19	0.10

Discussion

Our meta-analysis revealed that populations evolving under sexual selection often have higher values for multiple fitness traits, relative to populations where sexual selection was experimentally removed or weakened. Notably, sexual selection had small but beneficial effects on direct measures of fitness such as extinction rate, reproductive success (defined as the number of offspring produced) and the proportion of viable offspring. Fitness traits related to immunocompetence were an exception: sexual selection typically resulted in weaker immunity. This result is interesting in light of the hypothesised trade-off between sexually-selected phenotypes and immunity, for example due to immunosuppressive effects of sex hormones[39,40]. Furthermore, the overall benefit of sexual selection was greater for females than males, and this sex difference was magnified in stressful environments. Consistent with stronger selection on female fitness under stress, female phenotypic variance was substantially reduced when sexual selection was applied under stressful as opposed to benign conditions. These results suggest that sexual selection may contribute to population persistence under stressful conditions, such as fluctuating environmental change[33] or spatial variability[41], particularly since female reproductive output is often a limiting factor in population growth[42].

The results of the meta-analysis support predictions that sexual selection can improve population fitness and accelerate adaptation[10–12,33,43–45]. Specifically, our results support arguments that positive genetic correlations between male mating success and female non-sexual traits are common, allowing females to benefit from a genome that has been 'purged' of deleterious alleles through selection on males[8,10,11]. What is less clear is why sexual selection had a larger effect on female trait means and variances as opposed to males (the sex in which sexual selection is typically strongest). Below we discuss possible explanations for this result, in light of the core principle that the speed and magnitude of adaptation depends on additive genetic variance, the structure of genetic covariance, and the strength of selection[46,47].

Firstly, the response of male fitness to sexual selection may be constrained in species where the heritability of male fitness is influenced by the sex determination system. Males are heterogametic in many of the species in our sample (e.g. in species with XY or XO sex determination systems); this can reduce father-to-son heritability relative to mother-to-daughter, since sons never inherit the larger sex chromosome from their fathers[16,48–50], which might slow adaptation in male relative to female traits[50,51]. However, X-linkage is also likely to inflate the contribution of X-linked genes to fitness variance in the heterogametic sex. Under this view, X-linkage facilitates adaptation by increasing the efficacy of selection against recessive deleterious alleles and selection for rare (and recessive) beneficial alleles[52,53]. Perhaps as a result of these conflicting predictions, there seems to be no clear consensus regarding sex-based differences in heritability. For instance, a systematic review found no difference in mean heritability between male and female traits, although there was a male-biased skew in heritability of reproductive traits[54]. Conversely, a recent association study in humans found a number of traits showing higher heritability in females than males[55].

Secondly, selection on males might be weaker than selection on females, resulting in slower adaptation following experimental manipulation of sexual selection. This explanation may initially seem implausible, because net selection on males is generally assumed to be stronger than on females, due to the (actual or hypothesised) sex difference in sexual selection strength and inter-individual variance in fitness[56–59]. However, an oft-overlooked aspect is that selection might frequently be 'softer' on males and 'harder' on females[60], because the local competitive environment is usually more important for males than it is for females. For instance, a mediocre male genotype can have high fitness provided it outcompetes its local

rivals, while low-fitness female genotypes are likely to produce few offspring even when competing with other low-fitness females. Therefore, improvements in genetic quality should have stronger diminishing returns in males, possibly contributing to our finding that the genetic consequences of sexual selection lead to greater fitness benefits to females. Though this argument is speculative, we note that many experimental evolution designs exaggerate the sex difference in the nature of selection, relative to expectations for large, natural populations[61–63]. For example, many studies[64–68] have evolved insects in small sub-populations, each containing one female and multiple males, whose progeny are then mixed and randomly sampled to create the next generation; this design ensures that successful males simply needed to outcompete their rival(s) in the same sub-population, while each female's reproductive output is measured against the entire female population.

Our results suggest that the greater benefit of sexual selection to females than males is magnified in stressful environments. Recent work has emphasised that environmental stress should reduce the strength of sexually antagonistic selection relative to selection that is concordant between sexes. Theoretical models reaching this conclusion[33,69] have been supported by empirical studies[13,34,70]; for example, one study found that high fitness males produced low fitness daughters under benign conditions but high fitness daughters under stress[34]. However, other quantitative genetic studies have shown that stressful conditions do not always reduce sexual antagonism[27,30,71]. Variation in effects of sexual selection in stressful environments may be due to potentially variable responses amongst taxa[31] and environments. Notably, Connallon and Hall[33] predict that the dynamics of environmental change alter the strength of sexual antagonism; for instance, gradual directional selection may facilitate indefinite sexual antagonism, while rapid cyclical change can swiftly remove it. Our meta-analysis suggests that under directional selection imposed by environmental stress, sexual antagonism is likely dampened; allowing sexual selection to facilitate adaptation and persistence.

Although our meta-analysis revealed an overall positive effect of sexual selection, the variation in effect size across the dataset is high, as commonly is the case for studies in ecology and evolution[35]. Most of the heterogeneity was explained by study and the fitness component that was measured, while the taxon, number of generations of evolution, and use of blinding showed less impact on effect size. Taxonomic diversity is limited in experimental evolution studies, which mostly use lab-suitable invertebrates with similar mating systems and sex determination. However, a meta-analysis of macroevolutionary studies on sexual selection and speciation rate found no significant taxon-based differences across a diverse sample of vertebrates and invertebrates (fish, insects, birds, spiders, reptiles, mammals)[72]. Despite this, taxonomic differences in the effects of sexual selection on fitness or patterns of diversity are often expected due to differences in mating systems[62] and sex-determination systems[49]. For example, mate choice is thought to be more prevalent in taxa with well-developed parental care[73], affecting the strength and nature of sexual selection. Furthermore, male heterogametic taxa, including those commonly used in experimental evolution such as flies (Diptera), beetles (Coleoptera) and mice (Mammalia), are likely to have higher heritability of fitness from mothers to daughters. Our results – which show greater fitness effects accruing to females – align with this hypothesis.

Our findings have implications for fundamental and applied research. For example, the beneficial population-level consequences of sexual selection have been proposed as one possible resolution to the long-standing evolutionary puzzle presented by sexual reproduction[74]. If sufficiently strong, these benefits can more than compensate for the costs of sexual reproduction, and prevent sexual populations from being outcompeted by asexual mutants that arise in their midst[10,11,75]. Sexual selection is also important for conservation[6] and captive breeding programs[76]. Within captive breeding programs, genetic diversity is often managed

through the enforced monogamy of a strategically selected (genetically diverse) breeding pair[76]. Captive breeding programs may benefit from allowing sexual selection of 'good genes' or more compatible genes[77], or by increasing maternal investment by females paired with 'attractive' males[78–80]. Additionally, our findings imply that anthropogenic environmental changes that reduce the opportunity for sexual selection, such as eutrophication, pesticides, artificial light and noise pollution, could reduce the genetic quality of the population, and potentially compromise its long-term persistence[81–85]. Equally, our results support recent evidence that human activities that directly counteract sexual selection, such as selective harvesting of the largest or most ornamented males, can lower population fitness[86]. Based on the weight of evidence from experimental evolution, we suggest that sexually-selected populations may be more resilient to environmental change, including anthropogenic environmental pressures, over relevant time scales.

Materials and methods

Literature search

We searched *ISI Web of Science* and *Scopus* on 9th June 2017 for peer-reviewed, English language studies that manipulated the presence or strength of sexual selection using experimental evolution, and then measured some proxy of population fitness. A detailed list of search terms is given in the Electronic Supplementary Material (ESM).

After removing duplicates, we read the titles and abstracts of the remaining 1015 papers and removed those that did not fit our inclusion criteria (typically because they did not present primary experimental evolution data). This left 130 papers, for which we read the full text and applied the inclusion criteria outlined in the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) diagram (Fig. 4). Briefly, we included studies that 1) were conducted in a dioecious animal species, 2) experimentally manipulated the strength of sexual selection (e.g. via experimentally-enforced random monogamy or an altered sex ratio) for at least one generation, and 3) measured a trait that we judged to be a potential correlate of population fitness. This third criterion is the most subjective, because there is rarely enough data to determine whether a particular trait is (or is not) correlated with population fitness. We therefore relied on our best judgement when deciding what outcomes were correlated with population fitness. We categorised the fitness outcomes into three categories: ambiguous, indirect and direct (detailed in Table S1). Briefly, ambiguous measures of fitness were those that are reported to have an unclear or variable association with fitness (e.g. body size, mating duration and early fecundity). Indirect fitness components were those that are often used as a proxy of fitness but do not directly measure aspects of success in reproduction or population viability (e.g. lifespan, mating success and ejaculate quality/production). Finally, direct measures of fitness (reproductive success, offspring viability and extinction rate) are those that measure fitness through components of reproduction or long-term viability. The ESM describes why specific papers recovered in the literature search were included or excluded (Table S2).

Of the 130 papers subjected to detailed screening, 62 were excluded based on the PRISMA criteria (Fig. 4). Additionally, three papers presented insufficient information to calculate effect size. In these cases, we contacted the authors and attempted to obtain the missing data, with partial success. The final meta-analysis covered data from 65 papers.

Data extraction

From each paper, we first attempted to extract the arithmetic means, standard deviations and sample sizes of each of the different treatment groups, which facilitate calculation of effect size (see below). Typically, there were two or three treatments, which varied in the strength of sexual selection. For some papers, summary statistics were not written down, but were presented in a figure such as a bar chart: in these cases, we extracted the data using *WebPlotDigitizer* v.3.12[87]. If the treatment means were not reported (and the raw data were unavailable), we instead calculated effect size from test statistics comparing treatment means (*e.g.*, F, t, z or χ^2 values), which we used to estimate effect size using several formulae (see below).

Where possible, we extracted data for each independent replicate or experimental evolution line within a study; otherwise, we used pooled treatment means. For studies that repeatedly measured the same population across multiple generations, we only extracted data for the last reported generation.

In addition to the data used to calculate effect size, we collected a set of moderator variables for each paper (see Table S3 and associated ESM). The moderators were selected due to their ready availability, and because we hypothesised that they might explain some of the observed heterogeneity in effect size. A key moderator was whether the environmental conditions that a population evolved under were stressful (*e.g.*, elevated mutation load, novel/sub-optimal food source, increased sub-lethal temperatures). Additionally, we collected details for each effect size on: sex (male, female or a mixed sample of both), taxon (flies, beetles, mice, nematodes, mites, crickets and guppies), blinding of researchers to treatments and number of generations a treatment group underwent experimentally evolution. In the interests of creating a useful data resource, we also recorded details about each experiment that were not formally analysed due to a shortage of data, such as the type of sexual selection that was manipulated (pre-copulatory, post-copulatory, or both) and the male to female ratio, which is included in the ESM (Table S3).

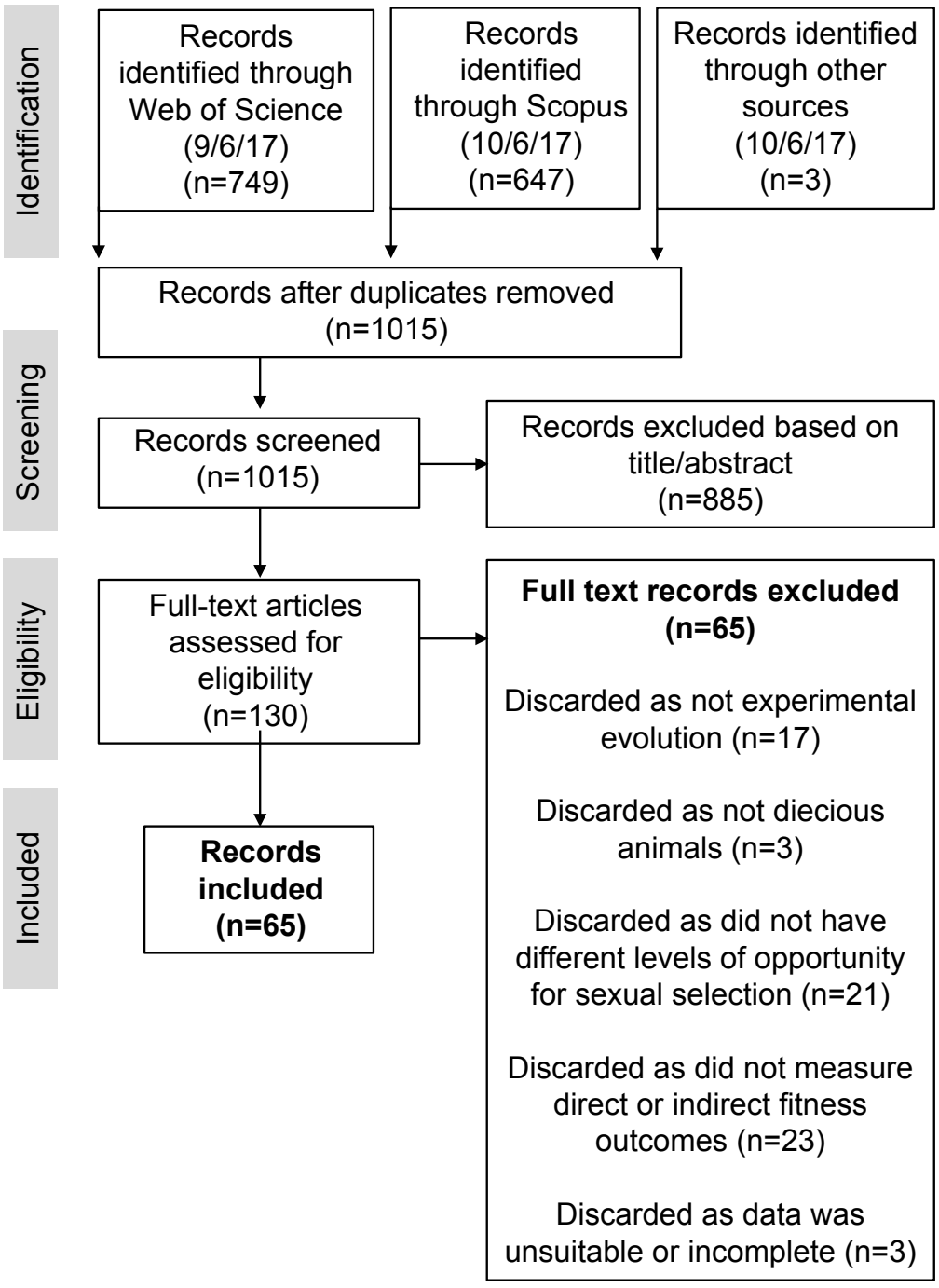

Figure 4: Flow of inclusion and exclusion of studies identified during the literature search, presented as a PRISMA diagram with number of published papers in brackets.

Effect size calculation

For each measurement of each pair of treatments, we estimated the standardised effect size Hedges' g[88]. Similar to Cohen's d, Hedges' g expresses the difference in means in terms of standard deviations (making it dimensionless), but it is more robust to unequal sampling and small sample sizes[89]. For comparisons of extracted treatment means, we calculated Hedges' g using the `mes` function in the *compute.es* R package[90]. To calculate Hedges' g from test statistics, we used the `fes`, `chies`, and `tes` functions in the *compute.es* package (for F, χ^2 and t statistics, respectively). The `propes` function was used to calculate effect size from a difference in proportions; in two cases[91,92], a proportion was equal to one (producing infinite effect sizes), and so we subtracted one from the numerator when estimating Hedges' g. In all cases, we selected a direction for the effect size calculation such that in our meta-analysis, negative effect sizes indicate that the removal of sexual selection was associated with higher fitness trait values, and positive effect sizes indicate higher fitness when sexual selection was elevated or left intact. We also inverted the sign of effect sizes pertaining to measurements that are expected to be negatively related to population fitness (e.g. parasite load, mutation load, extinction risk/rate, mating duration (females), mating latency (males), and rate of senescence). Because many of our 65 papers measured multiple fitness outcomes, studied multiple replicate populations, or had three or more sexual selection treatments, we calculated a total of 459 effect sizes.

For the meta-analysis testing whether sexual selection affects phenotypic variance (as opposed to the mean), we estimated the difference in variance between each pair of treatments using the natural logarithm of the ratio between the coefficient of variation for each group (termed $lnCVR$)[36]. The calculation of $lnCVR$ relies on the availability of arithmetic means, standard deviations and sample sizes for the two treatment groups[36,93], and so we were only able to calculate lnCVR for 356 of 459 comparisons.

Mixed effects meta-analysis

Firstly, we obtained a weighted mean effect size (Hedges' g) for the entire dataset, using both Bayesian and restricted maximum likelihood (REML) approaches for completeness. The weighted mean was obtained by fitting a model with no moderator variables (i.e. fixed effects), but fitness component (*e.g.*, body size, female fecundity), study ID, and taxon as random/group-level effects. That is, we separately model correlations between different effect sizes sourced from the same study, taxon, or pertaining to the same fitness component, and account for these interdependencies when estimating the overall effect. Given the small number of phylogenetically diverse species, we did not utilise phylogenetic corrections within the models.

Secondly, we fixed fitness component as a moderator variable in Bayesian and REML models (whilst maintaining study and taxon as group-level effects) to derive predictions for effect size within each of the 20 fitness components, using the relevant `predict` functions for each of the `R` packages used (see below). Additionally, we report Bayes Factors (BF), giving the likelihood ratio that the focal effect size differs from zero. Further details on model parameters can be found within the ESM.

Thirdly, we measured the impact of environment, sex and their interaction on the effect size (Hedges' g and $lnCVR$) associated with the manipulation of sexual selection, by fitting these predictors as moderators in a pair of separate mixed-effects meta-analyses. These meta-analyses were restricted to effect sizes calculated from 'unambiguous' outcomes (i.e. those scored as being directly or indirectly related to population fitness), as well as those where we were able to define the environmental conditions as either stressful or benign

(Hedges' g $n = 336$; $lnCVR$ $n = 277$). We again fit study ID, fitness component, and taxon as random/group level effects. Models investigating other moderators such as number of generations and blinding are presented in the ESM.

Meta-analyses fit by REML were implemented in the *metafor* `R` package (v. 2.0)[94], while their Bayesian equivalents used the *brms* `R` package (v. 2.3.1)[95] to specify and run models in the Stan programming language.

Publication bias

We tested for publication bias via funnel plots, using Egger's test to quantify plot asymmetry[96,97]. However, funnel plot asymmetry does not necessarily indicate publication bias; asymmetry can arise simply through between-study heterogeneity[98]. Thus, we utilised several other methods for checking publication bias. We investigated whether the time-lag bias[37] – where effect size magnitudes decrease over successive years – is present. Additionally, we assessed a potential source of publication bias through the correlation between effect size and journal impact factor[38], which can arise if null or countervailing results are more difficult to publish. The journal impact factor for each effect size at the time of publication was obtained using *InCites Journal Citation Reports*.

Code and data availability

The code and data used to perform this meta-analysis is available as Electronic Supplementary Material and on Github (**link hidden for double bind review**). The repository (**link hidden for double bind review**) provides complete data tables of effect sizes, variance and sample sizes needed to reproduce our results.

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