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Letter

Male harm suppresses female fitness, affecting the dynamics of adaptation and evolutionary rescue

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Abstract

One of the most pressing questions we face as biologists is to understand how climate change will affect the evolutionary dynamics of natural populations and how these dynamics will in turn affect population recovery. Increasing evidence shows that sexual selection favors population viability and local adaptation. However, sexual selection can also foster sexual conflict and drive the evolution of male harm to females. Male harm is extraordinarily widespread and has the potential to suppress female fitness and compromise population growth, yet we currently ignore its net effects across taxa or its influence on local adaptation and evolutionary rescue. We conducted a comparative meta-analysis to quantify the impact of male harm on female fitness and found an overall negative effect of male harm on female fitness. Negative effects seem to depend on proxies of sexual selection, increasing inversely to the female relative size and in species with strong sperm competition. We then developed theoretical models to explore how male harm affects adaptation and evolutionary rescue. We show that, when sexual conflict depends on local adaptation, population decline is reduced, but at the cost of slowing down genetic adaptation. This trade-off suggests that eco-evolutionary feedback on sexual conflict can act like a double-edged sword, reducing extinction risk by buffering the demographic costs of climate change, but delaying genetic adaptation. However, variation in the mating system and male harm type can mitigate this trade-off. Our work shows that male harm has widespread negative effects on female fitness and productivity, identifies potential mechanistic factors underlying variability in such costs across taxa, and underscores how acknowledging the condition-dependence of male harm may be important to understand the demographic and evolutionary processes that impact how species adapt to environmental change.

Keywords: sexual conflict, sexual selection, local adaptation, evolutionary rescue

Lay summary

For species to persist in the face of climate change, adaptation needs to be fast enough to prevent extinction. If population decline is too abrupt, adaptation will be less likely to promote recovery, leading to extinction. Therefore, numerous studies have sought to determine how species can adapt and escape extinction. Sexual selection can promote genetic adaptation, but often has a by-product, sexual conflict, that promotes adaptations beneficial for one sex and detrimental to the other. Such is the case of male adaptations that increase male reproduction by harming females (male harm). Male harm is widespread and has been shown to decrease female and population productivity in some species, facilitating extinction. Furthermore, there is increasing evidence that the degree of male harm to females depends on environmental changes and how well males are adapted to them. However, we ignore how strong the effects of sexual conflict across taxa are, or how ecological feedback on sexual conflict may affect the rate of adaptation and population recovery. Here, we first conducted a meta-analysis to quantify the effect of male harm on female fitness and show, across taxa, that there is an overall negative effect that seems to be dependent on proxies of sexual selection. Then, we used a series of theoretical models to show that, although eco-evolutionary feedback on sexual conflict can limit population decline, this comes at the cost of slowing down the rate of adaptation and population recovery. Our study suggests that understanding how quick environmental changes affect sexual conflict can increase our understanding of how populations adapt and recover in the face of climate change.

Introduction

Sexual selection can play a major role in adaptation and evolutionary rescue by promoting genetic adaptation through genic capture and purging the genome of deleterious mutations (Gómez-Llano et al., 2020, 2021; Grieshop et al., 2021; Lorch et al., 2003; Parrett & Knell, 2018; Rowe & Houle, 1996). Furthermore, given that sexual selection is usually stronger in males than females (Janicke & Morrow, 2018; Singh & Punzalan, 2018; Winkler et al., 2021), this

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can be accomplished while minimizing associated demographic costs (Agrawal, 2001; Grieshop et al., 2021; Martinossi-Allibert et al., 2018; Siller, 2001; Whitlock & Agrawal, 2009). However, sexual selection also tends to favor the evolution of sexual roles (Janicke et al., 2016), which fosters sexual conflict between the sexes (Arnqvist & Rowe, 2005). In particular, interlocus sexual conflict frequently leads to antagonistic co-evolution, favoring male adaptations that increase male reproductive fitness at the cost of female fitness, what we call male harm (Arnqvist & Rowe, 2005; Parker, 1979). Given that population growth is determined to a large extent by female fitness, male harm resulting from sexual conflict can reduce population growth and even increase extinction risk (Kokko & Brooks, 2003; Kokko & Rankin, 2006; Le Galliard et al., 2005; Martins et al., 2018; Rankin et al., 2007). In addition, male harm seems to be extraordinarily diverse and widespread across the tree of life but, although we assume it has net negative consequences for females across taxa, we have no quantitative evidence on whether this is the case in a comparative context, or whether its impact on female fitness depends on the type of harm or the intensity of sexual selection. Furthermore, if male harm has widespread demographic effects on populations, these could also impact the process of adaptation and population resilience to environmental change.

To persist in the face of climate change, adaptation must be fast enough to avoid extinction, a process called evolutionary rescue (Gomulkiewicz & Holt, 1995). The evolutionary rescue has an ecological and an evolutionary component. The ecological component is population demography, as population size first declines due to maladaptation, followed by stabilization and recovery as the population adapts (Carlson et al., 2014; Gomulkiewicz & Holt, 1995). Initial population size and rate of decline are key determinants of evolutionary rescue via bottleneck effects and increased inbreeding, which can further decrease reproduction and/or survival—pushing populations into an extinction vortex (Fox & Reed, 2011; Keller & Waller, 2002; Plesnar-Bielak et al., 2012). The evolutionary component is a genetic adaptation, as population recovery is achieved by an increase in the frequency of adapted genotypes (Carlson et al., 2014). Thus, if a population suffers an abrupt decline or remains at small population size, the likelihood of extinction increases (Gomulkiewicz & Holt, 1995; Gomulkiewicz & Houle, 2009; Gomulkiewicz & Shaw, 2013; Orr & Unckless, 2008), whereas genetic adaptation facilitates population recovery and dictates the rate of the recovery. The evolutionary and ecological components set the scene for sexual conflict to play a role in the evolutionary rescue, particularly so because recent evidence suggests that sexual conflict and male harm, and its impact on populations, depend on the environment. First, adaptations resulting from sexual conflict are typically condition-dependent, and therefore likely to depend on environmental conditions (Chung et al., 2021; Fricke et al., 2009; Marden & Rollins, 1994; Plaistow & Siva-Jothy, 1996; Rowe & Rundle, 2021). Second, independent of condition, environmental fluctuations can affect the expression and maintenance of traits involved in male harm and female resistance, and thus their impact on population viability (Baur et al., 2022; Fricke et al., 2009; García-Roa et al., 2020; Perry & Rowe, 2018; Plesnar-Bielak & Lukasiewicz, 2021; Rostant et al., 2020). This means that males in maladaptive environments will be less capable of harming females, and females less capable of resisting harm, raising intriguing questions about the interplay between environmental change, sexual selection, and evolutionary rescue.

In this study, we aimed to explore whether male harm negatively affects female fitness across taxa, examine some of the main factors that may modulate such harm, and study how ecological feedback on the costs of male harm and female resistance may affect evolutionary rescue in terms of both its ecological (i.e., demographic) and evolutionary (i.e., genetic adaptation) components. To achieve this aim, we first conducted a comparative meta-analysis on studies manipulating male harm levels and measuring the fitness consequences for females. We analyzed whether variation in costs of male harm depends on different proxies of pre- and post-copulatory sexual selection (i.e., sexual size dimorphism (SSD) and sperm competition intensity) and the type of harm (i.e., direct and indirect harm). Then, we developed a population genetic model and used numerical simulations to study the effects of male harm on the rate of genetic adaptation and population recovery after the environmental change. We further explored how variation in the mating system modulates the effects of male harm in evolutionary rescue.

Methods

Meta-analysis

We conducted a systematic review following the PRISMA protocol (Liberati et al., 2009; O'Dea et al., 2021) to look for studies that experimentally manipulated the level of male harm to females and measured its outcome in terms of female fitness (i.e., fecundity and/or offspring sired; see Supplementary Materials section 1.1 for details). We conducted three literature searches using Scopus, PubMed, and Web of Science (WoS) databases. The first one with the search terms "sexual conflict" & "male harm" OR "sexual conflict" & "female harm," the second with the search terms "sexual conflict" & "female fitness" OR "sexual conflict" & "female productivity" OR "sexual conflict" & "female fecundity" OR "sexual conflict" & "female reproductive success," and a final one with the search terms "sexual conflict" & "harassment." Overall, we collected 121 effect sizes from 32 species and 51 studies.

We used the standardized mean difference with a small sample correction (i.e., Hedges' g) to compare mean female fitness measures across experimental treatments. To correct for the possibility that population variances in the two treatments differ, we made use of a corrected version of Hedges' g that controls for heteroscedastic population variances (hereafter called SMDH (Bonett, 2009)). We also tested whether the variability in female fitness between the control and treatment groups differed using the log coefficient of variation Ratio (lnCVR) that compared the coefficient of variation between control and treatment groups (Nakagawa et al., 2015). Given lnCVR makes certain assumptions about mean-variance relationships, which are not always upheld, we also ran an "arm-based" analysis using the log of the standard deviation (lnSD) in a multilevel meta-analytic model. See Supplementary Materials for full details (section 1.4). Both results provided similar conclusions about the change in variability between control and treatment groups so we only present lnCVR. Given lnCVR is a log ratio, we can only use ratio scale data to compute the effect size (Nakagawa et al., 2015). In addition, data are assumed to be normally distributed; we tested this assumption using a modified Geary's test proposed by Lajeunesse (2011). Only six effect sizes failed the test, and three of these were not ratio scale data. As such, we excluded these effect size data from our lnCVR (and lnSD) analysis. In all cases, the CV of the control group was subtracted from the CV of the treatment group such that positive effect sizes suggest greater variance in the control group and negative effect sizes greater variability in the treatment group (after controlling for the mean).

Effect sizes were calculated by subtracting the treatment group mean from the control group mean. As such, positive effect sizes indicate that female "fitness/traits" or variance in control groups was higher than female "fitness/traits" or variance in treatment groups.

We first fit multi-level meta-analytic (MLMA) models (intercept only) including study and species-level random effects. An observation-level random effect was also included. We first fit a model that included a species-level random effect with a phylogenetic correlation matrix, but the model containing only a species-level random effect variance was better supported, so we did not include phylogeny in our models. We applied robust variance estimators to correct standard errors from our models given we obtained many effect sizes from the same study. Using our MLMA models, we also calculated effect size heterogeneity using I^2. (Higgins & Thompson, 2002).

We explored drivers of effect size heterogeneity using multi-level meta-regression (MLMR) models, which included fixed effects (i.e., moderators) that we *a priori* predicted would impact female fitness: (a) an index of SSD, (b) the type of male harm, and (c) sperm competition intensity. We also considered relevant interactions where sufficient data was available. We chose the model with the lowest AIC_c or the most parsimonious model. We fit all models with a maximum likelihood for model comparison of fixed effect structure, and subsequently re-fit with restricted maximum likelihood when we identified the fixed effect structure. We explored publication bias using a new method that relies on fitting an MLMR model accounting for all the moderators available to explain variation in effects (random and fixed effects) (Nakagawa et al., 2022). More specific details about all analytical procedures can be found in the Supplementary Material (section 1).

Population genetic model

To understand the interaction between an allele that confers adaptation to the environment and an allele that regulates how males interact with females, we built a population genetic model considering a haploid population of females and males. Specifically, the model is used as proof of concept to justify assumptions in our numerical simulation (see below). All individuals carry two loci: an adaptation locus with two alleles (0 for the allele with an optimal phenotype to the environmental conditions, 1 for the allele with suboptimal phenotype), and a harm locus, expressed only on males, with two alleles (0 for the allele that makes individuals harm their sexual partners, 1 for the allele that makes individuals not harm their sexual partners). Individuals go through viability selection, which is stronger in individuals carrying the allele 1 than those carrying the allele 0 on the adaptation locus. Surviving individuals become adults and enter the mating pool. We assume that males that can engage in more reproductive interactions (e.g., coercion and competition) have higher mating success and can inflict more damage to females. Therefore, males with the allele 0 on the harm locus have higher mating success than males with the allele 1. Males harm females in one of two different ways: (a) mating harm, where harm is induced by the mating partner (e.g., traumatic insemination), and (b) mating harassment, where harm to the females is induced by mating and non-mating males. After mating, a diploid zygote forms and recombination occurs between the two loci. Adults then die and new individuals are born.

Details about the derivation of the model can be found in the Supplementary Material (section 2). Briefly, assuming mating harassment, the change in frequency of the adaptation allele is

$$\Delta a = \frac{1}{2}\left(\frac{a(1-d)}{1-a(d-s)-s} + U_{00} + U_{01}\right) - a \quad (1)$$

and the change in frequency of the harm allele is

$$\Delta h = \frac{1}{2}\left(-h + \frac{(s-d)D}{1+a(s-d)-s} + U_{00} + U_{01}\right). \quad (2)$$

Assuming mating harm, the change in frequency of the adaptation allele is

$$\Delta a = \frac{1}{2}\left(\frac{a(1-d)}{1+a(s-d)-s} + \frac{(1-c)U_{00}+U_{01}}{(1-c)U_{00}+U_{01}+(1-cf)U_{10}+U_{11}}\right) - a \quad (3)$$

and the change in frequency of the harm allele is

$$\Delta h = \frac{1}{2}\left(-h + \frac{(s-d)D}{1+a(s-d)-s} + \frac{(1-c)U_{00}+(1-cf)U_{10}}{(1-c)U_{00}+U_{01}+(1-cf)U_{10}+U_{11}}\right). \quad (4)$$

We analyzed the model to find the evolutionary stable equilibrium of the population. In both cases, the change in linkage disequilibrium is $\Delta D = (x''_{00}x''_{11} - x''_{10}x''_{01}) - (x_{00}x_{11} - x_{10}x_{01})$, where x''_{ij} is the frequency of the genotypes in the next generation (with i, j = 0 or 1). Now we can solve the system of equations $\{\Delta a = 0, \Delta h = 0, \Delta D = 0\}$ to find the allele frequencies for which the population will no longer change. Unfortunately, ΔD is too complex to be solved and, therefore, we use a quasi-linkage equilibrium (Kimura, 1965) followed by a perturbation analysis to find approximated solutions to this system of equations. Finally, we do a stability analysis to find the stable equilibrium of the population (see Supplementary Material).

Numerical simulations

We ran a series of numerical simulations to track how male harm affects genetic adaptation and population recovery. To do this, we built a haploid genetic model of a population of males and females with the adaptation locus having two possible alleles: 0 for individuals adapted to the environment; and 1 for individuals not adapted to the environment. Importantly, from the results of the population genetic model, we know that the only evolutionary stable equilibrium is when both the adaptation allele and the harm allele are fixed in the population. Therefore, all males will be capable of harm but if there is an environmental change, only a minority will be adapted to the new environmental conditions. Importantly, we assume that female resistance is condition-dependent, and therefore females with the adapted allele can mitigate the costs of male harm, following empirical evidence (Rostant et al., 2020; Wigby & Chapman, 2005). Numerical simulations follow the same life cycle as the population genetic model, with the distinction that instead of two alleles (adaptation and harm) there is only one allele affecting survival and harm. Individuals go through viability selection (stronger in individuals with allele 1 than allele 0), after which surviving individuals enter a mating pool. During reproduction, males with allele 0 have higher mating success than those with allele 1 but impose higher costs on females through mating harassment or mating harm. Females with allele 0 can mitigate some of that harm. Offspring inherit the allele from either parent randomly and form a new generation.

With the numerical simulations, we first tracked the population recovery and rate of genetic adaptation in both scenarios of sexual conflict, mating harassment, and mating harm, when adapted males harm females to a higher degree than non-adapted males and when all males harm females to the same degree. Then, we explore the effect of sexual conflict in polygynous (i.e., males can mate with multiple females) and

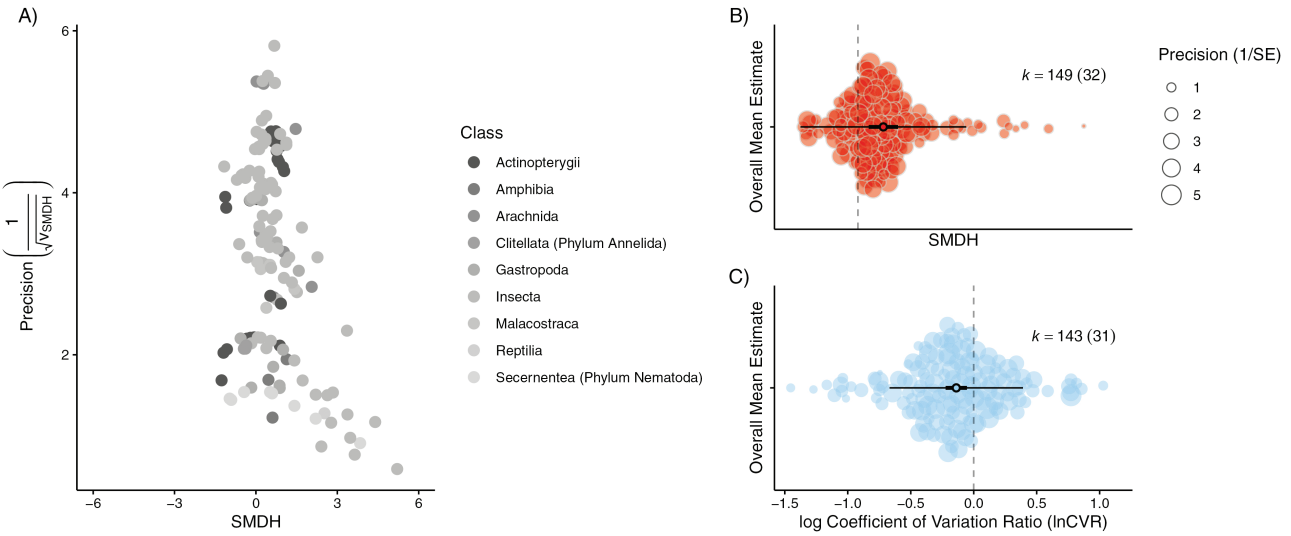

Figure 1. (A) Funnel plot of effect size as a function of precision (i.e., inverse of sampling standard error). (B) orchaRd plot of the overall meta-analytic mean SMDH. (C) Orchard plot of the overall meta-analytic mean for lnCVR. In (B) and (C) the mean is presented with 95% confidence intervals (thick black bars) and 95% prediction intervals (whiskers).

monogamous (i.e., males and females can only mate with one partner) populations. Detailed information regarding simulations can be found in the Supplementary Material (section 3). Briefly, under mating harassment, the birth rate of females with alleles 0 and 1 is

$$b'_0 = R_{00}(1 + b*(1 - Sc*(1 - r))) + \frac{R_{10}}{2}(1 + b*(1 - Sc*(1 - r))) + \frac{R_{01}}{2}(1 + b*(1 - Sc)) \quad (5a)$$

$$b'_1 = \frac{R_{10}}{2}(1 + b*(1 - Sc*(1 - r))) + \frac{R_{01}}{2}(1 + b) + R_{11}(1 + b*(1 - Sc)) \quad (5b)$$

where b is the intrinsic birth rate of females, which is affected by the strength of male harm (Sc) and the female resistance (r). We assume female resistance to male harm (r) is condition dependent. Thus, females with the adapted allele can mitigate the costs of male harm. We assume males with allele 0 impose higher costs than males with allele 1 by a scaling factor f. Therefore, the strength of male harm depends on the frequency of the adapted allele in males,

$$Sc = (c * M_0) + ((c * f) * (M_1)) \quad (6)$$

where c is the costs of sexual conflict imposed by males. Importantly, when $f = 0$ male harm is only imposed by adapted males, and when $f = 1$ adapted and maladapted males harm females to the same degree. In the mating harm model, we assume that harm is not imposed via precopulatory mating harassment but during (e.g., traumatic insemination) or following copulation (e.g., transfer of harmful seminal fluid proteins). Female resistance is condition-dependent and thus male harm can be mitigated by females with the adapted allele. Therefore, the costs of sexual conflict depend on the allele of both the male and female mating pair. Then, the birth rate of adapted and maladapted alleles is

$$b'_0 = R_{00}(1 + (b*c*(1 - r))) + \frac{R_{10}}{2}(1 + (b*(c*f*(1 - r)))) + \frac{R_{01}}{2}(1 + (b*c)) \quad (7a)$$

$$b'_1 = \frac{R_{10}}{2}(1 + (b'*(c*f*(1 - r)))) + \frac{R_{01}}{2}(1 + (b'*c)) + R_{11}(1 + (b'*(c*f))) \quad (7b)$$

In all simulations presented here, the population is initialized with a frequency of adapted alleles of 0.1. Moreover, we assume $d_0 = 0.01, d_1 = 0.1$ *and* $b_0 = b_1 = 0.1$. Female resistance is set to r = 0.2, following available empirical data (Rostant et al., 2020), in which case females with the adapted allele can mitigate the costs of male harm by 20%. Note that results do not change qualitatively across further scenarios of female resistance, which we present in the Supplementary Material (section 3). We run all simulations until populations recovered the initial population size (N = 100), or for 100 generations.

Results

Meta-analysis

Overall, we collected 149 effect sizes for a total of 32 species from 51 studies. Unsurprisingly, invertebrates (classes: Insecta, Gastropoda, Malacostraca, Arachnida, Clitellata, and Secernentea) made up most of the data (78.52%). We obtained 26 effect sizes from manipulations on species that resulted in direct harm (e.g., traumatic insemination), 60 from studies that manipulated indirect harm (e.g., mating harassment), and 63 effect sizes from experiments where females received both direct and indirect harm from male matings. We obtained 121 effects from 28 oviparous species, and 28 effects from four viviparous species. Unfortunately, effect sizes from viviparous species were all taken from studies on fish with indirect male harm. As such, we analyzed only "harm type" and an index of SSD.

Male harm negatively impacts female fitness

Experimentally manipulating female harm resulted in a strong decrease in female fitness overall (i.e., positive effect size with control group females having higher fitness than treatment groups: 0.59, 95% CI: 0.25–0.92, $n_{effects} = 149$, $n_{study} = 51$) (Figure 1B). This effect held even when accounting for within-study non-independence (meta-analytic mean using robust variance estimator: 0.59, 95% CI: 0.35–0.83). Increased female harm also resulted in a significantly higher variance in female fitness (Figure 1C) with females in treatment groups having an approximately 12.92% higher variance in fitness when controlling for

the mean (–0.14, 95% CI: –0.22 to –0.05, $n_{effects}$ = 143, n_{study} = 50). Visual inspection of funnel plot asymmetry suggested evidence for publication bias (i.e., missed effects sizes with low precision when female harm was worse in control treatments) (Figure 1A), which was confirmed through the identification of a significant slope between effect size and effective sample size (β = 2.13, 95% CI: 0.56–3.71). This result held true when accounting for heterogeneity using meta-regression models (β = 2.27, 95% CI: 0.56–3.98). Correcting for the possibility of missing studies resulted in the overall meta-analytic mean effect size being indistinguishable from zero (corrected meta-analytic mean: –0.14, 95% CI: –0.78 to 0.5). While these findings suggest that additional empirical studies may change the overall magnitude of effects, possibly showing weaker effects than what is currently published, our current state of knowledge suggests that female fitness is indeed compromised overall by male harm. When accounting for sampling variance there was high effect size heterogeneity (I^2_{Total} = 0.91, 95% CI: 0.89–0.93) with the most variance being the result of between study (I^2_{Study} = 0.46, 95% CI: 0.34–0.57) and species ($I^2_{Species}$ = 0.23, 95% CI: 0.14–0.34) effects. The trait type and phylogeny explained much less variation overall (I^2_{Trait} = 0.17, 95% CI: 0.09–0.26; $I^2_{Phylogeny}$ = 0.05, 95% CI: 0.04–0.07) (Table 1).

Negative effects on fitness in relation to the type of male harm to females

Male harm type appeared to impact the overall magnitude of effects explaining 12.12% of effect size variance (Figure 2A). Species where mating results in both direct and indirect harm to females have males that significantly impact female fitness (overall meta-analytic mean: 0.85, 95% CI: 0.52–1.18, $p \leq 0.0001$). Surprisingly, however, species with direct female harm only (i.e., traumatic insemination), showed a small and opposite effect on female fitness (overall meta-analytic mean: –0.21, 95% CI: –0.81 to 0.4; Figure 2A), however, this effect did not differ significantly from zero (p = 0.49). While indirect male harm also resulted in a smaller overall meta-analytic mean relative to species with both, it was still significantly positive (overall meta-analytic mean: 0.51, 95% CI: 0.17–0.85, p = < 0.01), and only marginally different than species with both direct and indirect male harm (Figure 2A). Regardless, differences among species exhibiting different types of female harm seemed to be driven primarily by a single study (Taylor, 2008) on *Drosophila* simulans (very high Cook's distance). Removing this effect resulted in no significant difference between harm-type categories. Harm type did not impact the overall variance in female fitness between control and treatment groups ($F_{2,140}$ = 0.15, p = 0.86; Figure 2B).

Female fitness appears more compromised with increasing SSD

As SSD becomes less biased toward females (i.e., female size decreases with respect to males), female fitness is more negatively impacted by male harm (unstandardized slope, β_{SSD}= 1.76, 95% CI = –0.05 to 3.56, p = 0.05, $R^2_{marginal}$= 11.22%; Figure 3). In addition, there was no evidence that the effect of SSD varied according to the type of male harm exhibited (i.e., no interaction between harm type and SSD; ΔAIC_c=37.6, with the main effects model having the lowestAIC_c). Interestingly, this pattern qualitatively appears to reverse in species where males are much larger than females (i.e., SSD > 0; Figure 3). However, there is only one species where SSD is greater than 0 (i.e., *Idotea balthica*), making any concrete conclusions premature.

Table 1. Number of effect sizes, studies, and species along with overall meta-analytic mean (average SMDH) and 95% confidence (CI) and prediction intervals (PIs). Heterogeneity estimates and 95% CIs are also provided for study, trait, species, and total heterogeneity (excluding sampling variance).

$n_{effects}$	$n_{studies}$	$n_{species}$	Meta-analytic mean	95% CI	95% PI	I^2_{study}	*I2trait*	$I^2_{species}$	I^2_{total}
149	51	32	0.59	0.35 to 0.83	–1.28 to 2.45	45.77% (34.28–57.49%)	16.96% (9.41–26.05%)	22.9% (14.45–33.5%)	90.93% (88.63–92.75%)

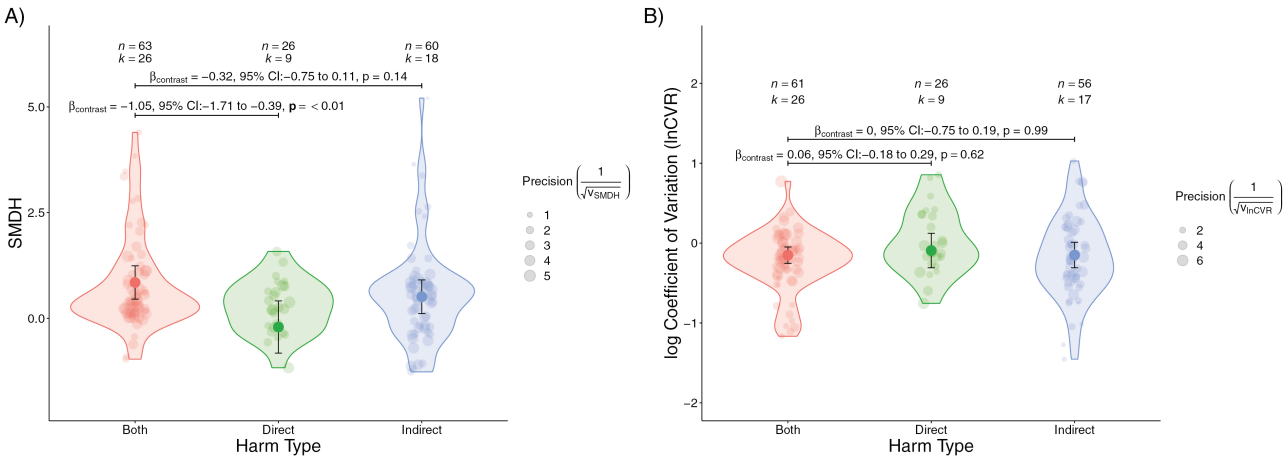

Figure 2. Distribution of effect sizes, overall meta-analytic mean estimates and 95% confidence intervals (CIs) across species with different types of male harm towards females. (A) The standardized mean difference with heteroscedasticity correction (SMDH) and (B) the log coefficient of variation (lnCVR). Total effect sizes (n) and total number of studies (*k*) are provided for each level of male harm type. Relevant contrasts between meta-analytic means are provided, along with 95% CIs and the significance of contrast. Note that this includes all data. Removing outlier point from single study results in no difference between harm-type categories.

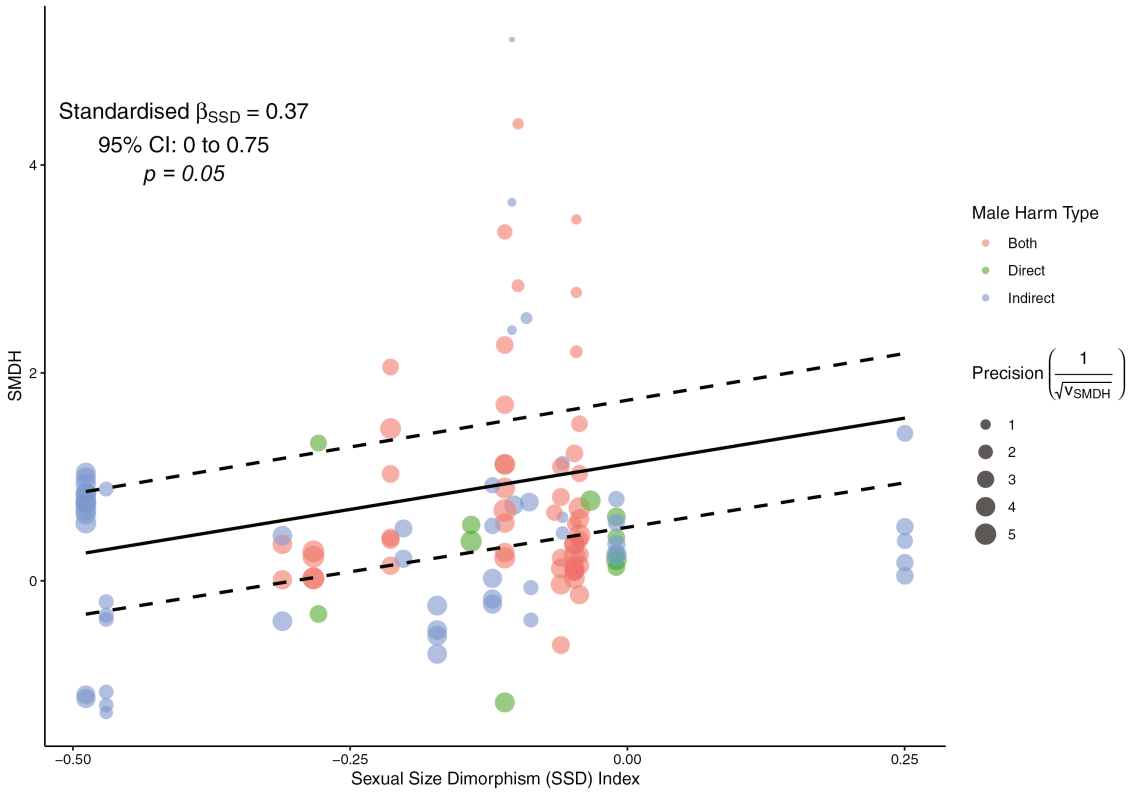

Figure 3. Relationship between the index of sexual size dimorphism for species (log(male/female)) and effect sizes (i.e., heteroscedastic standardized mean differences (SMDH)). Note that we provide an estimate of standardized slope (from a model with a z-transformed SSD index), along with 95% CIs and significance. A slope from a model using an unstandardized SSD index is plotted along with raw data for ease of interpretation and is provided in the text.

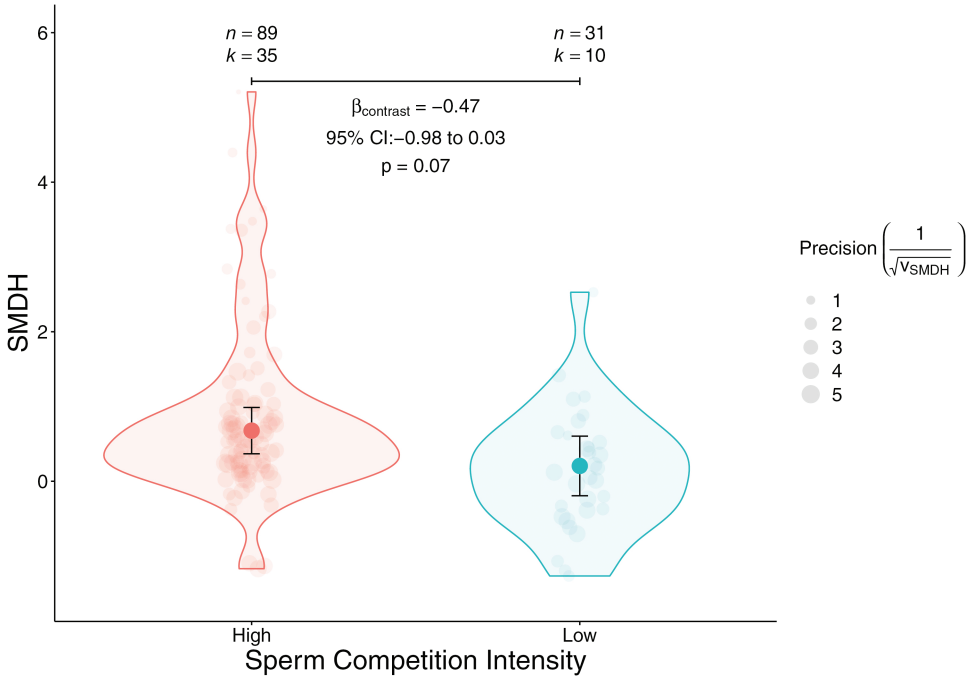

Figure 4. The effect of male harm on female fitness is dependent on sperm competition intensity. Overall, female productivity was affected in species with high sperm competition ($p < 0.001$) but the effect was not different from 0 in species with low sperm competition intensity. However, the difference between species with high and low sperm competition intensity was marginal. Shown are standardized mean differences with a small sample correction (SMDH) and contrast between meta-analytic means, along with 95% CIs and significance of contrast.

Male harm and sperm competition

Species with higher sperm intensity exhibited a significant reduction in female fitness with higher male harm (overall meta-analytic mean: 0.68, 95% CI: 0.37–0.99, $p \leq 0.0001$), whereas the same was not true of species with low sperm intensity (overall meta-analytic mean: 0.2, 95% CI: –0.19 to 0.6, $p = 0.31$; Figure 4). Overall, there was no significant difference between species deemed to have high sperm intensity compared to those with low sperm intensity (Figure 4).

Population genetic model

Results from the population genetic model show that the only candidate stable equilibrium for a population is when both the adaptation and harm alleles are fixed. This result is consistent across different levels of harm and the degree to which non-adapted males can harm females. It is also independent of whether harm comes from mating harassment or mating harm (Figure 5). When non-adapted males are incapable of harming females ($f = 0$; Figure 5), the selective pressure for the harm allele to increase in the population is initially low. As the adaptation allele increases in the population, more males become capable of harming and, accordingly, of extracting the benefits of having the harm allele. At that point, the harm allele starts to increase in the population and the population only stops changing when both the adaptation and the harm alleles are fixed. Stability analysis confirms that this is the only stable point for this population.

Numerical simulations

Condition-dependent sexual conflict can facilitate evolutionary rescue by decreasing the impact on population demography

In our model, populations are initialized in a maladapted state, thereby suffering an initial decline due to the high frequency of maladapted alleles. Once the frequency of adapted alleles is high, populations start to recover, following the U-shape pattern expected in evolutionary rescue (Carlson et al., 2014; Gomulkiewicz & Holt, 1995). The mating harassment model shows that, if males with the adapted allele are more harmful than males with the non-adapted allele ($f < 1$), under weak ($c = 0.2$) and strong sexual conflict ($c = 0.5$) the rate of population decline is reduced along with the extent of the decline. When the sexual conflict was weak, the minimum population size was reached after 52 generations when $f = 0$, compared to after 45 generations when $f = 1$. The extent of the decline also differs. The minimum population size was 34% of the initial size when $f = 0$, compared to 28% of the initial size when $f = 1$. In the case of

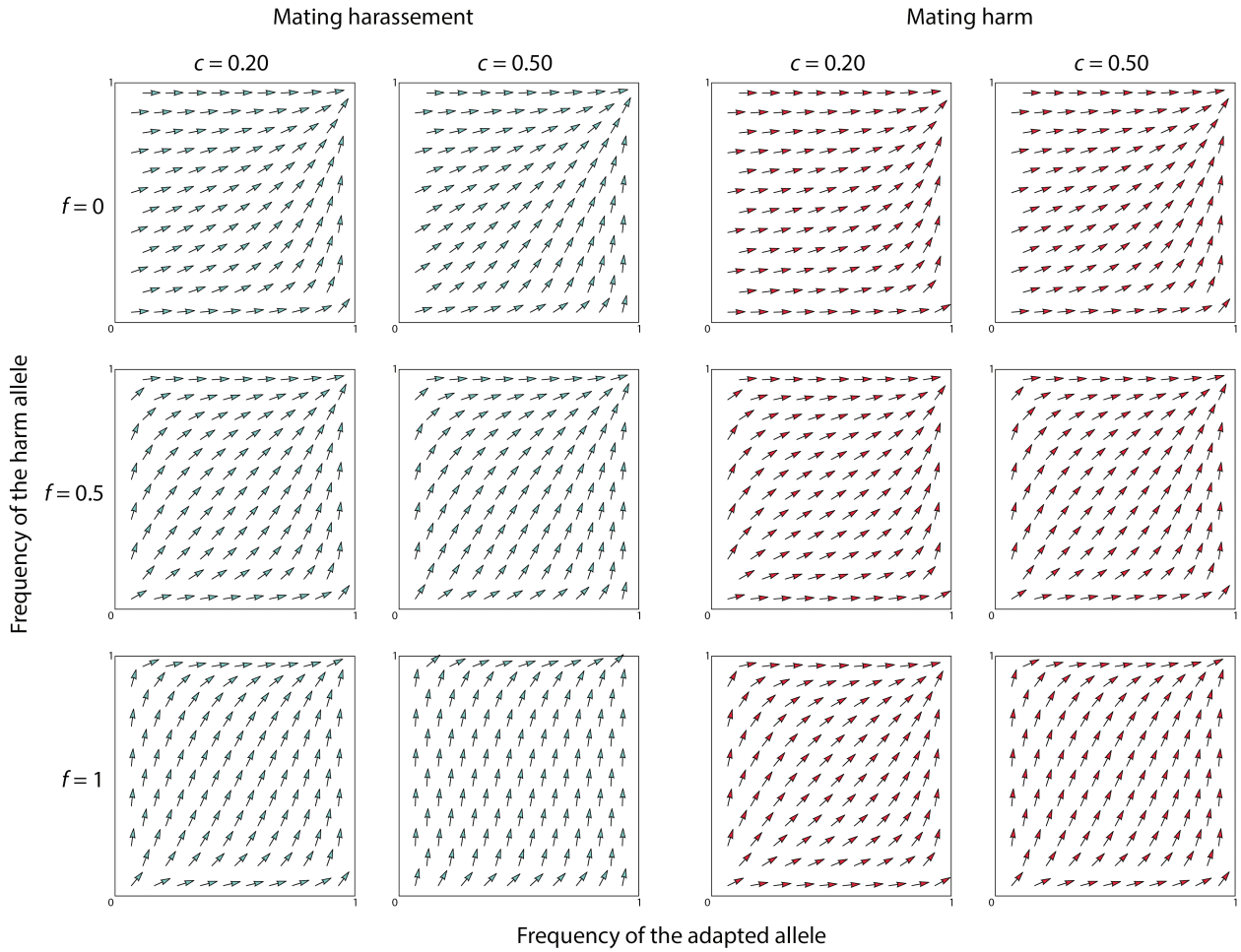

Figure 5. The only stable equilibrium is when both the adaptation allele and the harm allele are fixed in the population. Regardless of the cost of harm for females, the degree to which non-adapted males can harm the females, and the presence of mating harassment (right) or mating harm (left), the only stable equilibrium is when both the adaptation allele and the harm allele are fixed. Parameter values: viability cost of adapted males d = 0.01, viability cost of non-adapted males $s = 0.10$, recombination $r = 0.5$.

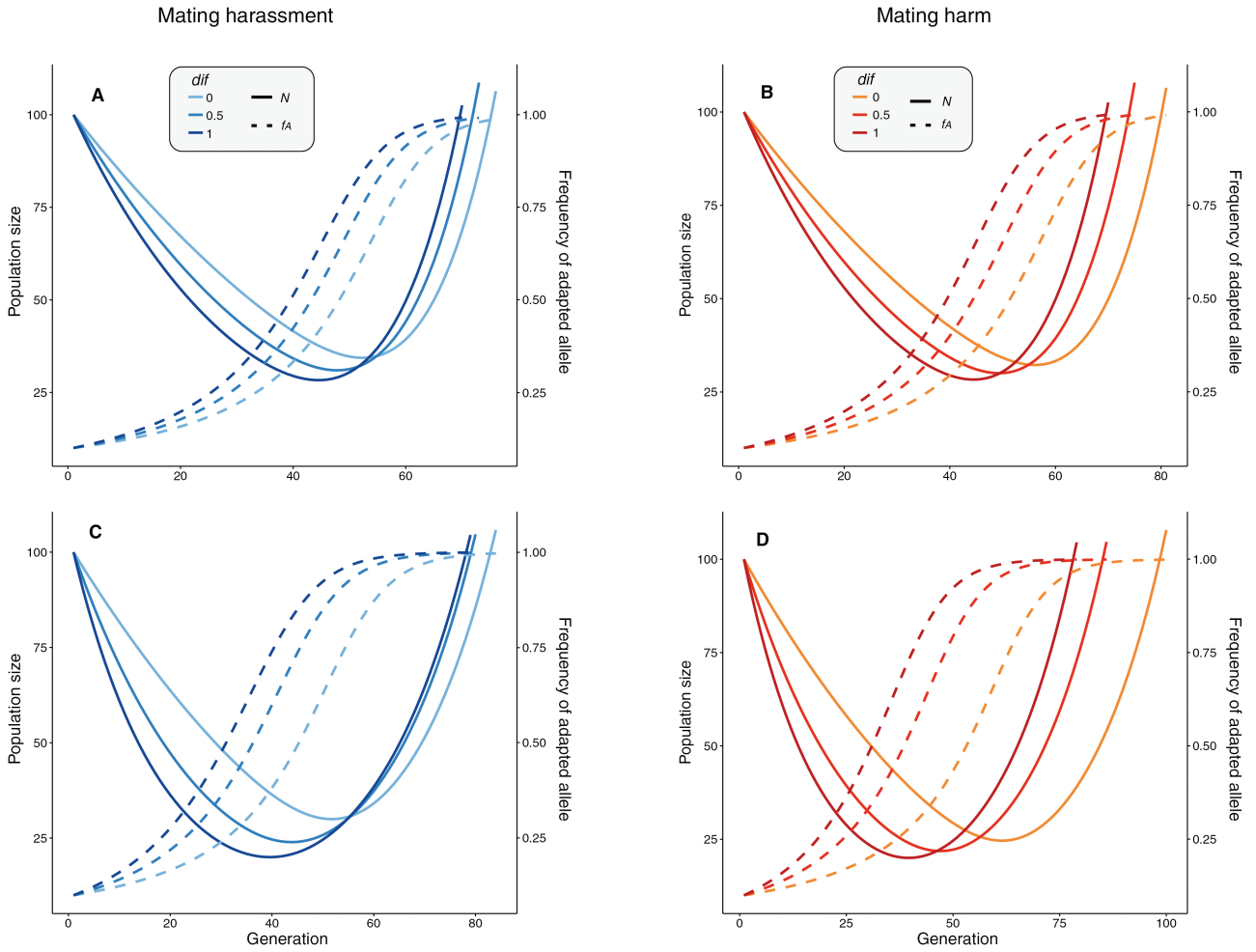

Figure 6. The effect of condition-dependent sexual conflict on evolutionary rescue depends on the strength and mechanism by which the costs are imposed on females. Mating harassment reduces population decline, but slows adaptation, if it is higher in adapted vs. non-adapted males (A, C). Importantly, when sexual conflict is weak ($c = 0.2$; A), populations recover faster if the level of male harm is similar between adapted and non-adapted males, but there is no difference if the sexual conflict is strong ($c = 0.5$; C). When the costs of sexual conflict are imposed through mating harm (B, D), higher harm by adapted males reduces the rate of population decline, but not the extent of this decline. However, populations take longer to recover if the costs are imposed more strongly by adapted males under both low ($c = 0.2$; B) and high sexual conflicts ($c = 0.5$; D), albeit more clearly in the latter case. Note that x axes differ between the top and bottom rows. This is because, under weak sexual conflict, populations recover their initial size much faster than under strong sexual conflict, especially in the case of mating harm. The solid lines reflect population size (N) and the dashed line depicts the frequency of the adapted allele (0). The scaling factor (f) reflects the costs of sexual conflict imposed by males with the non-adapted allele relative to males with the adapted allele ($f = 1$ all males impose equal sexual conflict costs, $f = 0.5$ males with the non-adapted allele impose half the costs than males with the adapted allele, and $f = 0$ only males with the adapted allele impose sexual conflict costs). In these simulations, $h = 0$ and $r = 0.2$.

strong sexual conflict, the minimum population size was 29% of the initial size at generation 52 and 20% at generation 20 for $f = 1$ and $f = 0$, respectively (Figure 6). However, genetic adaptation is faster if all males impose equal costs ($f = 1$). Interestingly, when sexual conflict is weak, populations recover faster if all males are equally harmful than if costs are dependent on the adapted allele, while there is no difference in the rate of recovery under strong sexual conflict. Specifically, when the sexual conflict was weak, the frequency of the adapted allele reached 90% after 63 generations when $f = 1$, and after 55 when $f = 0$. In the case of strong sexual conflict, the adapted allele reached 90% after 61 and 48 generations for $f = 1$ and $f = 0$, respectively (Figure 6).

The mating harm model shows some interesting differences from the mating harassment model. Namely, in the mating harm model, if males with the adapted allele are more harmful, the rate of population decline is reduced, but not so much the extent of the decline. When sexual conflict is weak, minimum population sizes were 32% and 28% of the initial size ($f = 1$ and $f = 0$, respectively), reached at generations 56 and 45 ($f = 1$ and $f = 0$). Similarly, in the case of strong sexual conflict, the minimum population size was 24% of the initial size at generation 62 and 20% at generation 40 for $f = 1$ and $f = 0$, respectively (Figure 6). Notably, when $f < 1$, because populations decline more slowly and maladapted alleles can remain for longer, genetic adaptation and population recovery are slower under both weak ($c = 0.2$) and, more markedly, strong sexual conflict ($c = 0.5$). When the sexual conflict was weak, the frequency of the adapted allele reached 90% after 67 generations when $f = 1$ and after 55 when $f = 0$. In the case of strong sexual conflict, the adapted allele reached 90% after 70 and 48 generations for $f = 1$ and $f = 0$, respectively (Figure 6). Therefore, the effects of sexual conflict on evolutionary rescue not only depend on the strength but also on the mechanism of sexual conflict.

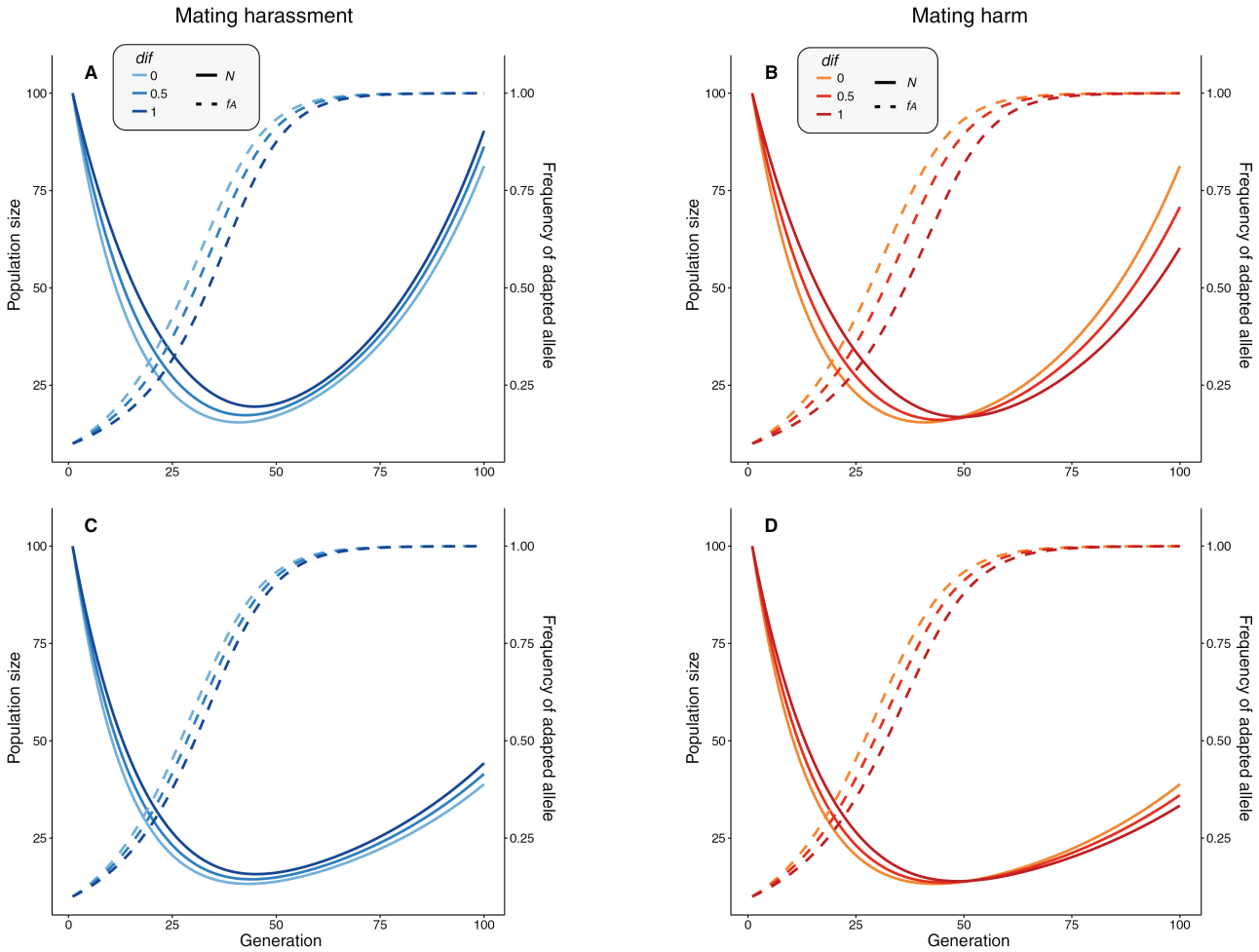

Figure 7. The demographic benefits of the condition-dependent sexual conflict disappear in monogamous populations. In the mating harassment model, more polygynous populations recover faster (h = 0.5, A) than monogamous populations (h = 1, C), and the benefit of the condition-dependent sexual conflict disappears. In the mating harm model, although polygynous populations recovered faster (h = 0.5, B) than monogamous populations (h = 1, D), the effect of the condition-dependent sexual conflict disappear. In all simulations c = 0.5 and r = 0.2. Shown are the population size (N) in the solid line and the frequency of the adapted allele (0) in the broken line.

The demographic benefits of condition-dependent sexual conflict disappear in monogamous populations

Contrary to extreme polygynous populations, in which populations declined less when the sexual conflict was imposed more strongly by adapted males, in monogamous and less extreme polygynous populations this demographic benefit disappeared. In the mating harassment model, when all males impose the same costs of sexual conflict, populations declined 4% less than when only adapted males impose costs of sexual conflict when h = 0.5, and 2% less when h = 1 (Figure 7). The difference was 9% when h = 0 (Figure 6C). Polygyny also facilitated population recovery; population size after 100 generations was twice as large in polygynous over monogamous populations. This is because, in monogamous populations, only a small subset of females received the benefit of mating with adapted males (i.e., higher viability offspring), although condition-dependent sexual conflict did not cause major differences between populations (Figure 7). In the mating harm model, we found no differences in the extent of population decline when the sexual conflict was imposed equally by all males or was more strongly imposed by adapted males; in neither h = 0.5 nor h = 1 (Figure 7). There were no large differences in the rate of genetic adaptation between polygynous and monogamous populations in either the mating harassment or mating harm model (Figure 7).

Discussion

Whether species can adapt fast enough to prevent extinction is a pressing question, especially given the rates of anthropogenic climate change. Although sexual selection has been shown to aid rapid adaptation, it is often associated with sexual conflict, which can have detrimental consequences for female and population fitness. We provide evidence that sexual conflict resulting from male harm has a negative effect on mean female fitness and increases variance in female fitness across taxa. In addition, we show that condition-dependent male harm can affect the evolutionary dynamics of populations, reducing population decline at the cost of slowing the process of adaptation. Therefore, our study suggests that condition-dependent sexual conflict can be an important factor affecting the response of species to climate change.

Our meta-analysis on available studies that have manipulated male harm levels and studied associated female fitness shows that male harm decreases mean female fitness while increasing

variance in female fitness across taxa. The latter finding, that male harm increases the variance in female fitness, is novel and could have interesting evolutionary implications. Namely, it suggests that increased male harm may enhance the opportunity for selection in females, potentially exacerbating condition-dependence selection on females but also increasing associated demographic costs for populations. Thus, and while preliminary, we suggest future studies should look closely into this preliminary but exciting result. The former finding, that male harm decreases mean female fitness, is in line with evidence that has been accumulating in some species for the last few decades (Arnqvist & Rowe, 2005; Parker, 1979). However, it is also important to note that several studies have failed to find a net impact of male harm on female fitness in some species (e.g., Mouginot et al., 2015; Nakata, 2016), and thus our results show that male harm has net negative effects on female fitness across taxa. Interestingly, our results suggest that indirect harm may be more costly for females than direct harm. The lack of negative effects of direct harm might reflect the finding that male sexual traits that are considered harmful for females, such as genital spines, can have beneficial effects in some species by increasing female productivity (e.g., Arnqvist et al., 2021). Our results also suggest that the net effect of male harm on female fitness may be affected by SSD and sperm competition. Although results are preliminary given the relative scarcity of data in this respect. These findings suggest an association between stronger sexual selection and higher male harm to females, which is in accordance with theoretical expectations.

Establishing that male harm is deleterious and increases variance in female fitness raises the intriguing question as to how this may affect population adaptability to novel environments, and how male harm generally interacts with the process of adaptation. We show that condition-dependent sexual conflict has the potential to affect the dynamics of evolutionary rescue. Our models suggest that condition-dependent sexual conflict can limit population decline, but at the cost of slower adaptation. Our results, therefore, suggest that condition-dependent sexual conflict can act like a double edge sword, reducing extinction risk by buffering population decline but delaying genetic adaptation (vs. populations that exhibit unconditional male harm). Unsurprisingly, such effects reduced population decline more when the costs of sexual conflict were larger, in agreement with empirical evidence (García-Roa et al., 2019). Our models further show that when sexual conflict is imposed only by adapted males, maladapted alleles can remain for longer in the population, slowing down the process of adaptation. This, however, is only true if a sexual conflict occurs through mating harassment. If male harm depends on male adaptation to the environment, polygynous populations and populations with mating harassment might be better equipped to adapt and persist in the face of climate change than monogamous populations or populations with mating harm. Importantly, our model assumes a sudden or rapid environmental change, and evolutionary rescue is more likely under gradual change (Bell & Gonzalez, 2011; Carlson et al., 2014), although faster recovery might be at the cost of long-term survival (Liukkonen et al., 2021). Exploring the effects of condition-dependent sexual conflict in gradually changing environments would be an interesting expansion of our work here.

A major assumption of our model is that sexual conflict is imposed more strongly by adapted males, an assumption that has been used theoretically (Bonduriansky, 2014; Connallon, 2015; Connallon & Hall, 2018) and supported empirically (Chenoweth et al., 2015; García-Roa et al., 2019; Long et al., 2012). Moreover, we also assume that females can evolve resistance to male harm and that this resistance is also condition-dependent, as reported for several species (Baur et al., 2022; Rostant et al., 2020; Wigby & Chapman, 2005). Based on this evidence, we believe our model reflects realistic biological scenarios, although the magnitude and generality of these assumptions still need further investigation.

An interesting expansion to our models is the case of assortative mating. Previous studies have shown that sexual conflict can be directed toward high-fecundity females (i.e., better adapted) (Chenoweth et al., 2015; Long et al., 2009), and that males in good condition (i.e., better adapted) can prevent males in poor condition from accessing females (Gómez-Llano et al., 2020). In such a case, the costs of sexual conflict will manifest in a subset of the population (i.e., adapted females), which could reduce the variance in female fitness and the efficiency of selection eliminating maladapted alleles. Similarly, kin selection may reduce the harm that males impose on females (Carazo et al., 2014; Faria et al., 2015, 2020; Rankin, 2011), reducing the fitness difference between adapted and non-adapted males. Therefore, while kin selection has been implied in reducing population costs from sexual conflict, it could maintain non-adapted alleles for longer periods in a population.

Given the current rate of human-induced environmental change decreasing population size and increasing extinction risk across species is expected (Ceballos et al., 2017; Dirzo et al., 2014; Wagner et al., 2021). Understanding what mechanisms modulate the processes of adaptation and population recovery is a pressing need in evolutionary and conservation biology. We have shown here that sexual conflict can be important to understand evolutionary rescue and population extinction at large. Our study underscores the need for a better understanding of the ecology of sexual conflict and its consequences for adaptive processes. Our models are a necessary but preliminary step in this direction but were clearly more theoretical, and empirical research is necessary. Ultimately, some degree of biodiversity loss in response to climate change is inevitable, but mitigation of this loss will require efficient use of conservation resources. To do that, it is vital to understand the processes that better equip species to adapt. Our work generates new sets of hypotheses that, we hope, may further both theoretical and empirical research.

Supplementary material

Supplementary material is available online at *Evolution Letters* (https://academic.oup.com/evlett/qrac002)

Data availability

All data are available at https://github.com/daniel1noble/maternal_eff_sex_conflict

Author contributions

M.G.L. and P.C. conceived the study. P.C., R.G.R., and D.N. did the systematic literature search and extracted effect sizes. DN performed the meta-analysis. G.R.F. did the population genetic model and M.G.L. did the numerical simulations. M.G.L. wrote the main text with substantial input from all the authors.

Conflict of interest: The authors declare no conflict of interest.

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