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Male harm offsets the demographic benefits of good genes

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Sexual conflict can arise when males evolve traits that improve their mating success but in doing so harm females. By reducing female fitness, male harm can diminish offspring production in a population and even drive extinction. Current theory on harm is based on the assumption that an individual's phenotype is solely determined by its genotype. But the expression of most sexually selected traits is also influenced by variation in biological condition (condition-dependent expression), such that individuals in better condition can express more extreme phenotypes. Here, we developed demographically explicit models of sexual conflict evolution where individuals vary in their condition. Because condition-dependent expression readily evolves for traits underlying sexual conflict, we show that conflict is more intense in populations where individuals are in better condition. Such intensified conflict reduces mean fitness and can thus generate a negative association between condition and population size. The impact of condition on demography is especially likely to be detrimental when the genetic basis of condition coevolves with sexual conflict. This occurs because sexual selection favors alleles that improve condition (the so-called good genes effect), producing feedback between condition and sexual conflict that drives the evolution of intense male harm. Our results indicate that in presence of male harm, the good genes effect in fact easily becomes detrimental to populations.

sexual conflict | sexual selection | good genes

Significance

Organisms vary in their biological condition. Those individuals in better condition show improved survival or fecundity, and so populations composed of such individuals should be more viable. However, high-condition individuals also express larger sexually selected phenotypes, some of which, in males, can harm females. The good genes hypothesis posits that sexual selection on condition-dependent traits indirectly increases mean condition and therefore population health. Here, using mathematical models, we show that this effect should rarely be expected when sexual traits cause harm: Instead, good genes selection leads to larger harming traits, reduced female fecundity, and potential population crashes.

Sexual selection frequently favors phenotypes that improve male siring success but diminish the fecundity or survival of interacting female partners (1, 2). Well-studied examples include damaging courtship or harassment behaviors (e.g., ref. 3), traumatic insemination (e.g., refs. 4 and 5), insertion of toxic seminal fluids (6), manipulation over parental care (7, 8), and infanticide (9, 10). The evolution of such "male harming" traits generates sexual conflict, negatively impacting female reproductive output, and thus population recruitment (11–13). For instance, male infanticide is the leading source of infant mortality in Serengeti lions (14) and several primate species (10, 15). Harming phenotypes can thus have severe population-level effects, resulting in reduced population size and even extinction (11, 16–20).

Current theory on male harm is based on the assumption that the phenotype of an individual is solely determined by its genotype (18, 21–29). This contrasts with the observation that many traits show condition dependence whereby individuals in better biological condition (e.g., better provisioned, less diseased, or carrying fewer deleterious mutations) can express more competitive phenotypes. This is especially true of sexually selected traits including those involved in sexual conflict (30–34). For example, male drosophila raised in more favorable environments grow larger body size, which leads them to having greater mating success (35) but also to inflicting more harm onto females (36). Meanwhile, in some species, females in better condition may be more able to mitigate the costs of harm they receive (37, 38). Such condition dependence is known to be relevant for the evolution of sexually selected traits like ornaments (e.g., refs. 39–42) and has been key to explaining how female mating preferences are maintained despite them imposing strong directional selection on male display phenotypes (resolving the "paradox of the lek" refs. 30, 42). However, the role of condition dependence in the evolution and resulting demographic effects of male harm remains unknown. On the one hand, populations with more individuals in better condition should be more viable owing to high-condition females having greater fitness, e.g., a population benefit of so-called "good genes" if condition is genetic; (43). On the other hand, because high-condition males may also express greater harm and thus reduce female recruitment, populations in good condition could be less viable due to sexual conflict.

To disentangle these effects, we developed demographically explicit models of sexual conflict where individuals can vary in their condition, either due to their developmental environment (e.g., the quality of an individual's natal patch) or due to genetic factors (e.g., the number of deleterious mutations carried). We ask whether condition-dependent

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expression evolves for sexual conflict traits, namely the propensity for males to harm females and the ability of females to mitigate harm, and if so, what are the consequences of this for population viability.

Model

We consider a population of diploid males and females who express sex-limited traits ($x \geq 0$ in males and $y \geq 0$ in females) that mediate sexual conflict (*SI Appendix*, Table S1 for a list of our model parameters). For example, x could be the size of a male weapon that improves mating success by forcing copulation but harms females, while y is a female "tolerance" trait that mitigates the damage experienced due to male harm, e.g., the spermalege organ that reduces the wounding caused by traumatic insemination in female bed-bugs (44, 45), see *Discussion* for alternative forms of sexual conflict traits including the evolution of female "resistance". Each male and female is also characterized by its individual condition ($v \geq 0$), which here is a quantitative trait with an environmental and/or genetic basis that increases fitness but also modulates the fitness consequences of conflict. Although different interpretations of "condition" exist in the empirical literature (46, 47), v in our model can be seen as a measure of general health that may reflect an individual's genetic "quality" or how well-provisioned they are (as in, e.g., refs. 30, 40–42, 48–52). This broad definition is agnostic with regard to the proximate biological basis of condition variation, and so the scope of our model encompasses a wide range of possible scenarios, including where condition arises from a combination of genetic or environmental factors or by interactions between them.

At each generation t, the population goes through the following life-cycle events (*SI Appendix*, Fig. S1). 1) $N_{\mathrm{f},t}$ adult females and $N_{\mathrm{m},t}$ adult males come together to mate. Males compete to fertilize females such that a focal male, say $i \in \{1, \ldots, N_{\mathrm{m},t}\}$, mates on average with a proportion ξ_i of females (we specify how relevant quantities such as ξ_i depend on traits and condition later in this section). 2) Females produce zygotes, with the fecundity, or expected number of offspring, of a focal female $j \in \{1, \ldots, N_{\mathrm{f},t}\}$ written as ϕ_j. Each zygote develops into a male or female juvenile with probability r and $1 - r$, respectively (so that r is the sex ratio at birth). 3) Adults die and juveniles become adults. Individuals then undergo sex-specific survival selection to reach the mating pool: a focal male survives with probability $s_{\mathrm{m},i}$, while a focal female with probability $s_{\mathrm{f},j}$. Surviving individuals become the mating adults of the next generation (i.e., the $N_{\mathrm{f},t+1}$ and $N_{\mathrm{m},t+1}$ females and males of generation $t + 1$). Variation in female fecundity and juvenile survival may thus cause demographic changes between generations of this population (i.e., $N_{\mathrm{f},t+1} \neq N_{\mathrm{f},t}$ and $N_{\mathrm{m},t+1} \neq N_{\mathrm{m},t}$).

Sexual conflict typically emerges because males increase their mating success but in so doing inflict harm on females reducing their fitness (1, 2, 21, 26). To capture this, we assume that the expected proportion of females that a focal male i mates with is given by

$$\xi_i = \frac{x_i^{\gamma}}{\sum_{k=1}^{N_{\mathrm{m},t}} x_k^{\gamma}}, \qquad [1]$$

where x_i is the trait expressed by male i (this is equivalent to male mating success in ref. 18 [their Equation 4], similar functions can also be found in models of female choice, e.g., refs. 40–42, 53 when females have a fixed preference for males with a positive trait value). A male's mating success thus increases with the relative size of his trait, and an increase in trait size produces greater mating returns when the parameter $\gamma > 0$ is large. This γ therefore controls the strength of male sexual competition.

The benefit of larger male traits during mating, however, trades off with other components of fitness, depending on condition. Specifically, we assume that expressing larger traits increases the likelihood of death before reaching the mating pool (i.e., decreases survival $s_{\mathrm{m},i}$, e.g., because expressing an extravagant trait increases the risk of predation) but that this cost tapers with individual condition. In other words, high-condition males can more cheaply produce larger phenotypes. Moreover, to reflect the notion that high-condition individuals tend to be in better baseline health (30, 54, 55), we also allow for condition to influence individual fitness independently of sexual traits. Overall, we assume that the survival probability of a focal male i expressing trait x_i and of condition v_i can be written as

$$s_{\mathrm{m},i} = s_{\mathrm{b}} e^{-c_{\mathrm{m}}(v_i)x_i + \alpha(v_i)}, \qquad [2]$$

where s_{b} scales intrinsic male survival and,

$$c_{\mathrm{m}}(v_i) = \frac{\chi_{\mathrm{m}}}{1 + \kappa_z v_i}, \qquad [3]$$

captures the condition-dependent cost of male trait expression, which decreases with v_i according to the parameter κ_z (see *SI Appendix*, Fig. S1*C*, note our Eq. **3** is identical to Equation 2b in ref. 41 and Equation 2a in ref. 56; see also ref. 48 for use of similar functions). When $\kappa_z = 0$, the cost of the male sexual trait is independent of condition so that all individuals pay an intrinsic cost χ_{m}, but as κ_z increases, males in better condition pay an increasingly small cost. The function $\alpha(v_i)$ in Eq. **2** captures the effects of condition on baseline fitness, i.e., independently of the sexual trait. For those effects to be comparable with condition dependence in sexual trait, Eq. **3**, we assume

$$\alpha(v_i) = -\frac{\alpha_0}{1 + \kappa_\alpha v_i}, \qquad [4]$$

where κ_α controls the degree of condition dependence in baseline fitness (similar to κ_z in Eq. **3**). So even where the sexual trait is absent ($x_i = 0$), the survival probability of a male increases as its condition v_i increases, with the rate of increase augmenting with κ_α (plug Eq. **4** into Eq. **2** with $x_i = 0$, *SI Appendix*, Fig. S1*D*).

Meanwhile, the fecundity or expected number, ϕ_j, of zygotes of a focal female j expressing trait y_j and with condition v_j has the following form,

$$\phi_j = \underbrace{\frac{b}{1 + \beta N_{\mathrm{f},t}}}_{\text{(i) density-dependence}} \times \underbrace{e^{-\frac{\eta(\bar{x}_t)}{1+dy_j}}}_{\text{(ii) male harm}} \times \underbrace{e^{-c_{\mathrm{f}}(v_j)y_j + \alpha(v_j)}}_{\text{(iii) condition}}, \qquad [5]$$

where

$$c_{\mathrm{f}}(v_j) = \frac{\chi_{\mathrm{f}}}{1 + \kappa_z v_j}. \qquad [6]$$

Eq. **5** consists of three terms. i) The first captures the effects of density dependence (e.g., due to female–female competition for reproductive resources) where $b > 0$ scales intrinsic female fecundity and $\beta > 0$ controls the strength of density dependence. ii) The second term captures the negative effects of male harm on female fecundity, where $\bar{x}_t$ is the average male trait at generation t, and

$$\eta(\bar{x}_t) = k\bar{x}_t \frac{N_{\mathrm{m},t}}{N_{\mathrm{f},t}}, \qquad [7]$$

corresponds to the amount of harm inflicted by males on a female (as in ref. 18). Such harm increases with the average male trait $\bar{x}_t$ according to a parameter $k > 0$ and the ratio of males to females in the mating pool, $N_{m,t}/N_{f,t}$, so that females experience a higher number of harmful interactions when they are outnumbered by males in the mating pool (as observed empirically, e.g., refs. 11, 57, 58). A female, however, can mitigate these negative effects through her own trait y_j according to $d > 0$, which can be thought of as the efficacy of the female trait. Note that here we assume all females in the mating pool receive an equal quantity of harm, but in some species harm may in fact be stronger for high-condition females than low-condition females because they encounter greater mating attention from males, e.g., ref. 59; we consider this assumption in more depth in our Discussion. iii) Finally, the third term of Eq. **5** represents the effects of individual female condition on fecundity. Mirroring survival in males Eqs. **2**–**4**), we assume high-condition females show greater baseline fecundity (according to κ_α, *SI Appendix*, Fig. S1*A*), and while female fecundity decreases with increased expression of the sexual trait (y_j), this decrease is weaker for individuals in high-condition (according to κ_z, with a maximum intrinsic cost of χ_f, *SI Appendix*, Fig. S1*B*). For simplicity, our analyses assume that trait and condition affect only fecundity in females (so that survival is unaffected, as in ref. 27, i.e., $s_{f,j} = 1$ and all female juveniles survive to adulthood).

Results

Sexual Conflict Is Stronger When Individuals Are in Better Condition. We are interested in how the evolution of sexual conflict traits (y and x) influences demographic dynamics ($N_{f,t}$ and $N_{m,t}$), and how these coupled evolutionary and demographic changes depend on variation in individual condition (v). As a baseline, we capture this variation by assuming that each individual can be in one of two condition classes: high or low (with condition v_H and v_L, respectively, where $v_H > v_L$). Condition is randomly assigned at birth but the propensity to be in high condition can differ among the sexes, with females and males being in high condition with probability P_f and P_m, respectively (and low condition with probability $1 - P_f$ and $1 - P_m$). This can reflect a situation where condition depends entirely on the quality of environment an individual experiences during development, where such experience is random with respect to genotype, has an additive effect on condition (i.e., there are no gene × environment interactions), and is independent for every individual in the population. We allow for the expression of sexual traits to be condition-dependent (i.e., plastic), with-high and low-condition males expressing x_H and x_L, respectively, and similarly y_H and y_L in females. We assume that these four traits evolve via rare genetic mutations that have small sex- and condition-specific effects (*SI Appendix*, Appendix A.1 for methods).

We find that independent of females, males evolve to an evolutionary equilibrium where high- and low-condition individuals express

$$x_H^* = \frac{\gamma(1+\kappa_z v_H)}{\chi_m} \qquad x_L^* = \frac{\gamma(1+\kappa_z v_L)}{\chi_m}, \qquad [8]$$

respectively (Fig. 1*A* and *SI Appendix*, Appendix A.3.1 for derivation). So males of both condition classes express greater sexual traits when the strength of sexual competition (γ) is strong, and the intrinsic cost of male expression (χ_m) is low. Eq. **8** further shows that condition-dependent expression readily evolves with males in high condition showing larger traits than those in low condition at equilibrium (i.e., $x_H^* > x_L^*$ as $v_H > v_L$, Fig. 1*A* blue curves), and that the trait difference between high and low-condition males is most pronounced when the condition sensitivity of trait costs (κ_z) is high.

As males evolve toward their equilibrium (Eq. **8**) the harm inflicted to a female converges to

$$\eta^* = \frac{e^{-\gamma}\gamma}{\chi_m} k s_b \Big[P_m e^{-\alpha(v_H)}(1+\kappa_z v_H) + (1-P_m)e^{-\alpha(v_L)}(1+\kappa_z v_L)\Big]\frac{r}{1-r}. \qquad [9]$$

(*SI Appendix*, Eq. A-5). Eq. **9** offers three relevant insights. First, harm decreases with the intrinsic cost of male trait expression (χ_m), as this constrains the size of the equilibrium male trait. Second, harm is strongest when mating competition is of intermediate strength ($\gamma = 1$). This occurs because weak mating competition ($\gamma < 1$) leads to the evolution of a small trait in males and therefore weak harm. Conversely, intense mating competition ($\gamma > 1$) favors a large male trait, but due to costs of trait expression, this increase in trait size results in excess male mortality and in turn biases the sex ratio in the mating pool toward females. As a result, the level of harm is diminished (as recall harm is density-dependent, Eq. **7**). The third relevant insight from Eq. **9** is that harm increases with the proportion of males in high condition at birth, P_m (first term within square brackets). This occurs because high-condition males express larger phenotypes ($x_H^* > x_L^*$) and show greater baseline survival (i.e., increasing the number of males in the mating pool).

We find that provided the level of harm (η^*) at the male equilibrium (Eq. **9**) reaches a threshold, females evolve a response characterized by

$$y_H^* = \sqrt{\frac{\eta^*(1+\kappa_z v_H)}{\chi_f d}} - \frac{1}{d} \qquad y_L^* = \sqrt{\frac{\eta^*(1+\kappa_z v_L)}{\chi_f d}} - \frac{1}{d}, \qquad [10]$$

in high- and low-condition individuals, respectively (otherwise, if η^* is too low, females do not express their sexual traits; Fig. 1*A* grey and red curves; *SI Appendix*, Appendix A.3.2 for details). This is because expressing tolerance is costly and so females must experience enough damage to warrant investment into their phenotypes. Unsurprisingly, this phenotypic response increases in both condition classes with male harm (η^*) and decreases with the intrinsic cost (χ_f) of female trait expression. Female phenotypic expression is also greatest when the efficacy d of the female trait in mitigating male harm is intermediate (specifically when $d = 4\chi_f/\eta^*$). This is because weak efficacy (d small) disfavors investment into female trait as it leads to poor protection against harm, but strong efficacy (d large) means females need only produce a moderate trait to avoid damage from males. Finally, like males, females also evolve condition-dependent expression of their sexual traits, with high-condition individuals evolving larger phenotypes than low-condition individuals ($y_H^* > y_L^*$), according to κ_z.

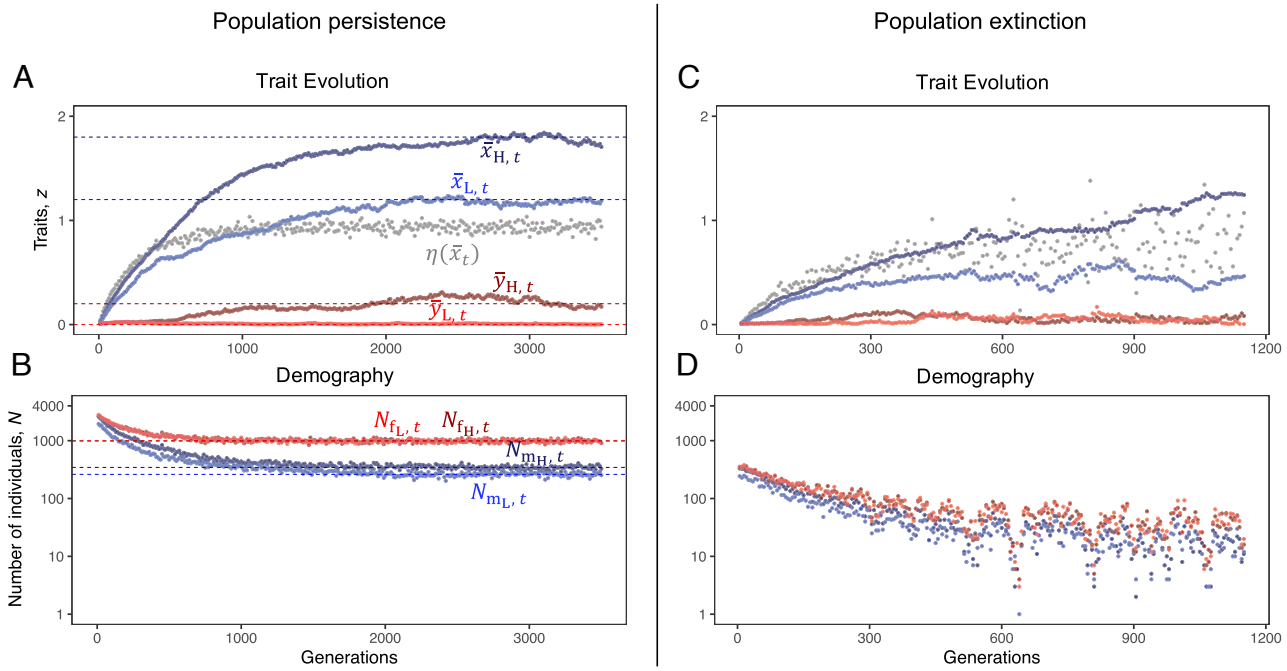

Fig. 1. Evolutionary dynamics of trait values and their population effect when condition is environmentally determined. (*A* and *B*) respectively, show trait expression and population sizes from a simulation where intrinsic female fecundity is high ($b = 50$, Eq. **5**), so that the population persists at an ecoevolutionary equilibrium. (*C* and *D*) respectively, show trait expression and population sizes from a simulation where intrinsic female fecundity is low ($b = 10$), so that the population goes extinct due to male harm (evolutionary suicide). In trait evolution panels (*A* and *C*), dots show trait values of high- and low-condition males and females (dark blue for high-condition male trait, $x_{H,t}$; light blue for low-condition male trait, $x_{L,t}$; dark red for high-condition female trait, $y_{H,t}$; light red for low-condition female trait, $y_{L,t}$). Dashed lines show expected trait expression at ecoevolutionary equilibrium (x_H^* and x_L^* from Eq. **8**, and y_H^* and y_L^* from Eq. **10**), using the same color scheme as for dots. In demography panels (*B* and *D*), dots show numbers of high- and low-condition males and females (dark blue for high-condition males, $N_{mH,t}$; light blue for low-condition males, $N_{mL,t}$; dark red for high-condition females, $N_{fH,t}$; light red for low-condition females, $N_{fL,t}$). Dashed lines show expected population sizes at ecoevolutionary equilibria (calculated by plugging phenotypic equilibria from Eqs. **8**–**10** into Eqs. A-1 and A-10). Values for all dots are calculated every tenth generation of individual-based simulations (*SI Appendix*, Appendix A.4 for details on simulation procedure). Other parameters used in all panels: $r = 0.5$, $\beta = 0.002$, $v_H = 0.8$, $v_L = 0.2$, $k = 2$, $\kappa_z = 1$, $\kappa_\alpha = 1$, $\chi_m = 1$, $\chi_f = 1$, $\gamma = 1$, $d = 0.8$, $P_m = P_f = 0.5$.

Strong Sexual Conflict Reduces Mean Fitness in High-Condition Populations. As males and females evolve toward their phenotypic equilibria, the population will either persist or be driven extinct, i.e., undergo "evolutionary suicide" (18, 60), due to the demographic effects of sexual conflict (Fig. 1 *B* and *D* and *SI Appendix*, Appendix A.2 for analysis). We find that which of these two outcomes unfolds depends on

$$\lambda^* = b(1-r)\left[P_f \times e^{-\frac{\eta^*}{1+dy_H^*} - c(v_H)y_H^* + \alpha(v_H)} + (1-P_f) \times e^{-\frac{\eta^*}{1+dy_L^*} - c(v_L)y_L^* + \alpha(v_L)}\right], \quad [11]$$

which is the expected number of newborn daughters produced by a female randomly sampled from the mating pool in a population at evolutionary equilibrium (i.e., where males express x_H^* and x_L^* given by Eq. **8** so that harm is given by Eq. **9**, and females express y_H^* and y_L^* given by Eq. **10**). This quantity λ^* is a proxy for mean fitness: the population persists when $\lambda^* > 1$ or goes extinct otherwise (when $\lambda^* \leq 1$). We further show in *SI Appendix*, Appendix A.2 that when $\lambda^* > 1$, the population stabilizes to a demographic equilibrium that increases proportionally with λ^*.

Together, Eqs. **8**–**11** reveal the impact of male and female trait evolution on demographics. In particular, Eq. **11** shows that population persistence becomes less likely as male harm η^* intensifies and thus is most compromised when intrinsic costs (χ_m) of male trait expression are weak, and mating competition is intermediate ($\gamma = 1$, Eq. **9**). Conversely, by negating the effects of harm, the evolution of the female trait (y_H^* and y_L^*) increases the likelihood that the population persists, especially when the female trait shows high efficacy in mitigating harm ($d \gg 0$).

Meanwhile, the effect of variation in condition on the ecoevolutionary dynamics of sexual conflict depends on how such variation is distributed across the sexes. In fact, we find that male and female conditions (through P_m and P_f) have antagonistic effects on population persistence (Fig. 2*A*). On the one hand, persistence becomes less likely as the proportion (P_m) of males in high condition increases, because this intensifies male harm. On the other hand, persistence becomes more likely when the proportion of females in high condition (P_f) increases, because high-condition females show greater baseline fecundity and pay weaker fecundity costs of trait expression than low-condition females.

The probabilities that males and females are in high condition may often be similar ($P = P_m = P_f$), such as where male and female conditions share a common environmental basis, e.g., because condition is determined by the availability of the same nutritional compounds. In this case, population persistence may become more or less compromised with the proportion (P) of individuals that are in high condition, depending on whether male harm or female fecundity increase more rapidly with condition (Fig. 2 *B* and *C*). Specifically, persistence becomes more likely with P if male trait expression leads to weak harm (small k) or baseline fitness is more sensitive to condition than trait costs (i.e., κ_z is small relative to κ_α, Fig. 2*B* purple area

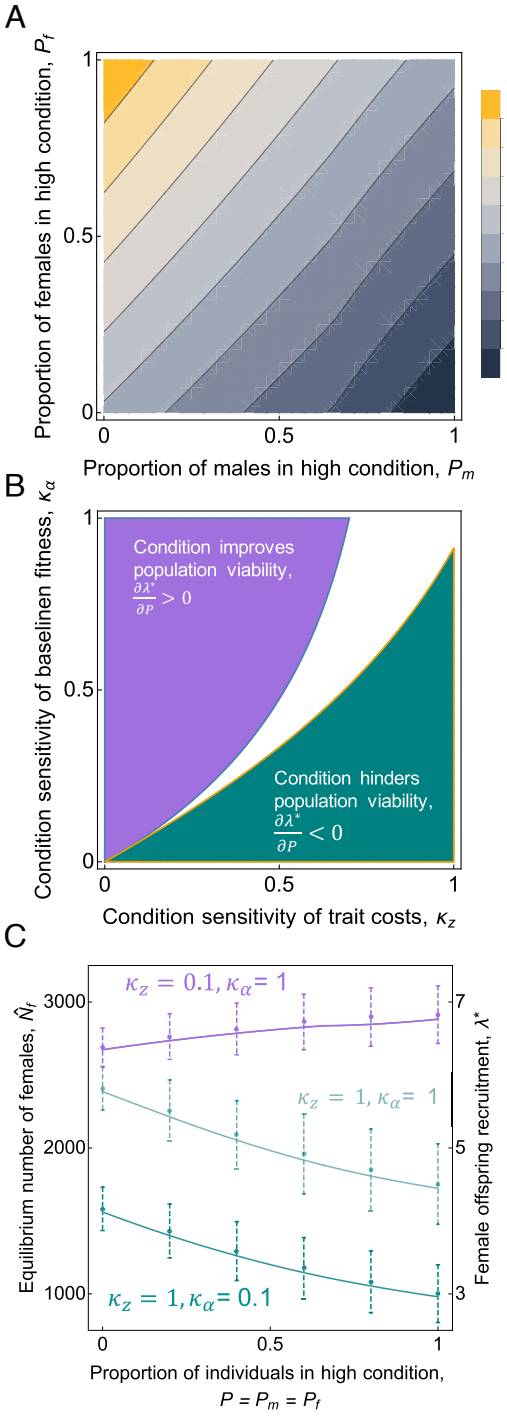

Fig. 2. Effects of male and female conditions on population viability. (*A*) shows recruitment of female offspring, λ^* (a proxy for population viability), as a function of the probabilities males (P_m) and females (P_f) are in high condition at birth, where $\kappa_\alpha = \kappa_z = 1$. (*B*) and (*C*) show the relationship between population viability and individual condition when male and female conditions are coupled ($P = P_m = P_f$), according to the condition sensitivity of sexual trait costs (κ_z, Eq. **3**) and baseline fitness (κ_α, Eq. **4**). In (*B*) the purple and green regions correspond to where population viability becomes improved ($\partial\lambda^*/(\partial P) > 0$) or compromised ($\partial\lambda^*/(\partial P) < 0$) with the proportion of individuals in high condition, respectively, while the white region corresponds to where population viability may increase or decrease with condition depending on the value of P. In (*C*) the purple curve shows λ^* and female population size, N_f, as a function of P when baseline fitness is more condition sensitive than sexual trait costs ($\kappa_\alpha > \kappa_z$), while green curves represent the opposite case (where $\kappa_\alpha < \kappa_z$). Dots refer to average female population size from corresponding individual-based simulations, while error bars represent the time SD across generations of the simulation (*SI Appendix*, Appendix A.4 for details). Other parameters used in all panels: $r = 0.5, b = 50, \beta = 0.002, v_H = 0.8, v_L = 0.2, \chi_m = 1, \chi_f = 1, \gamma = 1, d = 0.8, k = 1$.

and 2*C* purple curves). Meanwhile, persistence tends to become less likely with P when male trait expression generates strong harm (k is large) or where trait costs are strongly condition-dependent (i.e., κ_z is large, Fig. 2*B* green area, *C* green curves). When harming phenotypes show condition-dependent costs of expression, sexual conflict can thus lead to a misalignment between mean condition and mean fitness, so that populations with more individuals in high condition may be more likely to go extinct.

Good Genes Selection Exacerbates Sexual Conflict and Reduces Population Size. Our model so far assumes that condition is random with respect to genotype. However, condition is frequently viewed as a heritable and evolving trait with a wide genetic basis (30, 42, 55). In this case, condition is affected by a large number of loci across the genome and so is a reflection of an individual's genetic quality (i.e., the portion of variation in an individual's health that is attributable to additive genetic variation, see refs. 30 and 46).

To investigate the consequences of sexual conflict when condition is genetically determined, we used individual-based simulations that track the coevolution and demographic effects of sexual conflict phenotypes and genes encoding condition using SLiM v3 (61). These simulations depart from our analytical model in two main ways. First, condition, $0 \leq v \leq 1$, is now continuous and genetically determined by l_C loci. Each of these loci is diallelic with a wild-type and a mutant allele segregating in the population. Mutations changing a wild-type allele into a mutant allele occur at a rate μ_C, and vice-versa at a rate μ_B (where $\mu_B \ll \mu_C$). We assume that mutant alleles reduce condition and that the genotype-phenotype map for condition is multiplicative between and within loci, so that condition depends only on the total number of carried mutant alleles, see refs. 40–42. Specifically the condition of individual i carrying Ω_i mutant alleles is given by

$$v_i = (1-s)^{\Omega_i/10}, \qquad [12]$$

where s scales the effect on condition of a single mutation.

The second way our simulations departs from the initial model is that we now assume that male and female phenotypes, x and y, can show condition-dependent expression that changes linearly with v (following refs. 40 and 41). So x and y are each controlled by two quantitative genetic traits, τ_m and ρ_m in males and τ_f and ρ_f in females, such that

$$\begin{aligned} x &= \tau_m + \rho_m v \\ y &= \tau_f + \rho_f v. \end{aligned} \qquad [13]$$

Traits τ_m and τ_f thus contribute additive (i.e., condition-independent) genetic variance to the male and female phenotypes, while ρ_m and ρ_f control the sensitivity of x and y to condition v. Condition-dependent phenotype expression can therefore be envisaged as emerging from positive epistatic interactions between condition alleles and alleles encoding the sensitivity traits ρ_m and ρ_f. Each trait (τ_m, τ_f, ρ_m and ρ_f) is encoded by a single additive locus and evolves through mutations that occur at a rate μ_T with weak quantitative effects (with mean 0; i.e., we assume a continuum of alleles model, *SI Appendix*, Appendix B for details). All condition and trait loci are unlinked and segregate independently (note that as female tolerance does not influence male mating success here, we do not expect genetic covariances to develop between different traits, as in some models of female choice, e.g., refs. 42, 62–64).

We initiated simulations from a state of high condition ($v = 1$), i.e., fixed for wild type at all condition loci, and with all

individuals carrying genetic values of 0 for both gene copies at their trait loci (so $\tau_m = \tau_f = \rho_m = \rho_f = 0$). Simulations were run for 20,000 generations, which was sufficient time for evolutionary and demographic dynamics to converge to an equilibrium for all cases explored (*SI Appendix*, Appendix B for full details of the simulation procedure).

As a baseline, let us first consider what happens in the absence of sexual conflict (i.e., where $k = 0$), so that the male phenotype has no fitness consequences for females (see refs. 40–42, 53 for similar models), and where sexual trait costs show no sensitivity to condition (i.e., $\kappa_z = 0$). Additionally, since trait costs are condition independent, we also initially assume phenotypic expression is insensitive to condition ($\rho_m = \rho_f = 0$ in Eq. **13**), so that the sexual phenotypes are determined only by the additive genetic traits ($x = \tau_m$ and $y = \tau_f$). In this case, we see that due to the effects of mating competition, the male phenotype, (x) evolves (blue points in Fig. 3*A*, phase 1). However, since the male trait leads to no harm (as $k = 0$), the female trait does not evolve (consistent with our analytical results, Eq. **10**, red points in Fig. 3*A* phase 1). Condition (v) meanwhile, initially decreases as the population accrues mutations and eventually stabilizes to an intermediate level under mutation-selection balance (green points in Fig. 3*A*, phase 1). This evolution is accompanied by a moderate reduction in female population size (N_f) due to the effects of diminished condition on baseline fecundity, and a much larger drop in male population size (N_m) due to the additional mortality arising from male phenotypic expression (Fig. 3*B*, phase 1). We see that the evolutionary equilibrium for condition is greater when the condition sensitivity of baseline fitness, κ_α, is greater (dark curve in Fig. 3*C*). This is because κ_α determines the strength of "direct" natural selection (i.e., independent from the sexual traits, Eq. **4**) favoring high-condition individuals, so that condition is greater at mutation-selection balance when baseline fitness is strongly condition-dependent.

Second, we allow for the cost of the sexual phenotype to be condition-dependent ($\kappa_z > 0$) and for phenotypes to evolve condition-dependent expression (i.e., ρ_m, ρ_f are not fixed at 0). Individuals in better condition are thus able to produce larger phenotypes at smaller cost. This leads to two relevant evolutionary patterns. First, average condition v in the population is boosted (green points in Fig. 3*A* phase 2 and *C*). This is because sexual selection favors males expressing larger phenotypes who, when trait costs are condition-sensitive, tend to be in better condition (i.e., carrying fewer deleterious condition mutations). Sexual selection thus intensifies selection against condition mutations, increasing the genetic quality of the population. This has been referred to as the "good genes" effect of sexual selection, especially in the context of precopulatory female choice (42, 43, 55, 65, 66). Second, males evolve larger phenotypes with both phenotype size (x) and the condition sensitivity of

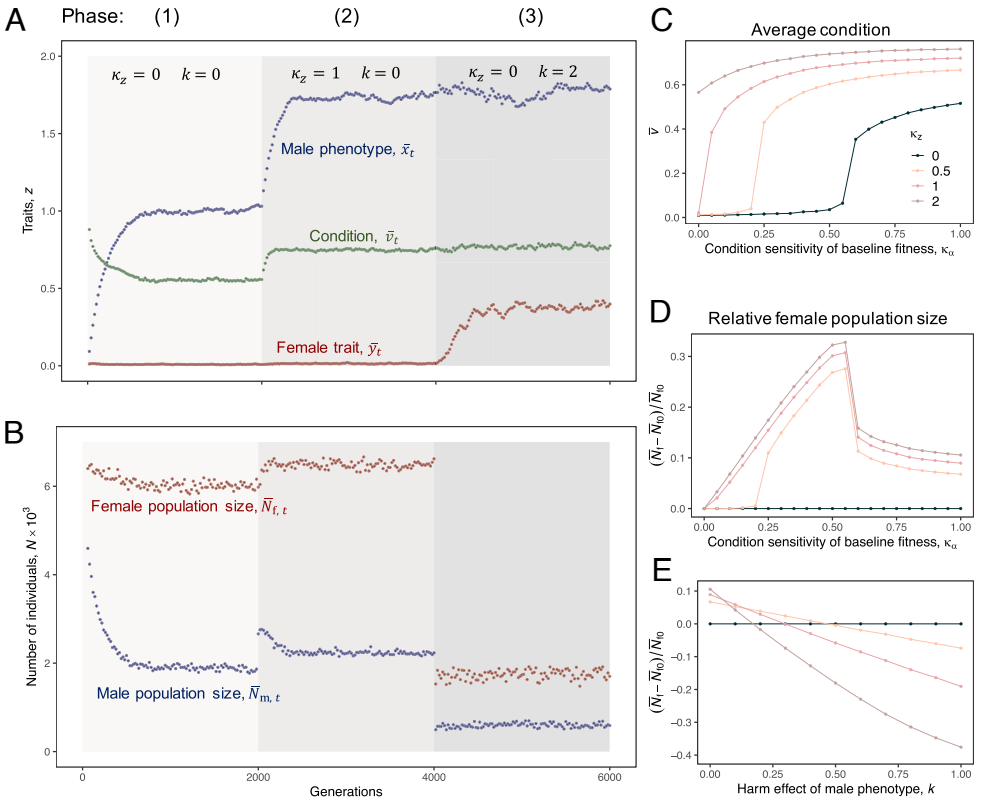

Fig. 3. Ecoevolutionary dynamics of sexual conflict and genetic condition. (*A*) and (*B*) show trait averages (condition, $\bar{v}_t$, and male, $\bar{x}_t$, and female, $\bar{y}_t$, sexual phenotypes) and population sizes ($N_{m,t}$ and $N_{f,t}$) through time, respectively. Dynamics are plotted for three different phases of parameter combinations, in 1) sexual phenotypic expression is condition independent and male harm is absent ($\kappa_Z = k = 0$), in 2) sexual phenotype expression costs become condition sensitive ($\kappa_Z > 0$), and in 3) male phenotype expression leads to male harm ($k > 0$). (*C*) shows average condition ($\bar{v}$) in the absence of male harm ($k = 0$), as a function of the condition-sensitivity of baseline fitness κ_α, while different curves represent different strengths of trait cost condition sensitivity κ_Z. (*D* and *E*) show the average number of females for different treatments ($\bar{N}_f$) relative to the case when $\kappa_Z = 0$ ($\bar{N}_{f0}$), where curves show $(\bar{N}_f - \bar{N}_{f0})/\bar{N}_{f0}$. (*D*) shows this ratio as a function of κ_α in the absence of harm ($k = 0$), while (*E*) as a function of the strength of male harm, k, assuming $\kappa_\alpha = 1$. Different curves again represent different strengths of sexual trait cost condition-sensitivity. Averages are taken over the last 10,000 generations of a simulation (section for details). Other parameters used in all panels (unless otherwise stated): $r = 0.5$, $s_b = e^{\frac{1}{1+\kappa_\alpha}}$, $\beta = 0.002$, $b = 50$, $\chi_m = 1$, $\chi_f = 1$, $\alpha_0 = 1$, $\gamma = 1$, $d = 0.8$, $\kappa_\alpha = 1$, $s = 0.5$.

phenotype expression (ρ_m) increasing with κ_z (blue points in Fig. 3*A* phase 2, *SI Appendix*, Fig. S2*A*). This occurs because males are generally in better condition (due to the good genes effect) and so pay increasingly cheap costs of trait expression as these become more condition-sensitive (i.e., as κ_z increases). Females, however, still do not evolve due to the absence of male harm (green points in Fig. 3*A* phase 2). As a result of male evolution and subsequent condition improvement via the good genes effect, we see an increase in both male and female numbers (Fig. 3*B* phase 2). This occurs because elevated condition in males and females leads to increases in baseline fecundity and survival. Thus, we see that in the absence of sexual conflict ($k = 0$), sexual selection has a positive effect on population size when trait costs are condition-dependent (Fig. 3*D*).

Interestingly, the demographic boost provided by sexual selection is most significant when the condition sensitivity of baseline fitness is intermediate (e.g., $\kappa_\alpha \approx 0.5$ in Fig. 3*D*). To understand this, consider the case when the condition dependence of baseline fitness is weak. Here, sexual selection has a strong and positive impact on condition, as direct natural selection is weak and deleterious condition mutations will accumulate in the absence of the good genes effect (κ_α is small, Fig. 3*C*). However, this condition boost translates into a weak demographic benefit because baseline female fecundity is not condition-sensitive. Conversely, when baseline fitness shows strong condition dependence (κ_α is large, Fig. 3*C*), natural selection is sufficiently intense to purge mutations (and so maintain a low mutation load), so that the good genes effect has a limited influence on condition. In other words, the good genes effect is strong when condition has little effect on baseline fitness/population size (κ_α small) but is weak when baseline fitness/population size is strongly condition sensitive (κ_α large). This means that the good genes effect affords a modest improvement to mean fitness in our model, thus posing a significant constraint on the demographic benefits that can be provided by sexual selection.

As a third and final extension, we allow for the male phenotype to cause harm to females ($k > 0$). This leads the female sexual phenotype to evolve (red points in Fig. 3*A* phase 3 and *SI Appendix*, Fig. S2*B*), as females are selected to mitigate the damage they experience from males, and also causes a reduction in population size due to the negative effect of harm on female fecundity (Fig. 3*B* phase 3). Both of these effects are stronger when trait costs are condition sensitive (i.e., $\kappa_z > 0$). Specifically, when κ_z is large, females on average produce larger phenotypes because of i) the reduced costs of female trait expression and because ii) males will also express large traits, leading to strong male harm. Moreover, mirroring males, females phenotypes also evolve condition-sensitivity (i.e., $\rho_f > 0$) in proportion to κ_z (*SI Appendix*, Fig. S2*B*). As such, condition-dependent trait costs ($\kappa_z > 0$) lead to the evolution of larger and more condition-dependent male and female phenotypes (due to cheaper trait expression), exacerbating sexual conflict. In turn, this intensified conflict—in particular, the high levels of male harm—leads to an increasingly sharp drop in population size with k when κ_z is large (Fig. 3*E*).

In fact, the negative demographic consequence of intensified sexual conflict can completely offset the demographic benefit provided by the good genes effect. Specifically, past a critical threshold for k, condition dependence (κ_z) has a net-negative effect at the population level (Fig. 3*E*). This threshold depends on the condition-sensitivity of baseline fitness and traits costs (Fig. 3*E* and *SI Appendix*, Fig. S3), but typically occurs at relatively low levels of k, especially when trait costs are strongly condition-dependent (e.g., $\kappa_z \geq 1$). Thus, a moderate level of male harm can be sufficient for sexual selection to reduce population size despite improving average individual condition. In other words, while good genes selection improves mean condition, we find that its population-level consequences are frequently negative due to the inflammatory effect of high-condition genotypes on the strength of sexual conflict.

Discussion

Here, we have integrated key aspects from two groups of models: i) sexual conflict (e.g., refs. 18, 22–24, 27, 29) and ii) condition dependence (e.g., handicap models, 40–42, 48). In doing so, we have uncovered some surprising insights into how sexual selection shapes trait evolution and population demography.

Our analyses indicate that, like other sexually selected traits (30, 41, 42), male harm and female tolerance readily evolve condition dependence such that male and female investment into sexual conflict increases with condition. Populations in better condition thus experience more intense conflict, which impairs offspring recruitment and can jeopardize population persistence. In particular, if the severity of male harm increases more strongly with condition than does baseline female fecundity, we observe a counter-intuitive pattern whereby high mean condition is associated with low mean fitness. Such a negative association between condition and fitness is especially likely to emerge where condition has a genetic basis in males and females, as selection favors "good genes" that improve individual condition but also increase the intensity of male harm.

Our results contrast with the common view that sexual selection on good genes also improves mean fitness (43, 55, 65, 66) and mitigates the costs of sexual conflict (42). This is because for the good genes effect to work, that is, for sexual selection to increase mean condition, sexual traits must show appreciable condition-sensitivity (e.g. large κ_z in our model). In other words, sexual selection acts strongly on condition when good genes confer large increases in the size of sexual traits that males can afford to express. Therefore, the good genes effect is strong where a reduction in mutation load is associated with a significant increase in male sexual trait expression (e.g. in Figure 3*A* when $\kappa_z = 1$, an increase in condition, green points, is associated with a much larger increase in male trait size, blue points, see also figure 1a in 42). Importantly, the repercussions of the good genes effect for male trait size are typically larger than for baseline female fecundity. This is because if condition genes greatly improve absolute baseline survival or fecundity, then natural selection should be intense enough to maintain them at relatively high frequency irrespective of the action of sexual selection (Figure 3*C* see difference between orange curves and black curve decreases with κ_α). The variation in genetic condition that is available for sexual selection to act upon is therefore limited, constraining the influence of the good genes effect on female fecundity relative to male trait expression. Altogether this means that, when male traits beget harm, the good genes effect has much greater scope to influence demography through sexual conflict than through baseline female fitness. Population benefits of good genes are thus easily reversed by their side-effect in exacerbating antagonistic male-female interactions.

In highlighting the relationship between condition and male harm, our results have implications for empirical work, in particular experimental evolution approaches to unpicking the

consequences of sexual selection. Some studies have identified population-level benefits of sexual selection by tracking variation in condition (inferred through condition-correlated traits, e.g., body size, male mating success, offspring viability, or immune function) as a proxy of population viability (e.g., refs. 67–70 reviewed in ref. 71). Our results indicate such traits are poor indicators of population viability in species exhibiting male harm. Without measuring mean fitness, these studies therefore provide ambiguous evidence for an advantage to sexual selection. Indeed, when experimental studies do track more direct measures of mean fitness (such as female fecundity), they typically detect a weaker increase than for indirect measures such as body condition, mutation frequencies, or male mating success (71, 72). For example, a study in fruit flies found that sexual selection was associated with lower mutation load but also diminished offspring recruitment, which was attributed to the effects sexual conflict (73). Furthermore, a number of experimental studies have found evidence that sexual selection improves persistence in populations experiencing environmental stress (e.g., temperature variation in refs. 74–76, reviewed in refs. 77 and 43). This has been attributed to the idea that sexual and natural selection should be more closely aligned in such cases, i.e., that condition genes are more likely to benefit both sexual and nonsexual fitness when both sexes are poorly adapted (78–80). However, our analyses provide an additional explanation here: Environmental stress, by lowering male condition, may reduce the expression of male harm and so increase mean female fitness. More generally, we suggest that condition dependence provides a mechanism for the strength of sexual conflict to diverge in different environments a pattern, for example, observed in drosophila (81), and water striders (82).

To produce tractable results, our baseline model makes a number of simplifying assumptions. We assumed a well-mixed population (i.e., no effects of spatial subdivision); the absence of genetic constraints that affect condition- or sex-specific phenotypic expression; that males direct their mating attention indiscriminately toward high- and low-condition females; and that females express a tolerance trait that mitigates harm but does not impact male mating success. Relaxing these assumptions may alter the ecoevolutionary dynamics of sexual conflict through kin selection (27, 29), the strength of natural selection on females (70, 83), and the presence of coevolutionary intersexual arms races that escalate sexual conflict (24, 28, 84). Moreover, we considered two forms of condition variation, with condition either 1) a purely environmental trait or 2) purely genetic, while in nature, condition may frequently be an intermediate of these two scenarios (46). To relax some of these assumptions, in *SI Appendix*, Appendices *C* and *D* we also analyzed extended versions of our baseline model that allowed us to consider alternative bases of condition and multiple ecological settings for sexual conflict. In certain cases, such as where condition-dependent plasticity is prevented by genetic constraints, or where genotype × environment interactions depress male condition, we find that male harm can be diminished, so aiding population persistence (*SI Appendix*, Appendix D). However, our main results remain robust, that is, sexual conflict generates a negative relationship between average condition (e.g., good genes) and population size in the vast majority of the cases considered. In fact, we find that the costs of condition-dependent harm for population persistence may be strongly exacerbated in many common scenarios of sexual conflict, such as where female resistance drives evolutionary arms races, or where male and female conditions are encoded by different genes. We therefore suggest that the effects of condition variation found in our model will be general features found across most forms of sexual conflict (*SI Appendix*, Appendix D for a deeper discussion of extensions to our baseline model and of modeling choice and implications).

Finally, while our study was presented in the context of sexual conflict, our results are also more broadly applicable to the evolution of competitive traits with demographic effects. For example, antagonistic social interactions commonly drive the evolution of weapon phenotypes in both sexes (85, 86). These traits, which are often condition dependent, can influence the survival of interacting conspecifics (12, 13, 20, 33, 87, 88), and so their expression may also diminish mean fitness in high-condition populations.

Conclusion

An outstanding question in evolutionary biology is whether sexual selection is beneficial or detrimental to populations (reviewed in refs. 71, 89–91). The current view is that the impact of sexual selection depends on the balance between two opposing forces: the genetic benefits of the good genes effect (55) versus the costs of sexual conflict (16, 18) and that understanding the net outcome of these forces requires only empirical data (71, 73). A key insight from our model is that the genetic benefits of sexual selection and the costs of sexual conflict can in fact interact, as male harm causes the fixation of good genes to impair female fitness. Such feedback has negative demographic consequences, even driving extinction when males inflict severe damage onto females. Furthermore, because social traits, such as aggression (92), boldness (93) and courtship effort (94) are often condition-dependent (reviewed in refs. 87, 95 and 96), the good genes effect will exacerbate agonistic interactions between competing individuals, and in turn, offset benefits to populations.

Data, Materials, and Software Availability. There is no data associated with this paper, but SLiM simulation code used to produce figure 3 and *SI Appendix*, figures is available at https://github.com/eoflintham/Male-harm-offets-the-demographic-benefits-of-good-genes.

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