doi:10.1111/evo.12272

THE ECOLOGY OF SEXUAL CONFLICT: BACKGROUND MORTALITY CAN MODULATE THE EFFECTS OF MALE MANIPULATION ON FEMALE FITNESS

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Received April 29, 2013
Accepted September 7, 2013

Sexual and parental conflicts can arise because males benefit by inducing elevated reproductive effort in their mates. For females, the costs of such manipulation are often manifested later in life, and may therefore covary with female life expectancy. Here, I outline a simple female life-history model where female life expectancy reflects extrinsic mortality rate, and elevated reproductive effort causes accelerated senescence. Using this model, I show that variation in extrinsic mortality rate can modulate the magnitude and sign of fitness effects that male manipulation has on females. This result has several interesting implications. First, it suggests that the fitness effects of sexual interactions can depend on ecological factors, such as predation, that influence life expectancy. Second, if mortality risk is condition-dependent but reproductive effort is not fully optimized in relation to individual condition, then sexual conflict intensity may increase with individual condition, selecting for condition-dependent reproductive strategies. Third, if males vary in manipulativeness, then the fitness effects of mating with a given male phenotype may depend on both female condition and extrinsic mortality rate. Fourth, life span extension in the laboratory can lead to overestimation of sexual and parental conflicts. Life expectancy may therefore be a key factor in sexual coevolution.

KEY WORDS: **Extrinsic mortality, life span, parental conflict, predation, senescence, sexually antagonistic selection.**

The role of ecology is implicit in sexual coevolution theory, in that the efficacy of strategies pursued by each sex, and their effects on fitness in the other sex, are both likely to depend on ambient conditions. However, the links between ecology and sexual coevolution have rarely been defined or examined explicitly, and remain poorly understood. For example, it has long been recognized that secondary sexual traits such as signals and weapons impose viability-related costs that tend to limit their exaggeration (Fisher 1930; Rowe and Houle 1996; Jennions et al. 2001), but the nature of such costs remains poorly known in most systems (Jennions et al. 2001; Kotiaho 2001; McCullough et al. 2013). Similarly, the efficacy of male sexual strategies may depend on ecological parameters such as signal permeability (Endler 1992; Boughman 2001). Even less is known about how viability selection acts on morphological and behavioral resistance traits in females, or how ecological factors modulate the effects of male reproductive strategies on female fitness. However, mounting empirical evidence and theory points to the importance of ambient environment in determining the magnitude and even sign of such effects (Fedorka and Zuk 2005; Hardling and Kaitala 2005; Maklakov et al. 2006; Fricke et al. 2009a, 2010; Gay et al. 2010; McLean et al. 2010; Garcia and Lemus 2011).

Here, I ask whether the intensity of sexual conflict can depend on female life expectancy and, thus, on ecological parameters that determine the extrinsic mortality rate (i.e., mortality resulting from factors external to the organism, such as predation).

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Although the consequences of variation in background mortality for the evolution of life-history strategies have been examined both theoretically and empirically (e.g., Reznick et al. 1990; McNamara et al. 2004; Reznick et al. 2006; Chen and Maklakov 2012), the role of background mortality in shaping sexual interactions has received little attention (although see Rowe 1994; Han and Jablonski 2010). I examine the link between extrinsic mortality and sexual conflict, and show that extrinsic mortality may be an important parameter in sexual coevolution.

In polyandrous species, female reproductive value is an exploitable resource over which males compete (Parker 1979; Arnqvist and Rowe 2005), and male reproductive strategies can affect many aspects of female life history (Promislow 2003; Wedell et al. 2006; Bonduriansky et al. 2008). Males are selected to pursue selfish strategies toward their mates because a male's fitness depends only on his short-term fitness gain from the mating, before the female remates with a different male. Selection will therefore favor manipulative male strategies that cause females to invest a disproportionate amount of their reproductive value in a given mating through the production of many offspring, or allocation of a large quantity of resources to each offspring (Chapman et al. 1995; Arnqvist and Nilsson 2000; Wedell et al. 2006), even though such male manipulation can reduce female lifetime reproductive output (Fig. 1). Overinvestment of resources in one mating leaves less to invest in future matings (Stearns 1989, 1992), and elevated reproductive effort can lead to accelerated ageing (Kirkwood 1977; Kirkwood and Rose 1991; Orell and Belda 2002). The costs of male manipulation for females are expected to be mostly latent (i.e., manifested as accelerated senescence), because immediate costs (e.g., female death or impaired reproduction soon after mating) would reduce the male's own fitness, whereas latent costs are unlikely to do so. Indeed, males may evolve to minimize the costs incurred by females if such costs reduce male fitness (Maklakov et al. 2005; Reinhardt et al. 2009). Empirical studies have provided evidence of latent costs to females of both reproduction (Gustafsson and Part 1990; Orell and Belda 2002; Nussey et al. 2006) and mating itself (Tatar et al. 1993; Chapman et al. 1998; Rogina et al. 2007). The intensity of sexual conflict may therefore reflect the balance between the benefits to females of male-induced elevation of reproductive output and costs of reduced fecundity later in life (Arnqvist and Nilsson 2000; Maklakov and Lubin 2004; Edward et al. 2010).

A well-known manifestation of this type of sexual conflict is the "toxic ejaculate" of *Drosophila melanogaster* males (Chapman et al. 1995). At least one ejaculate component—the sex peptide—appears to induce an elevated reproductive rate in females after mating, benefiting the male by enhancing fertilization success before the female remates with a different male (Wigby and Chapman 2005; Fricke et al. 2009b). However, such short-term elevation of female reproductive effort above the female's optimum level can impose costs that reduce female lifetime reproductive output (Chapman et al. 1995; Fricke et al. 2009a, 2010). Indeed, male ejaculates may harm females even when accompanied by nuptial gifts (Arnqvist and Nilsson 2000; Wedell et al. 2008; although see Perry and Rowe 2008). In effect, by overstimulating the female reproductive system, manipulative males truncate female reproductive life span.

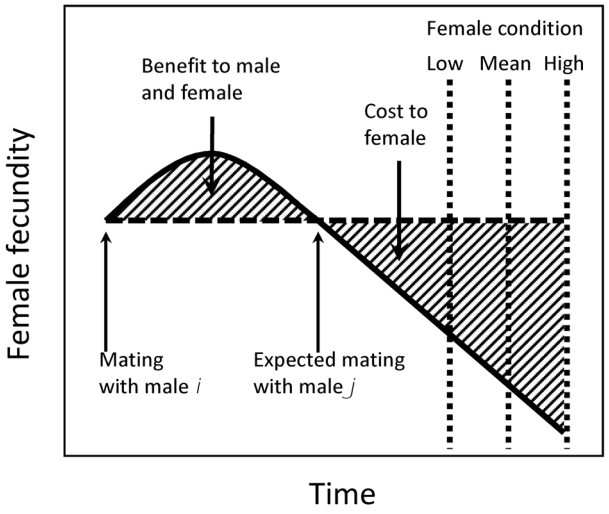

Figure 1. Sexual conflict over female reproductive allocation. The dashed horizontal line represents a female's optimum reproductive effort, whereas the solid line represents her realized reproductive effort following mating with manipulative male *i*. Male *i* induces an increase in the female's reproductive effort (shaded area above the dashed line) that maximizes his fitness gain before the female's expected remating with male *j*. This increase in reproductive effort also represents a fitness gain for the female, but this gain may be offset by latent costs in the form of reduced reproductive output later in life (shaded area below the dashed line) that are paid by the female but usually not by male *i*. All else being equal, the magnitude of these latent costs depends on female life expectancy (horizontal dotted lines), which may vary with habitat patch or female condition. This figure is modified from Bonduriansky et al. (2005).

Another hypothesized manifestation of the same type of sexual conflict is an epigenetically controlled parent-of-origin (genomic imprinting) effect on offspring growth. According to the "kinship" or "parental conflict" model, selection favors increased expression in offspring of paternally inherited alleles involved in the extraction of maternal resources before or after birth, even if the increased resource expenditure comes at a cost to the female or her future offspring, because the male's alleles are unlikely to be represented in the female's subsequent broods (Moore and Haig 1991; Haig 2000). Like a toxic ejaculate, this epigenetic paternal strategy can enable males to exploit the reproductive value of their mates to maximize the number and/or quality of offspring produced from each mating, and overinvestment in a single

reproductive bout is thought to reduce female fitness (Haig 2000). Such paternal manipulation of gene expression in offspring may evolve in taxa where females provision their offspring after fertilization (Moore 2001).

All else being equal, the mean magnitude of latent costs paid by females may be expected to reflect female life expectancy: the longer a female can expect to live, the greater her potential to lose fitness through accelerated senescence. This influence of female longevity suggests a link between sexual conflict and ecology. In captive animals, longevity largely depends on internal processes (i.e., rate of somatic deterioration); whereas, in natural populations, longevity is largely determined by extrinsic (background) mortality sources such as predation (Comfort 1979; Finch 1990; Kawasaki et al. 2008). Consequently, the intensity and even presence of sexual conflict may depend on female life expectancy and the ecological factors that influence extrinsic mortality rate. Below, I explore this relationship using a simple model.

A Model of Female Life History

I assume that population-mean female reproductive effort has evolved under the prevailing extrinsic mortality rate, and examine the consequences of perturbations of female life expectancy from the level to which females are adapted. Such perturbations can reflect temporal of spatial variation in extrinsic mortality or, alternatively, within-population (e.g., condition dependent) variation in mortality risk. My analysis thus focuses on the consequences for sexual/parental antagonism of short-term, patch-specific, or condition-dependent variation in female life expectancy.

I assume that female age-specific fecundity, f_t, depends on reproductive effort, N, offset by age-specific costs, c_t,

$$f_t = N - c_t. \tag{1}$$

f_t can be interpreted as total age-specific reproductive output (i.e., the product of offspring size and number). Age-specific costs are modeled as a logistic function, whereby costs increase with female age, t, at a rate proportional to female reproductive effort, N:

$$c_t = \frac{d}{d + (1-d)\, e^{-aNt}} N, \tag{2}$$

where e is Euler's constant and a and d are parameters that determine cost function shape. As a and d increase (assuming $d \ll 1$), age-specific fecundity declines more rapidly with age for a given level of early-life reproductive effort, N, but variation in a and d has no qualitative effect on results. Reproduction is assumed to begin at age $t = 1$, and $f_1 \approx N$, so N is equivalent to the early-life reproductive effort. The cost function (2) thus penalizes increased early-life reproductive effort with accelerated ageing (Fig. 2A), capturing the trade-off between early-life reproductive effort and somatic maintenance that is assumed by life-history theory (Williams 1957; Kirkwood 1977; Kirkwood and Rose 1991).

Ageing is modeled here as a decline in fecundity with age (reproductive ageing), and the model does not explicitly incorporate a decline in age-specific survival probability (actuarial ageing). However, a decline in mean fecundity with age can be interpreted as reflecting the effects of both reproductive and actuarial ageing (i.e., the product of age-specific fecundity and survival probability).

Female fitness (i.e., lifetime reproductive output) is the sum of female fecundity at all ages until death,

$$W = \sum_{t=1}^{s} f_t, \tag{3}$$

where s is female longevity, or maximum age that females attain under the prevailing level of extrinsic mortality. Although female mortality is not modeled here as a probabilistic process, the parameter s can be considered to represent female life expectancy in the current environment.

The model yields an optimum reproductive effort, N^*, for any combination of other parameter values (Fig. 2B). N^* is the value of N that maximizes mean female fitness by striking an optimum balance between early-life reproductive effort and latent costs, and I assume that mean female reproductive effort is close to this optimum. I also assume that females have adapted to the mean level of male manipulation (if any) that they have experienced over many generations, so that N^* can include the mean male effect on female fecundity. Consistent with life-history theory, N^* increases as life expectancy declines (Fig. 2B). Within populations, if females in low condition, or occupying risky habitat patches, have below-average life expectancies (Fig. 1), then their optimal reproductive effort, N_L^*, will exceed the population-mean optimum (i.e., $N_L^* > N^*$). Conversely, for females in high-condition, or occupying low-risk habitat patches, optimal reproductive effort, N_H^*, should be less than the population-mean (i.e., $N_H^* < N^*$). However, I assume that all females express the population-mean optimum reproductive effort, N^* (see Discussion).

Analysis

Imagine a male phenotype that induces a change, M, in female reproductive effort, and represents a deviation of male manipulativeness (i.e., the extent to which a male increases the reproductive effort of his mate via ejaculate components that stimulate the female reproductive system, or via a parent-of-origin effect on gene expression in offspring) from the mean level of manipulativeness (if any) experienced by females over many generations. A positive value of M can be interpreted as a highly manipulative genetic

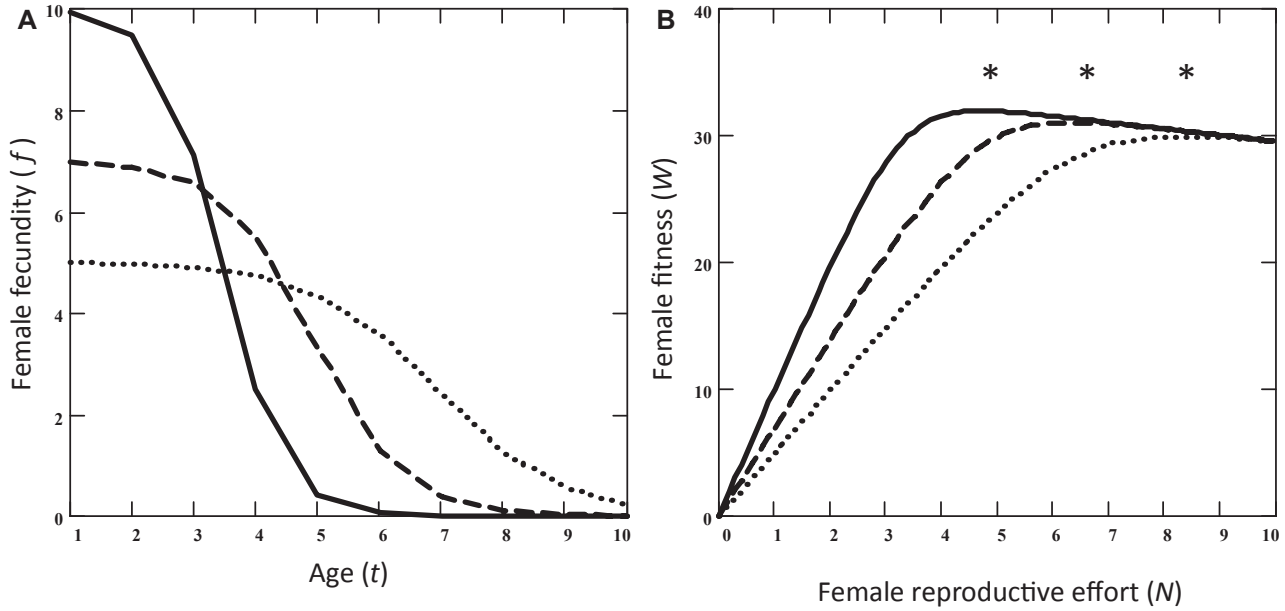

Figure 2. **(A) Female age-specific fecundity (f_t) for three levels of early-life reproductive effort: $N = 10$ (solid line), $N = 7$ (dashed line), and $N = 5$ (dotted line). (B) Female fitness (W) as a function of early-life reproductive effort (N) for three levels of life expectancy: $s = 10$ (solid line), $s = 7$ (dashed line), and $s = 5$ (dotted line). Optimum reproductive effort (N^*) at each life expectancy is indicated by *. Other parameter values: $s = 10$, $a = 0.2$, and $d = 0.001$.**

variant that appears in the population via mutation or gene flow. For simplicity, I assume that the effect of M is additive, and that M is independent of female condition (see Discussion). With the incorporation of this male effect, equations (1) and (2) become

$$f_t = (N + M) - c_t \tag{4}$$

and

$$c_t = \frac{d}{d + (1 - d)\, e^{-a(N+M)t}} (N + M). \tag{5}$$

By definition, when $N = N^*$, any nonzero value of M reduces mean female fitness by displacing females from their optimum reproductive effort, illustrating the toxic ejaculate effect (Fig. 3).

However, female fitness also depends on life expectancy, s. Given a change, P, in life expectancy, female lifetime reproductive output becomes

$$W = \sum_{t=1}^{s+P} f_t. \tag{6}$$

A negative value of P can represent the invasion of a new predator, or elevated predation rate within a particular habitat patch. If background mortality rate depends on individual condition, then P can also represent a condition-dependent deviation in life expectancy from the population mean (e.g., extent to which life expectancy is reduced by low condition).

The male effect, M, on female reproductive effort, and the change, P, in female life expectancy, are thus modeled as factors that perturb the female life history away from the optimum that has evolved over many generations. The question of interest is whether a highly manipulative male phenotype ($M > 0$) that, all else being equal, reduces female fitness, can become less harmful or even

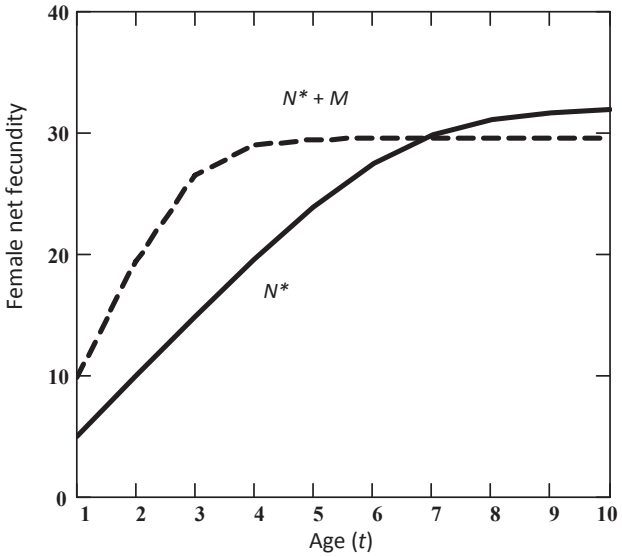

Figure 3. **Male manipulation reduces female fitness. Female net (cumulative) fecundity is plotted as a function of age (t) at the optimum early-life reproductive effort ($N = N^*$, solid line), and when male manipulation elevates female reproductive effort (N) above the optimum by $M = 5$ (dashed line). Other parameter values: $s = 10$, $a = 0.2$, and $d = 0.001$.**

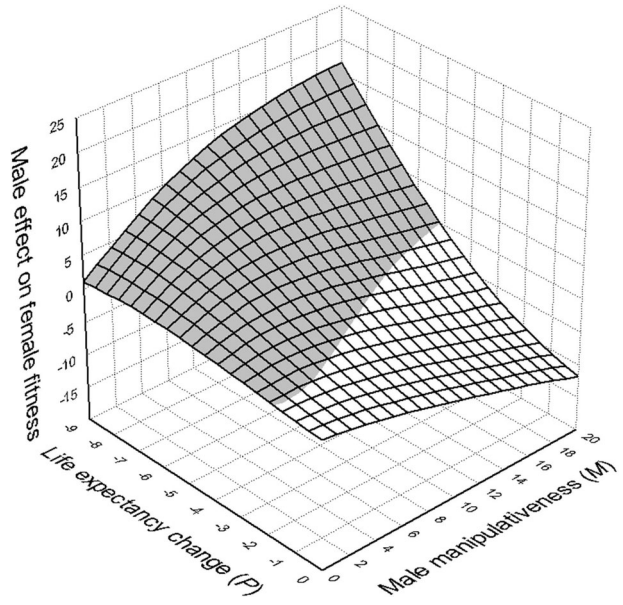

Figure 4. Female life expectancy modulates the effect of male manipulation on female fitness. For each value of *P* (i.e., extent of reduction in female life expectancy by ecological factors or low condition), female fitness in the absence of increased male manipulation ($M = 0$) was subtracted from female fitness at higher levels of male manipulation ($M > 0$) (other parameter values: $s = 10$, $a = 0.2$, and $d = 0.001$). These values are plotted as a response surface showing how varying levels of male manipulation affect female fitness when female life expectancy is reduced to varying degrees. Positive values (gray) represent a beneficial effect of increased male manipulation on female fitness (inequality (7) is satisfied for negative values of *P*), whereas negative values (white) represent a harmful effect of male manipulation. Male manipulation thus becomes less harmful when female life expectancy is reduced, relative to the level to which females are adapted. An increase in female life expectancy (positive *P*) would have the opposite effect, making male manipulation more harmful.

beneficial for females if life expectancy is reduced below the level to which females are adapted (i.e., $P < 0$). Substituting equations (4) and (5) into equation (6) yields the following inequality (where N^* represents optimum reproductive effort when $M = P = 0$) that must be satisfied for this to occur:

$$\sum_{t=1}^{s+P}\left[N^* - \frac{d}{d+(1-d)\,e^{-aN^*t}}N^*\right]_t \leq \sum_{t=1}^{s+P}\left[(N^*+M) - \frac{d}{d+(1-d)\,e^{-a(N^*+M)t}}(N^*+M)\right]_t. \quad (7)$$

Simplifying this inequality does not yield an interpretable analytical solution. However, numerical analysis shows that inequality (7) can be satisfied for a range of values of M and P (Fig. 4). In the discrete-time model developed above, when P is near zero, any $M > 0$ is harmful to females. However, for larger negative values of P, low values of M are beneficial for females, whereas higher values of M are harmful. At the largest negative values of P, any $M > 0$ is beneficial for females.

Increasing negative values of P can represent either increasing mean female mortality rate, or increasing mortality risk of individual females relative to the population mean (e.g., associated with a risky habitat patch or low individual condition). The above results thus show that highly manipulative males will increase female fitness if life expectancy is reduced sufficiently (moderate or high negative values of P), either by ecological factors that elevate background mortality rate, or by low individual condition. Conversely, if mean female life expectancy, or the life expectancy of an individual female within a population, is close to the historical average to which the population is adapted ($P \approx 0$), then highly manipulative males will be harmful. A symmetrical effect will occur in the converse case: if mean female life expectancy is increased ($P > 0$), a male effect that was formerly beneficial on average (i.e., brought females closer to N^*) can become harmful (Fig. S1). Similarly, within a population, a value of M that increases fitness of females whose life expectancy is below-average (high negative values of P) is less beneficial for females whose life expectancy is closer to the mean ($P \approx 0$), because $N_L^* > N^*$ (Fig. 4). Correspondingly, for females whose life expectancy is above-average, fitness will decrease with increasing M because $N_H^* < N^*$. Changing cost function shape (i.e., parameters a and d) does not alter the qualitative conclusions of this analysis (Figs. S2, S3).

Discussion

In polyandrous species, selection favors manipulative male strategies that induce females to elevate their reproductive effort above the female optimum via "toxic ejaculates" that elevate oviposition rate (Chapman et al. 1995; Arnqvist and Nilsson 2000; Wigby and Chapman 2005; Wedell et al. 2006) or parent-of-origin (genomic imprinting) effects that induce elevated resource investment in offspring (Moore and Haig 1991; Haig 2000). Although such male strategies have been assumed to harm females by reducing their life span and lifetime reproductive output (although see Reinhardt et al. 2009), recent studies suggest that the costs to females are strongly context dependent (Fricke et al. 2009a, 2010; Edward et al. 2010). Here, I show that ecological factors that determine the background mortality rate, such as predation pressure, can modulate transient or local variation in the mean intensity of sexual and parental conflict experienced by females. Similarly, within populations, condition-dependent or patch-specific variation in life expectancy can be associated with variation in the intensity of sexual and parental conflict.

Studies on a diverse array of species suggest that mortality rate can fluctuate considerably on ecological time scales, vary

spatially, and reflect individual condition in natural populations. In several long-lived mammal species, the mean coefficient of variation for interannual survival probability of adults is around 10% (Gaillard et al. 2000; Gaillard and Yoccoz 2003). In a long-lived shorebird, the Eurasian Oystercatcher (*Haematopus ostralegus*), survival probability of breeding adults varied among years from about 65% to nearly 100% (van de Pol et al. 2010). In a population of snapping turtles (*Chelydra serpentina*), interannual survival probability for adult females dropped from 97% to 55% following an increase in the size of the local population of otters (Brooks et al. 1991). There is evidence of spatial variation in background mortality rate in monk seals (*Monachus schauinslandi*): adult survival rates vary substantially among subpopulations occupying different breeding sites, as well as among years (Baker and Thompson 2007). Female vulnerability to extrinsic mortality sources is also likely to be condition dependent, with high-condition females generally experiencing lower extrinsic mortality rates and higher life expectancies than low-condition females (Carlson et al. 2007; Rantala et al. 2011; Hostetter et al. 2012).

If changed environmental conditions persist over many generations, or ambient conditions vary on a large-enough spatial scale to permit genetic divergence, female life history will evolve to suit the prevailing environment. It is possible (although not certain) that such persistent or large-scale variation will have no net consequences for sexual conflict intensity. For example, a persistent elevation in extrinsic mortality rate will (all else equal) select for elevated female reproductive effort, but males may coevolve to elevate female reproductive effort even further, potentially resulting in no net change in the degree of sexual or parental antagonism. However, in this study, I examine the implications of relatively transient temporal, fine-scale spatial, or condition-dependent variation in female mortality rate, and my results suggest that such variation can have interesting implications for sexual coevolution.

I found that the intensity of sexual/parental antagonism can fluctuate over time or space as a consequence of fluctuations in female life expectancy (reflected in variation in P in Fig. 4). Sexual selection and conflict are expected to drive rapid, continual coevolution of male strategies and female counter-strategies (Iwasa and Pomiankowski 1995; Holland and Rice 1998; Arnqvist et al. 2000; Gavrilets 2000; Gavrilets et al. 2001; Gavrilets and Hayashi 2006), and the dynamics of such a Red Queen process may be affected by spatial and temporal fluctuations in the intensity of sexually antagonistic selection. Moreover, if females can assess their mortality risk, selection may favor facultative, environment-dependent mating strategies. For example, females occupying risky habitat patches may benefit by resisting manipulative males less than females occupying safer habitat patches. Similarly, in short-lived animals, seasonal variation in mortality risk could select for variation in female resistance to males.

The above analysis also suggests that highly manipulative males, which are normally harmful to females, can (all else equal) become beneficial for females if background mortality rate increases sufficiently (i.e., for large negative values of P; Fig. 4). Males can exhibit varying degrees of manipulativeness, and it is clear that female responses to such males can affect female fitness (Wagner et al. 2001; Pitnick and Garcia-Gonzalez 2002; Moore et al. 2003; Wagner and Harper 2003). The degree of male manipulativeness may be reflected in the potency or dose of accessory proteins such as the sex peptide transferred to females (and resulting degree of elevation in female reproductive rate), or the strength of the parent-of-origin effect on the male's offspring (and resulting degree of elevation in maternal provisioning). Figure 4 shows that, if female life expectancy is substantially reduced below the long-term mean to which females are adapted (i.e., at large negative values of P), the most manipulative males will provide a service by inducing females to ramp up their reproductive system. If females are able to assess their mortality risk (e.g., predator abundance in the local habitat) as well as male manipulativeness, then selection may favor a facultative female mate choice strategy whereby highly manipulative males are avoided when mortality risk is low but preferred when mortality risk is high.

The same reasoning suggests a novel evolutionary explanation for polymorphisms in genomic imprinting (Xu et al. 1993; Bunzel et al. 1998; Naumova and Croteau 2004). Although selection is thought to favor increased expression in offspring of paternally inherited alleles involved in extraction of maternal resources, maternally inherited alleles are selected for reduced expression (and ultimately silencing) to counteract this male strategy (Haig 2000). If the strength of parental conflict depends on extrinsic mortality rate, which fluctuates over space and time, then selection for genomic imprinting via the parental conflict mechanism can also fluctuate over generations and between subpopulations, potentially allowing for polymorphisms to be maintained.

The above analysis also has implications for the evolution of within-population polymorphisms in female mating strategy. Female vulnerability to extrinsic mortality factors tends to be condition dependent (Carlson et al. 2007; Rantala et al. 2011; Hostetter et al. 2012), such that low-condition females experience high mortality risk relative to the population mean (i.e., $P < 0$) whereas high-condition females experience low mortality risk relative to the population mean (i.e., $P > 0$). A manipulative male ($M \geq 0$) will thus tend to increase the fitness of a low-condition female (because $N_L^* > N^*$) but reduce the fitness of a high-condition female (because $N_H^* < N^*$). This effect suggests a novel hypothesis for the evolution of condition-dependent mate choice. Condition-dependent choosiness or preference functions have been reported in a number of empirical studies (e.g., Hunt et al. 2005; Cotton et al. 2006; Eraly et al. 2009; Holveck et al. 2011), and it has been

hypothesized that high-condition females may evolve to be more choosy because they experience lower costs and greater opportunity for choice than low-condition females (Parker 1983; Hunt et al. 2005; Cotton et al. 2006). The present analysis suggests an additional factor: high-condition females may suffer greater costs from mating with a manipulative partner. Selection may therefore favor stronger discrimination against the most manipulative males, or generally stronger resistance to mating, in high-condition females.

Finally, the present analysis has implications for the interpretation of laboratory studies. By eliminating most natural sources of extrinsic mortality, captivity typically extends life span (Comfort 1979). A direct comparison of a natural and genetically similar captive population showed that life expectancy was increased threefold in captivity relative to the wild (Kawasaki et al. 2008). Because transfer to captivity can correspond to a large, positive value of P in my analysis, captive populations may experience more intense sexual conflict than their counterparts in the wild. Toxic ejaculates represent a manifestation of interlocus sexual conflict, whereby male manipulation and female resistance strategies are controlled by distinct genetic loci (Arnqvist and Rowe 2005). Interestingly, captivity may also exacerbate intralocus sexual conflict, the displacement of one sex from its sex-specific phenotypic optimum through a different selection pressure on the same loci in the other sex (Bonduriansky and Chenoweth 2009). Intralocus sexual conflict may be intensified in captivity because viability-related selection vectors that are largely shared by the sexes are weakened in captivity, whereas sex-specific selection vectors (such as sexual selection) are maintained. By weakening viability selection, the sheltered environment experienced by captive populations may therefore exacerbate both inter- and intralocus sexual conflicts. Of course, captive animals need not necessarily experience increased life expectancy relative to their wild counterparts: whether they do so or not depends on the conditions under which lab stocks and experimental animals are maintained and assayed. Indeed, if lab populations are maintained on a very short generation time (as is typical of *Drosophila* stocks), and male effects on female fitness are assayed at a very young age, then the intensity of sexual conflict may actually be under-estimated relative to the natural source population. Thus, if the aim is to use a captive population to estimate the intensity of sexual conflict experienced by the natural source-population, then it will be necessary to (1) estimate the life expectancy experienced in the natural environment; (2) limit the reproductive lifespan of captive animals to the typical life expectancy experienced in the wild; and (3) assay male effects on female fitness at an age that corresponds to mean female life expectancy in the wild.

The analysis presented here is based on a number of simplifying assumptions. First, I have assumed that females' optimum reproductive effort and male effects on female life history remain constant over the female lifetime. Although reproductive effort may be age specific in many species, and male effects on female life history can change as a function of female age and can even reverse at old age (Fricke et al. 2013), the present analysis can be interpreted as representing the expected effects at the typical age at mating, which is likely to be "young" in natural populations (Kawasaki et al. 2008). Second, I assume that females have no postreproductive life span. If female reproductive value drops to zero prior to death, then the costs of male manipulation will only be affected if female life expectancy declines sufficiently to reduce reproductive output. Third, I assume that low- and high-condition females, or females in more or less risky habitat patches, deviate from the population-mean optimum reproductive effort, such that $N_L^* > N*$ and $N_H^* < N*$. If females can optimize their reproductive effort according to their individual condition and patch-specific mortality risk, then my conclusions relating to within-population variation in sexual antagonism will not hold (although conclusions relating to fluctuations of population-mean life expectancy will still be valid). The available evidence suggests that individuals' ability to adjust their reproductive effort in response to perceived mortality risk varies considerably among species and environments (e.g., Magnhagen 1990; Candolin 1998; Mappes et al. 1998; Ghalambor and Martin 2000; Scheuerlein et al. 2001; Jochym and Halle 2012; Trebatická et al. 2012), and more research is needed before general conclusions can be reached. Fourth, I assume that male manipulation is independent of female condition. Violation of this assumption is unlikely to alter my qualitative conclusions: if male investment in mating is a function of female condition (e.g., Lupold et al. 2011), then low-condition females may experience weaker male manipulation than high-condition females (unless a given level of male manipulation affects low-condition females more strongly), but the sign of the fitness effects on low- and high-condition females will be unchanged.

In addition, fluctuations in background mortality rate may affect both sexes. However, changes in male life expectancy are unlikely to alter the qualitative predictions of this study because, as long as females are polyandrous, males will be selected to induce elevated reproductive effort in their mates. If males are typically selected to pursue "live fast, die young" strategies relative to females (Bonduriansky et al. 2008), a reduction in male life expectancy may favor a "live faster, die younger" strategy involving even greater investment in sexual competition, including manipulative strategies (e.g., see Magurran and Seghers 1994). Nonetheless, male sexual competition can also lead to increased female mortality rate (Reale et al. 1996), potentially selecting for increased early-life reproductive effort in females, so the net intensity of sexual antagonism may remain unchanged. The assumptions and predictions of this study can be tested empirically

by varying female mortality rate experimentally and examining effects on latent costs of male manipulation.

ACKNOWLEDGMENTS

The author is grateful to the members of the lab for helpful discussions. Funding was provided by the Australian Research Council through a Fellowship and Discovery Grant.

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Associate Editor: A. Maklakov

Supporting Information

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

Figure S1. Effects of varying levels of male manipulation (M) when female life expectancy is reduced ($P < 0$) or increased ($P > 0$).

Figure S2. Female age-specific fecundity, f, declines more steeply with age, t, with increasing values of cost-function parameters d and a.

Figure S3. Variation in parameters a and d does not alter the qualitative patterns detected in the analysis.