Male coercion, female resistance and the evolutionary trap of sexual reproduction

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ABSTRACT

Sexual conflict involving male coercion has been suggested as a possible mechanism for the maintenance of costly sexual reproduction, offering a potential resolution to the 'paradox of sex'. However, the potential for sexual conflict to promote sexual reproduction may depend on ecological and genetic factors that influence the dynamics of sexually antagonistic coevolution. We investigated the conditions whereby male coercion could impede the invasion of asexual mutants and prevent transitions from sexual to asexual reproduction using a series of individual-based simulation models that vary in the ecology and genetic architecture of sexual antagonism. We show that a mutant allele that gives virgin females the ability to reproduce parthenogenetically easily invades obligately sexual populations over a broad parameter range via the fecundity advantage of occasional reproduction before mating. However, male coercion prevents transitions from facultative to obligate asexuality unless females evolve effective resistance. The potential for loss of sex can therefore depend on the dynamics of sexual arms races. Our results reveal the complementary roles of mate scarcity and female resistance in promoting the spread of asexual strategies. Our results also suggest that the costs and limitations of female resistance can be key factors in the maintenance of sexual reproduction, and that males' ability to overcome female resistance can turn sex into an evolutionary trap.

INTRODUCTION

Sex is associated with many short-term costs that parthenogenetic organisms mostly avoid (Maynard Smith 1978; Lynch 1984; Lewis 1987; Barton 1995; Crow 1999; Kotiaho 2001; Otto 2003; Agrawal et al. 2005; Arnqvist and Rowe 2005; Lehtonen et al. 2012; Meirmans et al. 2012). Asexuality should therefore be more widespread than sex, all else being equal (Maynard Smith 1978); but sex rather than parthenogenesis is the dominant mode of reproduction in nature (Bell 1982). This mismatch between theoretical prediction and

empirical reality has been famously dubbed “the paradox of sex” (Williams 1975; Maynard Smith 1978). Although numerous hypotheses have been proposed to explain this paradox (Kondrashov 1993; Hartfield and Keightley 2012), a universal benefit of sex remains elusive.

It is widely assumed that sex is more common than parthenogenesis because it provides benefits that outweigh the large costs (Maynard Smith 1978; Lehtonen et al. 2012). Genetic recombination is thought to provide most of these benefits by promoting the elimination of deleterious alleles (Muller 1964; Kondrashov 1993; Agrawal 2001; Siller 2001) and creating beneficial and novel gene combinations (Fisher 1930; Muller 1932) that augment adaptive responses to environmental or ecological change (Williams 1975; Maynard Smith 1978; Hamilton 1980; Bell 1982; Ladle 1992; Agrawal 2006) and enhance “evolutionary potential” of populations (Weismann 1889; Burt 2000; Roze 2012). However, because the benefits of recombination generally apply to populations rather than individuals, hypotheses based on indirect genetic benefits tend to treat sex as a group-selected trait (Kondrashov 1993; Green and Noakes 1995). Moreover, the hypothesized benefits of sexual reproduction typically apply within a limited subset of ecological parameter space (Bell 1982; Suomalainen et al. 1987; Barton 1995; West et al. 1999; Burt 2000; Rice 2002; Otto 2003, 2009; de Visser and Elena 2007; Kouyos et al. 2007; Sloan and Panjeti 2010), whereas the widespread taxonomic and ecological distribution of sexual reproduction in nature suggests that a very general mechanism may be involved.

An additional problem is that many models fail to account for the ubiquity of *obligate* sex. Theory suggests that rare or occasional sex is as effective as obligate sex at enhancing purifying selection (Lynch and Gabriel 1983; Wagner and Gabriel 1990), creating advantageous allele combinations (Kondrashov 1984; Bell 1988; Hurst and Peck 1996), promoting adaptation (Lynch and Gabriel 1983; Sasaki and Iwasa 1987; Burt 2000), and facilitating evolutionary escape from coevolving parasites (Yamauchi 1999; Flatt et al. 2001; Yamauchi and Kamite 2003). Rare sex therefore seems to provide all the genetic advantages of obligate sex with much lower costs (Lynch and Gabriel 1983; Lewis 1987;

Hedrick and Whittam 1989; Green and Noakes 1995; Joshi and Moody 1995, 1998; Hurst and Peck 1996; Yamauchi and Kamite 2003). Thus, any mechanism that proposes to explain the maintenance of sexual reproduction must account for the dominance of obligate sex over facultative strategies. This requires understanding the factors preventing alleles that code for sexual reproduction from being supplanted by spontaneous mutations for facultative parthenogenesis – a reproductive mode where sexual and asexual reproduction are possible within a single individual.

It has been suggested that costly obligate sex could be maintained by physiological constraints. For example, limited diploidy restoration (Suomalainen et al. 1987), failed activation of unfertilised eggs (Stricker 1999), and genomic imprinting (Reik and Walter 2001), may impede the evolution of parthenogenesis in many sexual animals (Engelstadter 2008). Such constraints can limit asexual invasions by disrupting embryonic development, suppressing asexual egg production, or reducing offspring viability (Enghoff 1976; Lamb and Willey 1979; Lynch 1984). Likewise, physiological dependence on sexual stimulation could constrain the spread of spontaneously-derived parthenogenesis (Neiman 2004). However, if asexual reproduction is advantageous, long-term evolution may be expected to circumvent such constraints in many lineages. The widespread occurrence of obligate sexuality therefore suggests the existence of taxonomically widespread mechanisms or dynamics that not only prevent asexual reproduction from occurring in existing sexual populations, but also act as persistent impediments to evolutionary transitions to asexuality in diverse sexual lineages, turning sex into an evolutionary trap.

The sexual behaviour of males has been suggested as an ecological mechanism that could directly constrain parthenogenetic invasions (Dagg 2006; Singh and Artieri 2010). During sexual interactions, males often direct coercive behaviours toward females to maximise their own reproductive fitness, even at the expense of their mates (Parker 1979; Arnqvist and Rowe 2005). In particular, males typically benefit from each additional mating whereas females have a lower optimum mating rate, and selection therefore favours male strategies

that coerce females into mating and reduce female fecundity (Maklakov et al. 2005) or longevity (Martin and Hosken 2003). Given the potentially large costs that females can suffer due to male harassment, sexual coercion (and sexual conflict more generally) has been proposed as an additional cost of sex (Rankin et al. 2007). However, it has also been suggested that sexual coercion could promote the persistence of sex (Dagg 2006; Kawatsu 2013*a*, 2013*b*, 2015; Burke et al. 2015; Gerber and Kokko 2016). In a sexual population, a mutant allele that makes parthenogenetic reproduction possible may be expected to flourish and spread due to the demographic advantage of producing all-female offspring (Maynard Smith 1978), as well as the ecological and physiological advantages of reproduction without mating. However, if asexual mutants suffer greater costs than sexual females as a consequence of encountering coercive males, parthenogenesis may fail to spread (Kawatsu 2013*b*). Such differential costs could manifest as coercion-induced declines in female fecundity or survival (Burke et al. 2015). Moreover, coercion could inhibit the spread of parthenogenesis by forcing females to reproduce sexually because, in many facultatively asexual animals, only virgin females are able to reproduce parthenogenetically (Bell 1982; Normark and Kirkendall 2009).

Male coercion can select for female resistance, potentially setting off a sexual “arms race” (Rice and Holland 1997; Holland and Rice 1998; Gavrilets 2000). Thus, female resistance could play an important role in counteracting the suppressive effect of coercion on parthenogenetic reproduction. A recent theoretical study suggests that obligate parthenogenesis can evolve from facultative parthenogenesis if females acquire high resistance mutations (Kawatsu 2013*a*). However, the outcome may also depend on the dynamics of sexual coevolution. For example, if sexual conflict is resolved in favour of males by the fixation of high coercion alleles before parthenogenetic mutants are born into the population, the potential for parthenogenesis to invade may be limited. By contrast, if high resistance genotypes are present in the population and become genetically associated with parthenogenesis, parthenogenetic mutants may be able to avoid sex via resistance.

Another theoretical analysis suggests that female resistance to mating may be more effective in promoting parthenogenesis when mate encounter rate is low (Gerber and Kokko 2016). However, this analysis did not fully disentangle the relative roles of resistance versus mate scarcity in promoting evolutionary transitions to asexuality. Disentangling these mechanisms requires assessment of the timing of parthenogenetic reproduction in relation to mating attempts: mate scarcity can select for parthenogenesis whenever facultatively parthenogenetic virgins reproduce asexually prior to encountering a mate; whereas resistance can select for parthenogenesis whenever virgin females reproduce asexually after resisting sexual encounters. The contribution of each of these mechanisms to the invasive potential of facultative asexuality therefore remains unclear (although see Burke et al. 2015).

We investigated the roles of sexual conflict and mate scarcity in the invasion of facultative mutants and the potential for transitions to obligate asexuality using a series of individual-based simulation models (IBMs) that varied in intensity of sexual conflict, dynamics of sexual coevolution, ecological conditions, and genetic background. We first investigated the conditions whereby a sexual population can be invaded by a mutant allele that allows virgin female carriers to reproduce parthenogenetically. We then investigated the conditions whereby populations can transition from facultative to obligate parthenogenesis (i.e., undergo complete loss of sex). Finally, we examined the consequences of the invasion of parthenogenetic mutants on sexual conflict and population demographics.

METHODS

Overview of IBMs

We aimed to examine conditions that promote invasion of a mutant asexual strategy against a background of on-going sexually antagonistic coevolution, and to explore the interaction of sexual conflict and mate scarcity in this process. We therefore used individual-based simulation models (IBMs), which afford maximum flexibility for investigating these

probabilistic and dynamic processes, and minimize the need for simplifying assumptions and approximations. Our models examine scenarios varying in the genetic architecture of sexual antagonism (i.e., the relative efficacy of coercion genotypes versus resistance genotypes), the costs of mating and resistance, the density of populations, the fecundity of parthenogenetic females relative to sexual females, and the timing of introduction of facultatively parthenogenetic mutants. All models are run in the modelling program NetLogo (U. Wilensky 1999).

We consider a finite population of diploid organisms with overlapping generations inhabiting a gridded environment of square patches wrapped around at the edges to create a world without boundaries. This explicit spatial structure enabled us to create high and low population densities, which are known to affect evolutionary outcomes in facultative systems (Gerber and Kokko 2016), by setting the number of patches in the world to low (11 x 11) and high (51 x 51), respectively.

The sexes in our models experience sexual conflict over mating rate such that multiple matings always benefit males but can be costly to females. Although species vary in the number of matings females can obtain before additional matings become costly (Parker and Birkhead 2013), for simplicity, our models follow the formulation of Arnqvist and Rowe (Arnqvist and Rowe 2005) and assume that any more than one mating is costly for females. The sexes in our models experience antagonistic sexual coevolution, a predicted consequence of sexual conflict (7). We model coercion and resistance as discrete traits, each controlled by a diploid autosomal locus with two alleles, *c* and *C*, and *r* and *R*, respectively, with additive, sex-limited effects. We model resistance and coercion this way because we wanted to investigate cases where sexually antagonistic selection on standing genetic variation could reach a stable end point, at which either male coercion or female resistance gains the upper hand. In a separate set of models that simulate an escalating arms race, we allow coercion and resistance to coevolve without constraint by treating them as continuously distributed values (see Online Appendix).

The reproductive mode of females is controlled by a sex-limited autosomal locus with two alleles, *p* and *P*, with wild-type *pp* individuals capable only of sexual reproduction. *P* is a dominant mutant allele that allows females to reproduce without mating (i.e., via facultative parthenogenesis), but has no phenotypic effect in males. For simplicity, we assume that a single locus controls all physiological traits involved in the switch between sexual and asexual reproduction in mutants (but we revisit the biological realism of that assumption and implications of its violation in the Discussion).

The mutant *P* allele is introduced either at time-step 0 (i.e., at initialization), or at time-step 10,001 (i.e. after simulations have run for 10,000 time-steps, allowing for sexually antagonistic selection to alter allele frequencies and for genetic associations to build-up in the population). We hereafter refer to models setup this way as 'non-equilibrated' and 'equilibrated', respectively.

Initialization of simulations

At the start of each simulation and before the mutant *P* allele is introduced, 500 females and 500 males are randomly distributed throughout the environment, each facing a randomized direction. Each individual is allocated a maximum lifespan of 100 time-steps, and an age of 0 time-steps that increases by 1 every time-step. Males and females are both allocated coercion and resistance genotypes according to Hardy-Weinberg probabilities (i.e., individuals have a probability of 0.5 of obtaining heterozygous genotypes and a probability of 0.25 of obtaining either of the alternative homozygous genotypes). The carrying capacity of the environment (i.e., maximum population size) is fixed at 2,500.

Once per simulation, at either time-step 0 or 10,001, mutants carrying the *P* allele are introduced. A random sample of 2% of the current population of males and 2% of the current population of females receive one or two copies of the mutant *P* allele at the reproductive mode locus, with genes at other loci unaltered.

Life cycle

During each time-step, organisms perform tasks in four ordered phases: moving, mating, reproducing and dying. In the moving phase, individuals randomly turn to face a new direction (between 0 and 90 degrees relative to their current direction) and then move forward one unit (the length of a patch).

During the mating phase, each male randomly chooses a female in his patch that has not mated in the current time-step and tries to mate with her. Males can make only one attempt at mating per time-step, but females can be courted sequentially by more than one male if successive mating attempts in a time-step are unsuccessful. Mating occurs when a male's coercion genotype beats the resistance genotype of the female.

Models are designed so that one of the sexes can evolve to permanently gain the upper hand in sexual coevolution. Males can gain the upper hand when *CC* males are able to beat all resistance genotypes (hereafter, 'coercion' models), whereas females can gain the upper hand when *RR* females are able to beat all coercion genotypes (hereafter, 'resistance' models). Thus, mating outcomes are determined as follows: in coercion models, *CC* males can mate with any female; *Cc* males can mate with *Rr* or *rr* females; *cc* males can mate with *rr* females. In resistance models, *CC* males can mate with *Rr* or *rr* females; *Cc* males can mate with *rr* females; *cc* males cannot mate with any females. Our models therefore represent a sexual arms race where male coercion and female resistance traits are each determined by a single, bi-allelic locus, and sexually antagonistic coevolution occurs via selection on standing genetic variation at those two loci. Mutation rate is set at zero in these models because we were interested in how sexual conflict influenced the invasion probability of the *P* allele both before and after sexual coevolution had stabilized at coercion-dominated versus resistance-dominated states.

Resistance is either costly or non-costly for females. When resistance is costly, females incur a 10-time-step reduction to their remaining lifespan every time they successfully resist

a mating attempt (i.e., if mating is attempted but does not take place). Successful matings result in the transfer of paternal alleles via male gametes (sperm) which females store internally. Following other models of sexual conflict (e.g., 72), we assume that females store enough sperm from one mating to reproduce sexually their whole life. Last male precedence, which is widespread in a diversity of animal taxa (Smith 1984), is assumed in all models.

Males and females incur sex-specific survival costs of mating, applied as penalties of 0, 5, 10, 15 or 20 time-steps deducted from an individual's remaining lifespan. This generates sexual conflict over mating rate: when the female mating cost is > 0, a single mating per lifetime is optimal for females, whereas male fitness increases with each additional mating regardless of the cost.

The mating phase is followed by the reproducing phase. Reproduction is a lottery that occurs every time-step as long as the current population size is less than the fixed carrying capacity. Each female capable of reproducing (i.e., any previously mated female, and any virgin female carrying at least one copy of the *P* allele) is allotted a random number between 0 and 1 every time-step. Previously mated females with a random number < 0.1 reproduce sexually. For virgin females that carry the *P* allele, reproduction probability per time-step is set at either 0.05 or 0.1, representing either 50%-reduced parthenogenetic fecundity (i.e., a genetic/physiological constraint on asexuality; 49,71) or equal fecundity of parthenogenetic reproduction relative to sexual reproduction. A female can win the reproductive lottery multiple times during her life, and produces one offspring each time. Females that reproduce parthenogenetically produce only daughters, while females that reproduce sexually produce daughters and sons with equal probability. As occurs in many facultatively parthenogenetic taxa (Bell 1982), females that mate reproduce sexually thereafter, even if they are capable of parthenogenesis (i.e., even if they carry the P allele). There are no costs of gamete or offspring production.

All offspring inherit parental alleles and trait values for reproductive mode, coercion, and

resistance. We assume that daughters of unmated mothers are produced via apomixis, the most common mechanism of animal parthenogenesis (Bell 1982), and that they therefore inherit their mothers' genotype without modification. Sexually produced offspring inherit parental alleles following Mendelian rules of segregation (i.e., one randomly selected allele at each locus from each parent).

The dying phase follows the reproducing phase. An individual's survival value, *S*, is determined as:

$$S = 1 - \left(\frac{a + cm}{L} \right)$$

where *a* is an individual's current age in time-steps, *c* is the sex-specific cost of mating in time-steps, *m* is an individual's cumulative number of matings, and *L* is the potential lifespan at birth (set at 100 in all models). Death occurs when $S \leq 0$.

Analysis

We performed 25 simulation runs of each of 800 unique parameter combinations to determine the proportion of simulations that ended in *P* allele fixation, *P-p* polymorphism or *P* allele extinction, and the proportion that ended in obligate sex, facultative parthenogenesis, obligate parthenogenesis or population extinction. In one additional run, we collected data every time-step on population size, sex ratio, *P* allele frequency, mean lifetime mating costs, antagonistic genotype frequencies, and number of offspring. All simulation runs lasted 20,000 time-steps following the emergence of the *P* allele, except in cases of prior population extinction. A list of all model parameters is provided in table 1.

RESULTS

Sexual conflict as a function of differential costs of mating

Relative male and female costs of mating have consistent and important demographic effects prior to the introduction of the *P* allele. When the cost per mating is set higher for females than for males, male-biased sex ratios are generated because of reduced female longevity; whereas when the cost per mating is set higher for males than for females, female-biased sex ratios are generated because of reduced male longevity. This pattern can be seen in the first 10,000 time-steps of Figures 1 *A* and *B* and: male sex ratios are less than 0.5 when male costs are higher than female costs, but are greater than 0.5 when female costs are greater than male costs. A male-biased sex ratio increases sexual encounter rates and mean female mating rates, elevating female mortality. This demographic pattern is observable in the mean lifespan penalties incurred by mated females during the first 10,000 time-steps of Figures 1 *C* and *D*. The consequences of these dynamics are discussed below.

Conditions for the invasion of the P allele

The *P* allele spreads via two non-mutually exclusive mechanisms: the ability to reproduce asexually prior to encountering any males (mate scarcity), and the ability to reproduce asexually by resisting males (sexual conflict). The mate scarcity mechanism contributes to the spread of the *P* allele in all models, but especially in coercion models where resistance is suppressed (Figure 2 *A*). The *P* allele easily invades in these models even if all males are capable of coercing all females into mating. This is because some females fail to encounter a male, and the P allele gives these females the opportunity to reproduce parthenogenetically. Separate analyses (not shown) confirm that the *P* allele spreads as a consequence of this general fecundity advantage and not due to drift.

In non-equilibrated resistance models, mate scarcity and high resistance interact to drive the *P* allele to fixation (Figure 2 *B*). In these models, positive linkage disequilibrium develops

between the *R* allele and the *P* allele, creating strong epistasis for fitness: females attain higher fitness when the capacity for parthenogenesis is coupled with high resistance (Figure 3). However, this is possible only when the cost of resistance is sufficiently low (Figure 2 *B*). When linkage disequilibrium can build up, resistance plays a greater role than mate scarcity in promoting the spread of parthenogenesis during initial stages of invasion (Figure 4 *B*). However, as invasions progress and sexual encounters decline with shrinking male sex-ratio, mate scarcity becomes the dominant driver of parthenogenesis (Figure 4 *B*). By contrast, in non-equilibrated coercion models, resistance partially contributes to the spread of parthenogenesis immediately following *P*'s introduction because some resistance is still possible at this stage (Figure 4 *A*). Once the *C* allele fixes, however, mate scarcity becomes the sole mechanism by which parthenogenesis can spread (Figure 4 *A*).

The timing of the *P* allele's introduction also determines whether positive epistasis for fitness between parthenogenesis and resistance develops. This is because model equilibration alters the genetic background of antagonistic traits. In equilibrated resistance models when the *P* allele is introduced after 10,000 time-steps of coevolution, the *R* allele is already extinct and thus linkage disequilibrium between the *P* and *R* alleles is unable to build up. However, when resistance models are non-equilibrated (i.e., when mutants are introduced at the start of simulations), resistance alleles are at Hardy-Weinberg equilibrium and thus the *P* allele can rapidly associate with high resistance genotypes and invade over a large range of the parameter space (Figure 2 *B*). By contrast, timing of introduction has little effect on the *P* allele's spread in coercion models (Figure 2 *A*) because high coercion evolves to beat resistance in all situations (Figures 5 *A* and *C*) and therefore prevents linkage disequilibrium regardless of when the *P* allele is actually introduced.

Low patch number (and therefore high density) facilitates an increase in sexual conflict by

elevating mating rates and increasing realised mating costs for females, which has a significant effect in all models on the realised parameter space in which populations persist and parthenogenesis can invade. The opposite is also true: sexual conflict is exacerbated by high density in worlds with low patch number. Increased sexual conflict leads to an increase in population extinction (Figures 2 *C* and *D*) because females mate more frequently in high densities, have shorter lifespans, and therefore produce fewer offspring. Resistance costs intensify this pattern of extinction in high density populations, especially in resistance models (Figure 2 *D*), because the more matings that females resist, the shorter their lifespans, and the fewer opportunities to reproduce. These dynamics allow *P* to fix more frequently in populations that inhabit worlds with high patch number (i.e., low population densities) than low patch number (Figures 2 *A* and *B*).

Conditions for the establishment of obligate parthenogenesis

The introduction of *P*-allele-carrying mutants into obligately sexual populations leads to one of three distinct evolutionary outcomes: (1) The *P* allele dies out, leaving populations to reproduce via obligate sex; (2) The *P* allele spreads either to an intermediate frequency or to fixation such that sex and parthenogenesis coexist within and/or between individuals; (3) The *P* allele spreads to fixation and parthenogenesis becomes obligate as a result of the complete extinction of males (Figures 2 *C* and *D*). The coexistence of parthenogenetic and sexual reproduction is the most common evolutionary outcome, whereas obligate sex resulting from the loss of the *P* allele is the least common result (Figures 2 *C* and *D*). These patterns are generated by the same resistance and mate scarcity mechanisms explained above. Mate scarcity universally favours parthenogenesis because some virgin females are always able to reproduce asexually prior to encountering a male. However, by mating with females that carry the *P* allele, highly coercive males ensure such females reproduce sexually for the rest of their lives, limiting future opportunities for parthenogenesis. Male coercion, therefore, effectively hinders evolutionary transitions from facultative to obligate parthenogenesis. This constraining effect is particularly evident in coercion-model

simulations where fecundities of sexual and parthenogenetic reproduction are unequal: obligate parthenogenesis is completely suppressed in such simulations (Figure 2 *C*). But even when fecundities are equivalent in coercion models, coercion successfully staves off the complete loss of sex in the majority of the parameter space (Figure 2 *C*). The exception to this is in cases where mating is high for both sexes but especially high for males (Figure 2 *C*). Here, sex is lost because mated females die more quickly and produce fewer offspring than virgin females, resulting in a reduction in the proportion of sexually-produced recruits. More males die than are born in some time-steps, and random drift eventually causes the extinction of the last surviving males. However, when parthenogenetic reproduction imposes higher fecundity costs on females relative to sexual reproduction, this dynamic does not occur and male extinction is averted (Figure 2 *C*) because fewer sexual recruits are displaced when parthenogenesis is costly.

High resistance in non-equilibrated resistance models releases females from the constraints of high coercion and facilitates rapid transitions to obligate parthenogenesis across a very broad parameter space, but only if costs incurred due to resistance are not too high (Figure 2 *D*). By contrast, the number of simulations ending in obligate parthenogenesis in equilibrated resistance models closely resembles the outcome for equilibrated coercion models (compare Figures 2 *C* and *D*). This similarity is due to sexually antagonistic selection favouring the high coercion *C* allele and suppressing the high resistance *R* allele in both equilibrated models (Figures 5 *A* and *B*). Selection always promotes the *C* allele due to the universal benefit to males of mating multiply (Figure 5); whereas the *R* allele is strongly selected against in the absence of the *P* allele because *RR* females successfully resist all mating attempts and produce no progeny when parthenogenesis is not possible. This effect is particularly evident in equilibrated resistance models where *R* is highly disadvantageous in the absence of *P*, and the least resistive allele *r* becomes fixed before mutants carrying the *P* allele appear in the population (Figure 5 *B*).

386 ***Population effects of sexual conflict and parthenogenesis***

387 In equilibrated models, fixation of the *P* allele is associated with either a reduction or an
388 increase in sexual conflict, depending on the relative costs of mating. When the cost of
389 mating for females is less than or equal to the cost for males, populations tend to reach
390 carrying capacity and sex ratios are either female-biased or equal at the time the *P* allele is
391 introduced at time-step 10,001 (Figures 1 *A* and *B*). During the spread of the *P* allele
392 thereafter, parthenogenetic reproduction increases in frequency causing sex ratios to
393 become even more female-biased (Figures 1 *A* and *B*). This reduces the probability of
394 sexual encounters and lowers conflict over mating rate (Figures 1 *C* and *D*). However, by
395 contrast, higher female than male mating costs generate population sizes lower than
396 carrying capacity and sex-ratios that are male-biased (Figures 1 *A* and *B*). As facultative
397 parthenogenesis spreads in this context, populations increase to carrying capacity and sex
398 ratios become slightly less male-biased, but never female-biased (Figures 1 *A* and *B*).
399 Females in these larger, male-biased populations experience higher mating rates after the
400 introduction of the *P* allele but are unable to resist effectively because high resistance
401 genotypes are already extinct (Figure 5 *B*). These dynamics result in most cases in an
402 increase in sexual conflict over mating rate.

403 A higher mating cost for females than for males generates oscillating population sizes and
404 sex ratios (Figure 6), the dynamics of which operate in the following way. As mentioned
405 above, populations become male-biased when costs of mating are higher for females, and
406 mating multiply in the presence of so many males increases female mortality and leads to
407 population decline. Sexual encounter rates also decline, providing greater opportunities for
408 females with the *P* allele to reproduce parthenogenetically. With the production of a larger
409 number of parthenogens, the population rapidly increases, and the cycle continues.
410 However, this pattern of oscillation causes populations to crash completely in some extreme
411 cases (see Figure 6 *B* where male mating cost is 0 and female cost is 20).

412

DISCUSSION

Previous studies have suggested that male coercion can promote the maintenance of sexual reproduction in facultatively asexual systems (Kawatsu 2013*a*, 2013*b*, 2015; Burke et al. 2015; Gerber and Kokko 2016). Results from these studies suggest that sexual conflict could play a key role in the maintenance of sexual reproduction, and thus contribute to a resolution of the 'paradox of sex'. However, sexual conflict gives rise to sexually antagonistic coevolution—a process whose dynamics are likely to reflect both ecological parameters (such as costs of mating, sexual encounter rates, and resistance) and the genetic architecture of sexually antagonistic traits. Understanding the role of sexual conflict in the maintenance of sex requires elucidating how these factors and resulting coevolutionary dynamics promote/inhibit transitions from sexual to asexual reproduction. Moreover, previous studies have not clearly differentiated the role of sexual conflict from the role of mate scarcity in the evolution of reproductive mode. We used individual-based models with varying ecological and genetic parameters to investigate the conditions whereby an asexual (parthenogenetic) strategy can invade sexual populations undergoing sexually antagonistic coevolution.

Our results indicate two mechanisms by which sexually coevolving populations can be invaded by facultative parthenogenesis: mate scarcity and female resistance. It has long been suggested that mate scarcity favours the spread of facultative parthenogenesis in small or female-biased populations where mating is either difficult or impossible (Stalker 1956; Kramer and Templeton 2001; Schwander et al. 2010), and might therefore account for the evolution of some facultative systems, including some Phasmatids (Schwander and Crespi 2009), Ephemeropterids (Brittain 1982), and Dipterans (Markow 2013). The mate scarcity mechanism applies to any situation where virgin females have an opportunity to reproduce prior to initial sexual encounters. Our analysis allowed us to distinguish between instances of parthenogenetic reproduction facilitated by mate scarcity (i.e., parthenogenesis before initial mating attempts) and by resistance (i.e., parthenogenesis after initial mating attempts), and

therefore to identify the relative contribution of each mechanism to the spread of facultative parthenogenesis. We found that the *P* allele invaded successfully and displaced alleles for obligate sex across most of the realised parameter space due largely to the mechanism of mate scarcity. Even when all matings were coerced, the *P* allele typically fixed, albeit slowly. High coercion was unable to prevent the invasion of facultative parthenogenesis because fecundity selection favoured females that produced additional offspring prior to encountering a mate, generating positive selection on the *P* allele. However, when successful resistance was possible, the *P* allele invaded across a greater portion of the parameter space because mate scarcity and resistance-mediated mechanisms acted in tandem, making parthenogenesis possible both before and after sexual encounters. In other words, the *P* allele conferred the greatest advantage and experienced the strongest positive selection when in positive linkage disequilibrium with alleles conferring a capacity for effective female resistance to mating. High resistance therefore increased the number of offspring produced parthenogenetically, and facilitated rapid and widespread fixation of the mutant allele.

The introduction of the *P* allele into obligately sexual populations led to one of three distinct evolutionary outcomes: obligate sex, facultative parthenogenesis, or obligate parthenogenesis (loss of sex). The distribution and frequency of each of these reproductive modes was strongly determined by the genetic architecture of sexual antagonism at the time of the *P* allele's emergence. When males successfully evolved the capacity to coerce any female to mate, facultative parthenogenesis was the predominant outcome, and male extinction rarely occurred. By contrast, when females evolved effective resistance, male extinction (and therefore obligate parthenogenesis) was the most common result. Importantly, this finding occurred both when coercion and resistance were modelled as traits determined by single loci (and therefore when evolution occurred via selection on standing genetic variation), and when coercion and resistance were modelled as multi-locus traits representing a large mutational target (and therefore when sexual arms races could escalate without limit; see Online Appendix) This suggests that when effective resistance cannot

evolve, male coercion can impede transitions to obligate parthenogenesis and contribute to the maintenance of sex (albeit often in combination with parthenogenesis). By contrast, if coercion can be overcome by effective resistance, transitions to obligate asexuality are likely.

Our finding that resistance can be a key factor in transitions to facultative and obligate parthenogenesis is consistent with results of previous studies (Kawatsu 2013*a*; Burke et al. 2015; Gerber and Kokko 2016). However, our analysis provides a number of additional insights. In particular, we show that the potential for invasion of facultatively parthenogenetic mutants is strongly dependent on the relative costs of mating for males and females and the dynamics of sexual coevolution. Consistent with previous findings (Kawatsu 2013*a*), we found that the evolution of obligate parthenogenesis depended on whether alleles for effective female resistance were present in the population at the time of the *P* allele's introduction, and therefore whether positive epistatic associations between resistance and parthenogenesis could build up. In addition, our analysis showed that the evolution of obligate parthenogenesis was contingent on the weight of resistance costs: populations evolved to be obligately asexual only to the extent that females were able to bear the cost of resisting mating attempts. This result supports previous findings that facultatively parthenogenetic females should resist only at low population densities where encounter rates are low (Gerber and Kokko 2016). Our analysis also extends previous work by clarifying the roles of both mate scarcity and sexual conflict, and the consequences of fecundity costs of parthenogenetic reproduction, in evolutionary transitions to facultative and obligate parthenogenesis.

The limitations of resistance highlighted by our analysis have important implications for understanding the incidence of sex and parthenogenesis in nature. In many species, males tend to "win" sexual arms races due to intense and persistent selection for effective coercion, whether by mechanically overpowering females to force matings (Rowe et al. 1994), by chemical manipulation (Chapman et al. 1995; Andersson et al. 2004), or by pre-copulatory

494 exploitation of sensory biases (Holland and Rice 1998; Arnqvist and Rowe 2005). By
495 contrast, high female resistance genotypes may be rare or absent from many populations
496 due to strong selection against absolute resistance, or due to selection favouring
497 convenience polyandry when costs of resistance are high (Rowe 1992). Moreover, many
498 resistance behaviours are plastic, with virgin females often the least resistant to mating (79,
499 but see 80), while fixed strategies of high resistance are probably rare in natural populations.
500 These factors may severely inhibit transitions to asexuality because without the prior
501 evolution of effective resistance transitions to obligate parthenogenesis might be almost
502 impossible. However, limitations of resistance may only inhibit parthenogenesis derived from
503 spontaneous mutations. Parthenogens originating from interspecies hybridisation are often
504 immediately reproductively isolated from their progenitors (Simon et al. 2003), and such
505 isolation could facilitate immediate escape from sexual antagonism and avert any
506 dependence on high resistance for the evolution of obligate parthenogenesis. This may be
507 one reason why many parthenogenetic organisms – including all known obligately
508 parthenogenetic vertebrates (Avise et al. 1992; Simon et al. 2003) – have a hybrid origin.

509 The evolution of facultative parthenogenesis is expected to mitigate some of the costs of sex
510 at the population level (Hurst and Peck 1996). By having the capacity to reproduce both
511 sexually and asexually, females should gain the benefits of sex while avoiding some of the
512 costs associated with males, including costs of mating (Green and Noakes 1995). However,
513 one of the novel findings of our study is that invasions of facultative parthenogenesis can
514 either reduce or exacerbate ongoing sexual conflict over mating rate, depending on the
515 relative costs of mating for males and females. Our findings also show that facultative
516 parthenogenesis can reduce or exacerbate the risk of population extinction, depending on
517 the effectiveness and cost of resistance. This suggests that the potential benefits of
518 facultative reproduction may be more context-dependent than generally assumed. Sexually
519 antagonistic selection may have dynamic effects on facultative systems that current theory
520 fails to capture. This highlights the need to apply sexual conflict theory more

521 comprehensively to theoretical assessments of the maintenance of obligate sex, an
522 approach that has already proven fruitful in the study of alternative reproductive strategies in
523 hermaphroditic systems (Abbott 2011; Schärer et al. 2015).

524 Including sexual conflict in models of the maintenance of sex could point to interesting
525 avenues for research. Our results provide several testable predictions. If male coercion
526 inhibits the evolution of obligate parthenogenesis, taxa with greater potential for coercion
527 may be less likely to exhibit obligately asexual forms. For example, at a broad phylogenetic
528 scale, the rarity of asexuality in animals compared to plants (Otto and Whitton 2000) may
529 reflect the far greater variety of opportunities for coercion in animal systems. For example,
530 male animals can coerce females using behaviours and morphology (e.g., chasing (den
531 Hollander and Gwynne 2009), and holding mates during copulation (Rowe et al. 1994)), as
532 well as chemicals and pheromones (e.g., toxic ejaculates (Wigby and Chapman 2005), and
533 anti-aphrodisiacs (Andersson et al. 2004)). Coercive strategies may be more limited in
534 plants, which lack self-propelled motility, sense organs and nervous systems. Similarly,
535 sessile animals that reproduce via external fertilisation may experience less coercion than
536 free-living, internally-fertilising animals, and parthenogenesis may therefore be more
537 common in such taxa. Studies are needed to confirm these predictions.

538 Given the broad parameter space over which facultative parthenogenesis spreads in our
539 simulations via the mate scarcity mechanism, the fact that invasions by facultatively
540 parthenogenetic mutants appear to occur so rarely in animal populations requires an
541 explanation. Several factors could contribute to this discrepancy. First, our models
542 introduced asexual mutants in relatively large numbers, which reduced the likelihood that
543 parthenogenesis would disappear via drift. Natural populations, however, probably give rise
544 to facultatively parthenogenetic mutants at very low rates (Schwander et al. 2010), and drift
545 may therefore play an important role in constraining mutant invasions. Second, the complex
546 cytological and physiological changes associated with parthenogenetic reproduction (such
547 as spontaneous development of unreduced eggs; 91) may require mutations at multiple loci,

548 which may be highly unlikely to occur simultaneously (Neiman et al. 2014). Third, even if
549 lineages can overcome such genetic constraints, facultative mutants may be less fecund
550 than wild-type females (Lamb and Willey 1979). For example, facultatively parthenogenetic
551 mutants of the cockroach *Nauphoeta cinerea* produce ten times fewer offspring than non-
552 mutant individuals (Corley and Moore 1999), a relative rate of parthenogenetic reproduction
553 far lower than the 50% reduction in fecundity we investigated in our models. Fourth, the
554 spread of mutants may be further constrained in nature by costs associated with plastically
555 switching between sex and parthenogenesis. In the facultatively parthenogenetic stick
556 insect, *Extatosoma tiaratum*, females that switch to sex after initially ovipositing
557 parthenogenetically suffer elevated mortality and decreased egg production compared to
558 females reproducing exclusively sexually or asexually (Burke et al. 2015). Such factors could
559 greatly reduce the potential for sexual populations to generate and be invaded by
560 facultatively parthenogenetic mutants.

561 However, physiological constraints alone seem unable to explain the wide distribution of
562 obligate sex in the animal kingdom. If parthenogenesis is beneficial, any constraint on
563 parthenogenetic fecundity should be eliminated by long-term selection, as has been
564 demonstrated in artificial selection experiments on the facultatively parthenogenetic fly
565 *Drosophila mercatorum* (Stalker 1956; Carson 1967). Thus, a mechanism that consistently
566 inhibits parthenogenesis in multiple taxa over long evolutionary time frames seems
567 necessary. Sexual conflict mediated by male coercion could be such a mechanism. Mating
568 rate is a widespread source of sexual conflict in animals (Wedell et al. 2006), and frequently
569 selects for coercive strategies in males (Arnqvist and Rowe 2005). Because coevolution of
570 male coercion and female resistance can lead to never-ending sexual arms races (Rice and
571 Holland 1997; Holland and Rice 1998; Gavrilets 2000), sexual conflict could be a consistent
572 long-term inhibitor of parthenogenesis in many lineages. However, given our finding that
573 high coercion cannot prevent alleles for facultative parthenogenesis from invading via the
574 mate scarcity mechanism, a combination of constraints - including developmental,

575 physiological, and ecological constraints like sexual coercion - may interact to prevent
576 facultatively parthenogenetic mutants from invading sexual populations in the natural world.

577 In addition, coercive sons could provide indirect fitness benefits to sexually reproducing
578 females in facultatively parthenogenetic systems (Kawatsu 2015). These indirect benefits
579 would increase as parthenogenesis spreads and as populations become more female-
580 biased, selecting for male-biased offspring sex-ratios. Such benefits may compound the
581 constraining effect of coercion on transitions to obligate asexuality.

582 The widespread distribution and long-term maintenance of obligate sexual reproduction is an
583 enduring paradox. Identifying genetic benefits of sexual reproduction has been the dominant
584 approach to this problem (Hartfield and Keightley 2012). But constraints that inhibit invasions
585 of spontaneously derived parthenogenesis could enable obligate sex to persist (Vrijenhoek
586 1989; Neiman 2004; Engelstadter 2008), and recent studies suggest that male coercion
587 could play this role (Kawatsu 2013*a*; Burke et al. 2015; Gerber and Kokko 2016). Our results
588 clarify the contribution of sexual conflict and sexually antagonistic coevolution in the
589 maintenance of obligate sex. We show that facultatively parthenogenetic mutants can invade
590 sexual populations because of the benefit to females of reproducing prior to encountering
591 males (mate scarcity mechanism), but the probability of facultative populations transitioning
592 to obligate asexuality depends largely on the potential for females to evolve effective, low-
593 cost resistance to mating, such that linkage disequilibrium can build up between alleles for
594 female resistance and alleles for facultative parthenogenesis. This is because, although
595 females may benefit by reproducing parthenogenetically instead of sexually, obligate
596 parthenogenesis is likely to evolve only if females can overcome male coercion and thereby
597 reproduce without paying the costs of sex. The difficulty of such a feat suggests that sex
598 may be an evolutionary trap imposed on populations by the evolution of coercive males.
599 However, since coercion alone appears to be unable to prevent the invasion of alleles for
600 facultative parthenogenesis, additional factors impeding the development and/or life-history

601 of parthenogenetic mutants may interact with sexually antagonistic selection to maintain
602 obligate sex in natural populations.

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629 **Table 1**

630 **Parameters used in simulations**

Parameter	Description	Parameter levels investigated
Number of patches	The number of patches that make up the world and that therefore determine population density	High (2,601 patches; results in low density populations) Low (121 patches; results in high density populations)
Male mating cost	The number of time-steps deducted from the lifespan of males due to mating	0 5 10 15 20
Female mating cost	The number of time-steps deducted from the lifespan of females due to mating	0 5 10 15 20
Relative efficacy of coercion and resistance	The trait that has the upper hand in sexual encounters	Coercion (i.e., males with the most coercive genotype can mate with any female) Resistance (i.e., females with the most resistant genotype can resist any mating attempt)
Cost of resistance	The number of time-steps deducted from the lifespan of females that successfully resist	0 10
Timing of *P* allele introduction	The time-step at which mutants carrying the *P* allele are introduced into the population	0 10,000
Cost of parthenogenesis	The proportion of fecundity lost by parthenogenetic females relative to sexual females	0 0.5

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Figure 1

Time-dependent changes in population statistics (A and B) and mean realised costs of mating (C and D) for a single simulation per mating-cost combination of equilibrated models.

Left-hand graphs (A and C) are from coercion models; right-hand graphs (B and D) are from resistance models. In upper graphs (A and B), population frequency (in red) is the population size divided by the carrying capacity, with extinction indicated by a frequency of 0. Male sex ratio (in green) is the number of males divided by the population size, with sex ratios above 0.5 indicating male bias. *P* allele frequency (in black) is the number of individuals carrying the P allele divided by the population size. In lower graphs (C and D), realised cost of mating is the mean lifespan penalty due to mating accumulated by females over their lifetime. Arrows indicate the time-step at which the *P* allele is introduced. Other parameter settings are: number of patches: high; cost of resistance: 0; P allele timing: 10,000 (i.e., with equilibration); cost of parthenogenesis: 0.

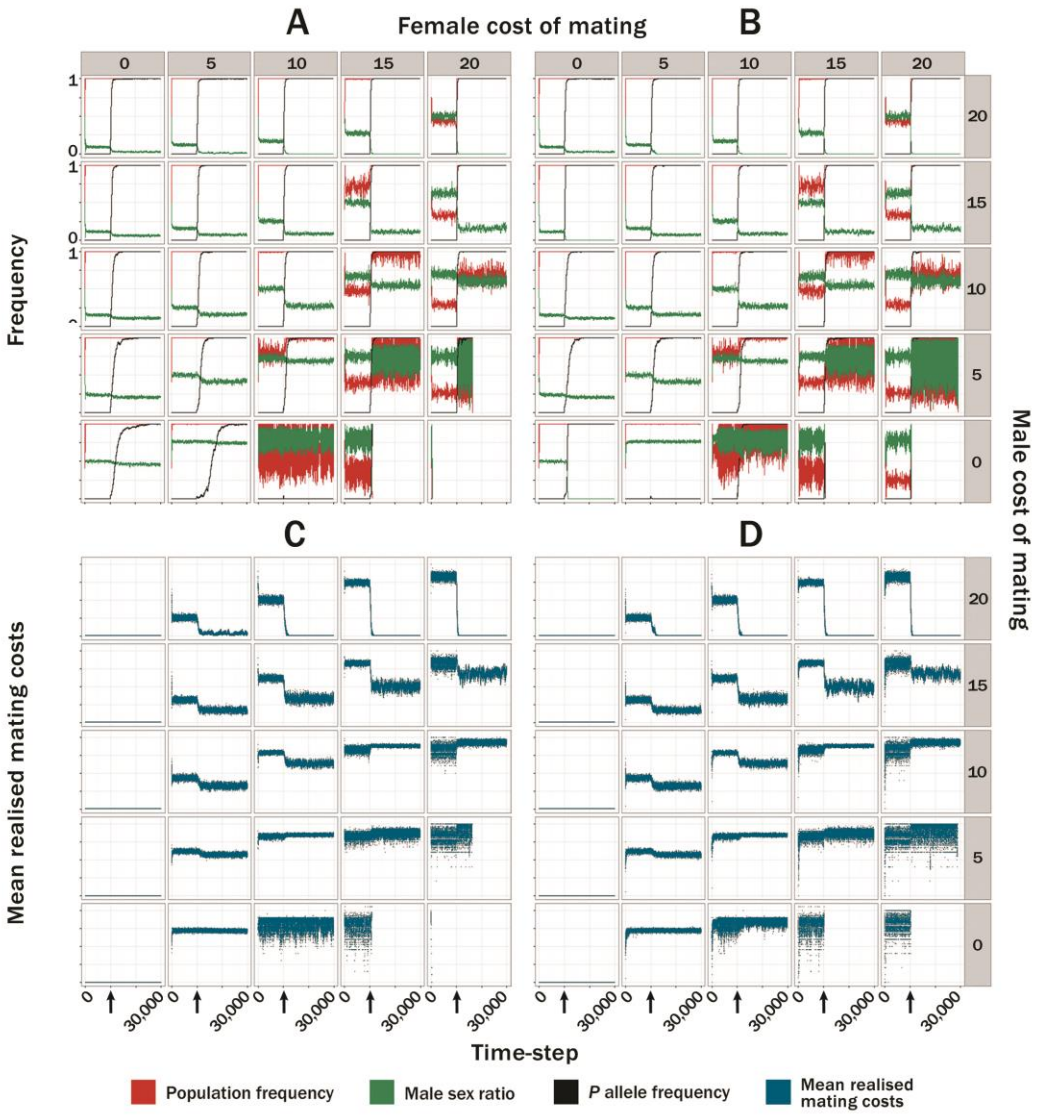


Figure 2

Evolutionary outcomes following the introduction of the *P* allele.

Left-hand graphs (A and C) are from coercion models; right-hand graphs (B and E) are from resistance models. Upper graphs (A and B) show *P* allele outcomes; lower graphs (C and D) show reproductive mode outcomes. The small coloured squares in upper graphs show the proportion of 25 simulation runs per parameter combination ending in *P* fixation (dark orange), *P-p* polymorphism (light orange), *P* extinction (beige), and *P* never arising (white). Note that the *P* allele is always set to arise in non-equilibrated simulations (see *Initialization of simulations* in Methods for details). Coloured squares in lower graphs show the proportion of 25 simulation runs per parameter combination ending in obligate parthenogenesis (dark blue), facultative parthenogenesis (medium blue), obligate sex (light blue), and population extinction (white).

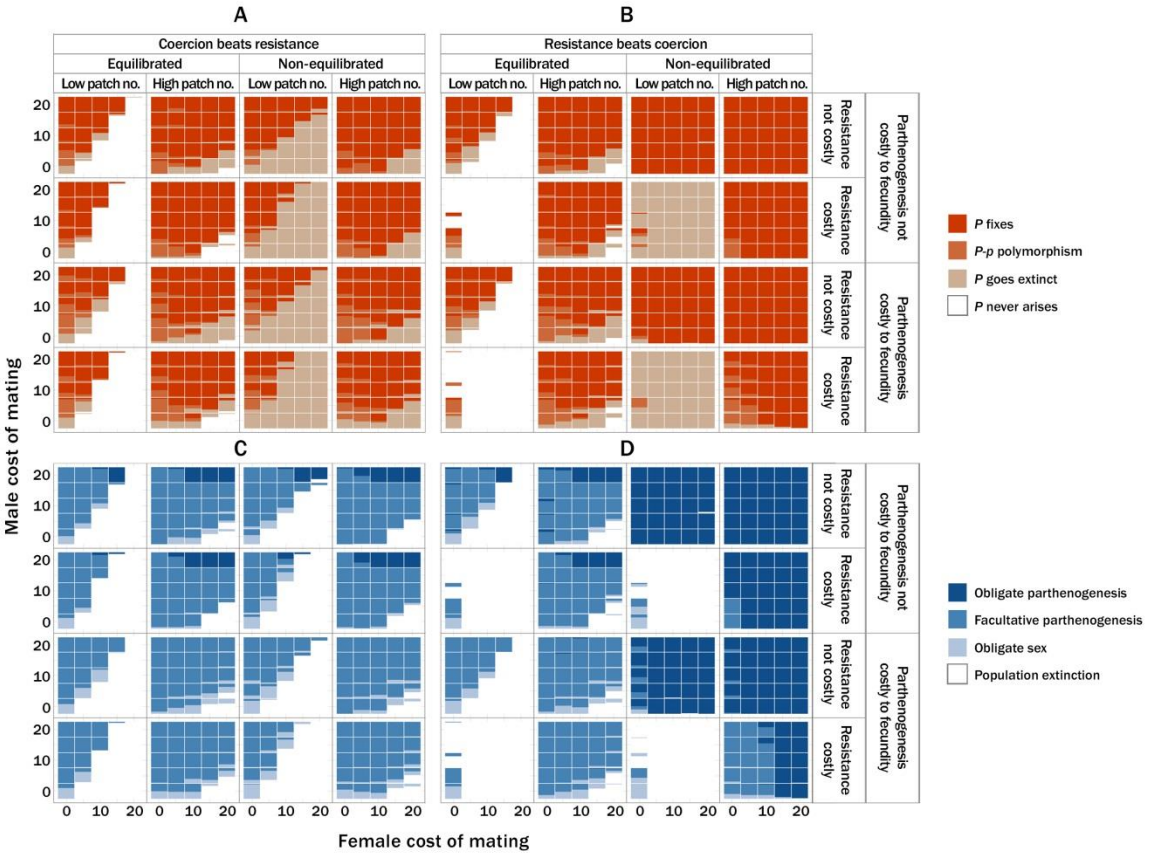


Figure 3

Mean cumulative sum (+ SE) of offspring produced by female genotypes.

Data are obtained from the first 1,200 time-steps of simulations of non-equilibrated resistance models when resistance can beat coercion and linkage disequilibrium is able to build up between *R* and *P*. The parthenogenesis allele *P* is most successful when associated with the most resistant female genotype *RR*. Other parameters are: number of patches: high; cost of resistance: 0; cost of parthenogenesis: 0; female mating cost: 10; male mating cost: 10. N = 25 simulations.

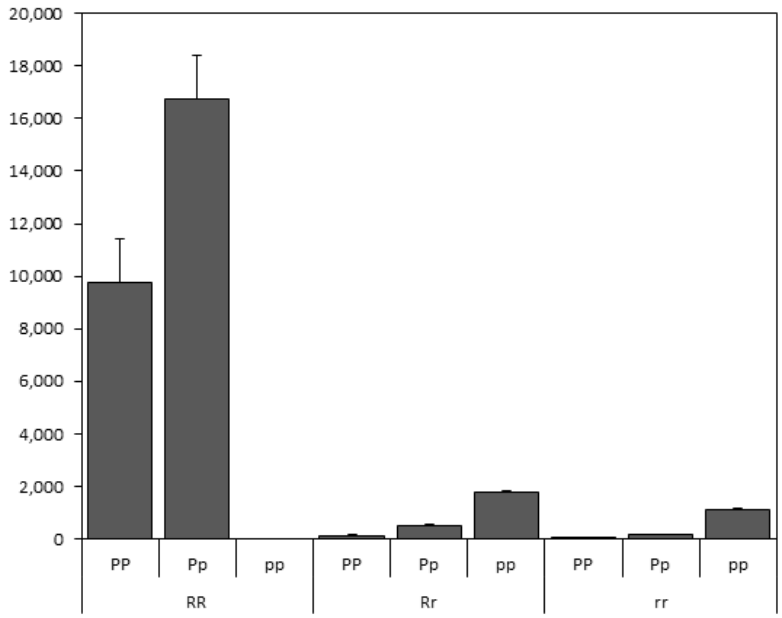


Figure 4

Mean proportion (+ SE) of offspring produced parthenogenetically by females of different resistance genotypes during the first 300 and 1,200 time-steps of simulation runs before and after initial sexual encounters.

Data are from non-equilibrated coercion models (A), and non-equilibrated resistance models (B). Other parameters are as in Figure 3. When coercion has the upper hand (A), most parthenogenetic reproduction occurs before females encounter males (i.e., via the mate scarcity mechanism). This proportion increases as time elapses (contrast the proportion of parthenogenetic reproduction for each genotype before initial sexual encounters during the first 300 time-steps versus the first 1,200 time-steps). By contrast, when resistance has the upper hand (B) during the early stages of invasion (i.e., during the first 300 time-steps), parthenogenetic reproduction occurs more frequently *after* mate encounters; that is, due to the resistance mechanism. But, as invasions progress (i.e., during the first 1,200 time-steps), more parthenogenetic reproduction occurs *before* mate encounters; that is, due to the mate scarcity mechanism. In both cases, *RR* females produce the most offspring parthenogenetically.

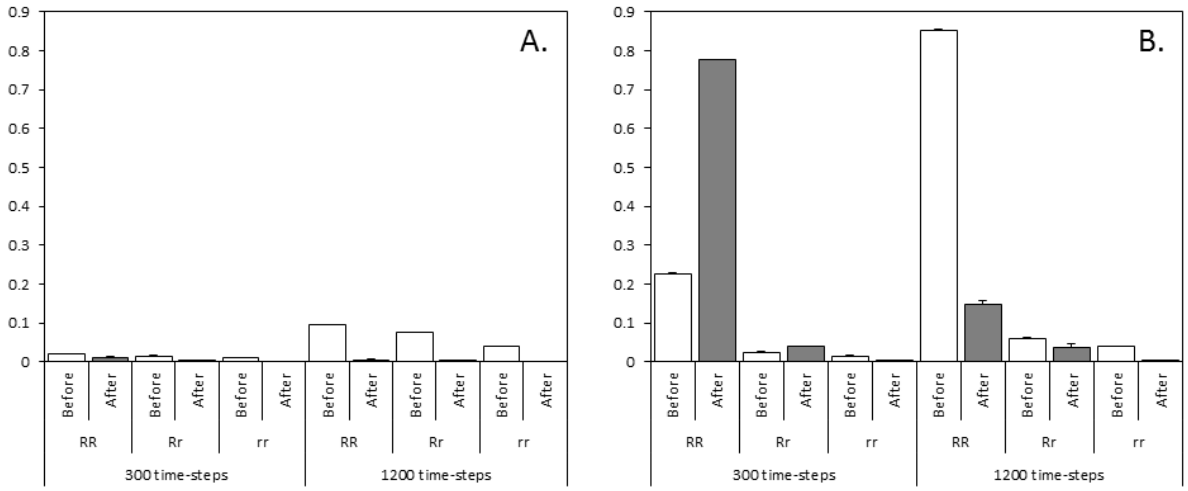


Figure 5

Time-dependent changes in coercion and resistance genotypes for a single simulation per mating-cost combination.

The top graphs (A and B) are from models in which the *P* allele is introduced after equilibration; the lower graphs (C and D) are from models in which the *P* allele is introduced without equilibration at time-step 0. Left-hand graphs (A and C) are from coercion models; right-hand graphs (B and D) are from resistance models. Female resistance genotypes *RR*, *Rr* and *rr* are shown in red, orange and yellow, respectively; male coercion genotypes *CC*, *Cc* and *cc* are shown in dark blue, medium blue and light blue, respectively. Arrows indicate the time-step at which the *P* allele is introduced. Other parameters are as in Figure 1.

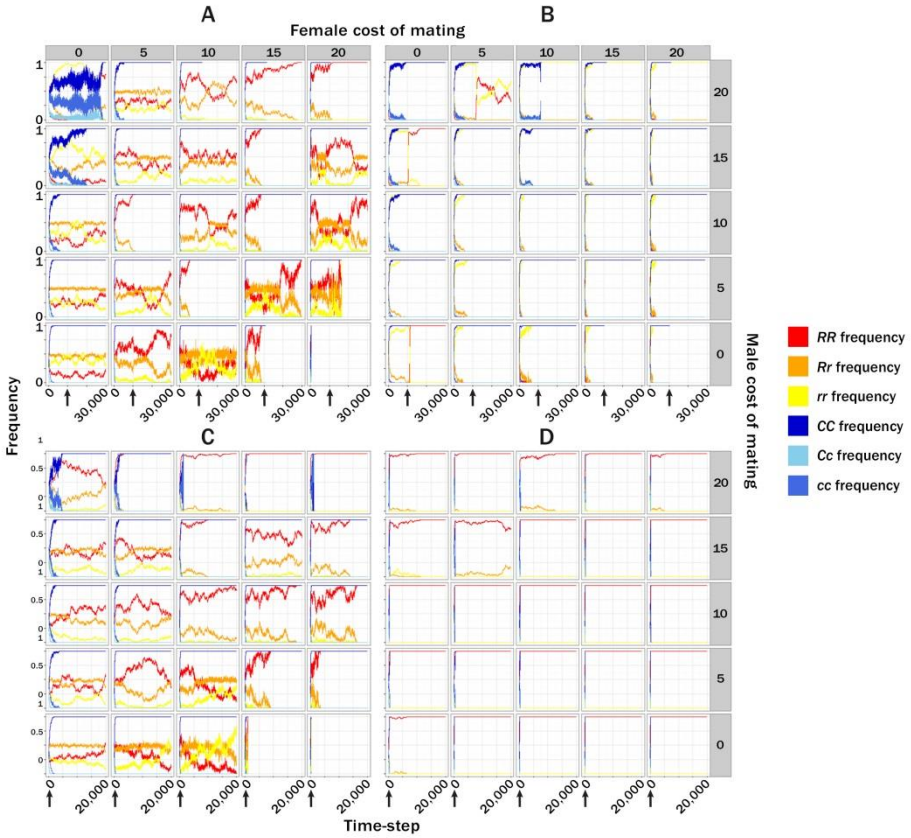
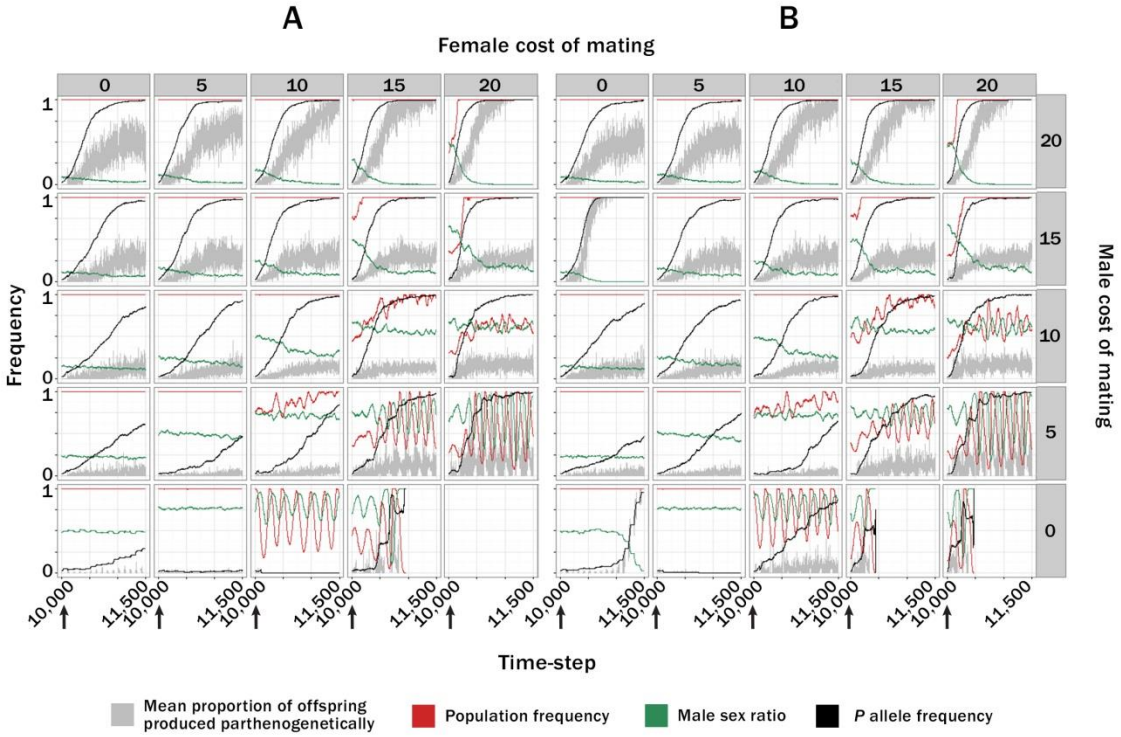


Figure 6

Snapshot of time-dependent changes in population statistics after equilibration for a single simulation per mating-cost combination.

Each graph is a snapshot of Figures 2 *A* and *B*, respectively, between time-steps 10,000 to 11,500. Graph A shows results from coercion models; graph B from resistance models. Population frequency (in red), male sex ratio (in green), and *P* allele frequency (in black) are calculated the same as in Figure 2. The mean proportion of parthenogenetic offspring (in grey) is calculated as the total number of offspring produced parthenogenetically divided by the total number of offspring produced sexually and parthenogenetically. Arrows indicate the time-step at which the *P* allele is introduced. Other parameter settings are the same as in Figure 1. Each graph shows distinct cycling dynamics in population size, sex ratio and realised parthenogenetic reproduction when female mating costs are greater than male mating costs.



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REFERENCES

Abbott, J. K. 2011. Intra-locus sexual conflict and sexually antagonistic genetic variation in hermaphroditic animals. Proceedings of the Royal Society B: Biological Sciences 278:161–169.

Agrawal, A. F. 2001. Sexual selection and the maintenance of sexual reproduction. Nature 411:692–695.

Agrawal, A. F. 2006. Evolution of sex: why do organisms shuffle their genotypes? Current Biology.

Agrawal, A. F., L. Hadany, and S. P. Otto. 2005. The evolution of plastic recombination. Genetics 171:803–812.

Andersson, J., A. K. Borg-Karlson, and C. Wiklund. 2004. Sexual conflict and anti-aphrodisiac titre in a polyandrous butterfly: male ejaculate tailoring and absence of female control. Proceedings of the Royal Society B: Biological Sciences 271:1765–1770.

Arnqvist, G., and L. Rowe. 2005. Sexual Conflict. Princeton University Press, Princeton.

Avise, J. C., J. M. Quattro, and R. C. Vrijenhoek. 1992. Molecular clones within organismal clones: mitochondrial DNA phylogenies and the evolutionary histories of unisexual vertebrates. Evolutionary Biology 26:225–246.

Barton, N. H. 1995. A general model for the evolution of recombination. Genetical Research 65:123–144.

785 Bell, G. 1982. The Masterpiece of Nature : The Evolution and Genetics of Sexuality.
786 University of California Press, Berkeley.

787 ———. 1988. Recombination and the immortality of the germ line. Journal of Evolutionary
788 Biology 1:67–82.

789 Brittain, J. E. 1982. Biology of mayflies. Annual Review of Entomology 27:119–147.

790 Burke, N. W., A. J. Crean, and R. Bonduriansky. 2015. The role of sexual conflict in the
791 evolution of facultative parthenogenesis: a study on the spiny leaf stick insect. Animal
792 Behaviour 101:117–127.

793 Burt, A. 2000. Perspective: sex, recombination, and the efficacy of selection--was Weismann
794 right? Evolution 54:337–351.

795 Carson, H. L. 1967. Selection for parthenogenesis in Drosophila mercatorum. Genetics
796 55:157–171.

797 Chapman, T., L. F. Liddle, J. M. Kalb, M. F. Wolfner, and L. Partridge. 1995. Cost of mating
798 in Drosophila melanogaster females is mediated by male accessory-gland products. Nature
799 373:241–244.

800 Corley, L. S., and A. J. Moore. 1999. Fitness of alternative modes of reproduction :
801 developmental constraints and the evolutionary maintenance of sex. Proceedings of the
802 Royal Society B: Biological Sciences 266:471–476.

803 Crow, J. F. 1999. The omnipresent process of sex. Journal of Evolutionary Biology 12:1023–
804 1025.

805 Dagg, J. L. 2006. Could sex be maintained through harmful males? Oikos 112:232–235.

806 de Visser, J. A. G. M., and S. F. Elena. 2007. The evolution of sex: empirical insights into the
807 roles of epistasis and drift. Nature Reviews Genetics 8:139–49.

808 den Hollander, M., and D. T. Gwynne. 2009. Female fitness consequences of male
809 harassment and copulation in seed beetles, Callosobruchus maculatus. Animal Behaviour
810 78:1061–1070.

811 Engelstadter, J. 2008. Constraints on the evolution of asexual reproduction. Bioessays
812 30:1138–1150.

813 Enghoff, H. 1976. Taxonomic problems in parthenogenetic animals. Zoologica Scripta
814 5:103–104.

815 Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Oxford University Press,
816 Oxford.

817 Flatt, T., N. Maire, and M. Doebeli. 2001. A bit of sex stabilizes host-parasite dynamics.
818 Journal of Theoretical Biology 212:345–54.

819 Gavrilets, S. 2000. Rapid evolution of reproductive barriers driven by sexual conflict. Nature
820 403:886–889.

821 Gerber, N., and H. Kokko. 2016. Sexual conflict and the evolution of asexuality at low
822 population densities. Proceedings of the Royal Society B: Biological Sciences
823 283:20161280.

824 Green, R. F., and D. L. G. Noakes. 1995. Is a little bit of sex as good as a lot. Journal of
825 Theoretical Biology 174:87–96.

826 Hamilton, W. D. 1980. Sex versus non-sex versus parasite. Oikos 35:282–290.

827 Hardling, R., and A. Kaitala. 2005. The evolution of repeated mating under sexual conflict.
828 Journal of Evolutionary Biology 18:106–115.

829 Hartfield, M., and P. D. Keightley. 2012. Current hypotheses for the evolution of sex and
830 recombination. Integrative Zoology 7:192–209.

831 Hedrick, P., and T. Whittam. 1989. Sex in diploids. Nature 342:231.

832 Holland, B., and W. R. Rice. 1998. Perspective: Chase-away sexual selection: Antagonistic
833 seduction versus resistance. Evolution 52:1–7.

834 Hosken, D. J., O. Y. Martin, J. Born, and F. Huber. 2003. Sexual conflict in Sepsis cynipsea:
835 female reluctance, fertility and mate choice. Journal of Evolutionary Biology 16:485–490.

836 Hurst, L. D., and J. R. Peck. 1996. Recent advances in understanding of the evolution and
837 maintenance of sex. Trends in Ecology & Evolution 11:46–52.

838 Joshi, A., and M. E. Moody. 1995. Male gamete output of asexuals and the dynamics of
839 populations polymorphic for reproductive mode. Journal of Theoretical Biology 174:189–197.

840 Joshi, a, and M. E. Moody. 1998. The cost of sex revisited: effects of male gamete output of
841 hermaphrodites that are asexual in their female capacity. Journal of Theoretical Biology
842 195:533–542.

843 Kawatsu, K. 2013*a*. Sexual conflict over the maintenance of sex: effects of sexually
844 antagonistic coevolution for reproductive isolation of parthenogenesis. PLoS One 8:e58141.

845 ———. 2013*b*. Sexually antagonistic coevolution for sexual harassment can act as a barrier
846 to further invasions by parthenogenesis. The American Naturalist 181:223–234.

847 Kawatsu, K. 2015. Breaking the parthenogenesis fertilization barrier: direct and indirect
848 selection pressures promote male fertilization of parthenogenetic females. Evolutionary
849 Ecology 29:49–61.

850 Kondrashov, A. S. 1984. Deleterious mutations as an evolutionary factor. 1. The advantage
851 of recombination. Genetical Research 44:199–217.

852 Kondrashov, A. S. 1993. Classification of hypotheses on the advantage of amphimixis.
853 Journal of Heredity 84:372–387.

854 Kotiaho, J. S. 2001. Costs of sexual traits: a mismatch between theoretical considerations
855 and empirical evidence. Biological Reviews 76:365–376.

856 Kouyos, R. D., O. K. Silander, and S. Bonhoeffer. 2007. Epistasis between deleterious
857 mutations and the evolution of recombination. Trends in Ecology & Evolution.

858 Kramer, M. G., and a R. Templeton. 2001. Life-history changes that accompany the
859 transition from sexual to parthenogenetic reproduction in Drosophila mercatorum. Evolution
860 55:748–761.

861 Ladle, R. J. 1992. Parasites and sex: catching the Red Queen. Trends in Ecology &
862 Evolution.

863 Lamb, R. Y., and R. B. Willey. 1979. Are parthenogenetic and related bisexual insects equal
864 in fertility? Evolution 33:774–775.

865 Lehtonen, J., M. D. Jennions, and H. Kokko. 2012. The many costs of sex. Trends in
866 Ecology & Evolution 27:172–178.

867 Lewis, W. M. 1987. The cost of sex. Pages 33–57 *in* S. Stearns, ed. The Evolution of Sex
868 and its Consequences. Birkhäuser, Basel.

869 Lynch, M. 1984. Destabilizing hybridization, general-purpose genotypes and geographic
870 parthenogenesis. Quarterly Review of Biology 59:257–290.

871 Lynch, M., and W. Gabriel. 1983. Phenotypic evolution and parthenogenesis. The American
872 Naturalist 122:745.

873 Maklakov, A. A., T. Bilde, and Y. Lubin. 2005. Sexual conflict in the wild: elevated mating
874 rate reduces female lifetime reproductive success. The American Naturalist 165 Suppl:S38–
875 S45.

876 Markow, T. A. 2013. Parents Without Partners: Drosophila as a Model for Understanding the
877 Mechanisms and Evolution of Parthenogenesis. G3 3:757–762.

878 Martin, O. Y., and D. J. Hosken. 2003. Costs and benefits of evolving under experimentally
879 enforced polyandry or monogamy. Evolution 57:2765–2772.

880 Maynard Smith, J. 1978. The Evolution of Sex. Cambridge University Press, Cambridge.

881 Meirmans, S., P. G. Meirmans, and L. R. Kirkendall. 2012. The costs of sex: facing real-
882 world complexities. Quarterly Review of Biology 87:19–40.

883 Muller, H. J. 1932. Some genetic aspects of sex. The American Naturalist 66:118–138.

884 Muller, H. J. 1964. The relation of recombination to mutational advance. Mutation
885 Research/Fundamental and Molecular Mechanisms of Mutagenesis 1:2–9.

886 Neiman, M. 2004. Physiological dependence on copulation in parthenogenetic females can
887 reduce the cost of sex. Animal Behaviour 67:811–822.

888 Neiman, M., T. F. Sharbel, and T. Schwander. 2014. Genetic causes of transitions from
889 sexual reproduction to asexuality in plants and animals. Journal of Evolutionary Biology.

890 Normark, B. B., and L. R. Kirkendall. 2009. Parthenogenesis in insects and mites. Pages
891 753–757 *in*Encyclopedia of Insects. Academic Press, London.

892 Otto, S. P. 2003. The advantages of segregation and the evolution of sex. Genetics
893 164:1099–1118.

894 ———. 2009. The evolutionary enigma of sex. The American Naturalist 174:S1–S14.

895 Otto, S. P., and J. Whitton. 2000. Polyploid incidence and evolution. Annual Review of
896 Genetics 34:401–437.

897 Parker, G. A. 1979. Sexual selection and sexual conflict. Pages 123–166 *in* N. Blum and M.
898 Blum, eds. Sexual Selection and Reproductive Competition in Insects. Academic Press, New
899 York.

900 Parker, G. A., and T. R. Birkhead. 2013. Polyandry: the history of a revolution. Philosophical

Transactions of the Royal Society B: Biological Sciences 368:20120335–20120335.

Rankin, D. J., K. Bargum, and H. Kokko. 2007. The tragedy of the commons in evolutionary biology. Trends in Ecology & Evolution.

Reik, W., and J. Walter. 2001. Genomic imprinting: parental influence on the genome. Nature Reviews Genetics 2:21–32.

Rice, W. R. 2002. Experimental tests of the adaptive significance of sexual recombination. Nature Reviews Genetics 3:241–51.

Rice, W. R., and B. Holland. 1997. The enemies within: intergenomic conflict, interlocus contest evolution (ICE), and the intraspecific Red Queen. Behavioral Ecology and Sociobiology.

Ringo, J. 1996. Sexual receptivity in insects. Annual Review of Entomology 41:473–494.

Rowe, L. 1992. Convenience polyandry in a water strider: foraging conflicts and female control of copulation frequency and guarding duration. Animal Behaviour 44:189–202.

Rowe, L., G. Arnqvist, A. Sih, and J. J. Krupa. 1994. Sexual conflict and the evolutionary ecology of mating patterns: Water striders as a model system. Trends in Ecology & Evolution 9:289–293.

Roze, D. 2012. Disentangling the benefits of sex. PLoS Biology 10:e1001321.

Sasaki, A., and Y. Iwasa. 1987. Optimal recombination rate in fluctuating environments. Genetics 115:377–388.

Schärer, L., T. Janicke, and S. A. Ramm. 2015. Sexual conflict in hermaphrodites. Cold Spring Harbor Perspectives in Biology 7:a017673.

Schwander, T., and B. J. Crespi. 2009. Multiple direct transitions from sexual reproduction to apomictic parthenogenesis in Timema stick insects. Evolution 63:84–103.

924 Schwander, T., S. Vuilleumier, J. Dubman, and B. J. Crespi. 2010. Positive feedback in the
925 transition from sexual reproduction to parthenogenesis. Proceedings of the Royal Society B:
926 Biological Sciences 277:1435–1442.

927 Siller, S. 2001. Sexual selection and the maintenance of sex. Nature 411:689–692.

928 Simon, J. C., F. Delmotte, C. Rispe, and T. Crease. 2003. Phylogenetic relationships
929 between parthenogens and their sexual relatives: the possible routes to parthenogenesis in
930 animals. Biological Journal of the Linnean Society 79:151–163.

931 Singh, R. S., and C. G. Artieri. 2010. Male sex drive and the maintenance of sex: evidence
932 from Drosophila. Journal of Heredity 101:S100–S106.

933 Sloan, D. B., and V. G. Panjeti. 2010. Evolutionary feedbacks between reproductive mode
934 and mutation rate exacerbate the paradox of sex. Evolution 64:1129–1135.

935 Smith, R. L. 1984. Sperm Competition and the Evolution of Animal Mating Systems.
936 Academic Press, New York.

937 Stalker, H. D. 1956. On the evolution of parthenogenesis in Lonchoptera (Diptera). Evolution
938 10:345–359.

939 Stricker, S. A. 1999. Comparative biology of calcium signaling during fertilization and egg
940 activation in animals. Developmental Biology 211:157–176.

941 Suomalainen, E., A. Saura, and J. Lokki. 1987. Cytology and Evolution in Parthenogenesis.
942 CRC Press, Boca Raton.

943 U. Wilensky. 1999. NetLogo. Center for Connected Learning and Computer-Based
944 Modeling, Northwestern University, Evanston.

945 Vrijenhoek, R. C. 1989. Genetic and evolutionary constraints on the origin and establishment
946 of unisexual vertebrates. *in* R. Dawley and J. Bogart, eds. Evolution and Ecology of
947 Unisexual Vertebrates. University of the State of New York, New York.

948 Wagner, G. P., and W. Gabriel. 1990. Quantitative variation in finite parthenogenetic
949 populations : what stops Muller's ratchet in the absence of recombination? Evolution 44:715–
950 731.

951 Wedell, N., C. Kvarnemo, C. M. Lessells, and T. Tregenza. 2006. Sexual conflict and life
952 histories. Animal Behaviour 71:999–1011.

953 Weismann, A. 1889. The significance of sexual reproduction in the theory of natural
954 selection. Essays Upon Heredity and Kindred Biological Problems. Clarendon Press, Oxford.

955 West, S. A., C. M. Lively, and A. F. Read. 1999. A pluralist approach to sex and
956 recombination. Journal of Evolutionary Biology 12:1003–1012.

957 White, M. J. D. 1973. Animal Cytology and Evolution (3d ed.). Cambridge University Press,
958 Cambridge.

959 Wigby, S., and T. Chapman. 2005. Sex peptide causes mating costs in female Drosophila
960 melanogaster. Current Biology 15:316–321.

961 Williams, G. C. 1975. Sex and Evolution. Princeton University Press, Princeton.

962 Yamauchi, A. 1999. Evolution of cyclic sexual reproduction under host-parasite interactions.
963 Journal of Theoretical Biology 201:281–291.

964 Yamauchi, A., and Y. Kamite. 2003. Facultative sexual reproduction under frequency-
965 dependent selection on a single locus. Journal of Theoretical Biology 221:411–424.

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APPENDIX

Continuous-trait model

In an additional IBM (hereafter, "continuous-trait model"), we treat coercion and resistance as continuously distributed values, corresponding to a genetic architecture involving many loci of small effect. This model allows for long-term evolution with unlimited escalation of coercion and resistance via mutation and selection, such that neither sex can permanently gain the upper hand in sexual coevolution. All other parameters are set up as in the discrete-trait models described in Methods, the only difference between the continuous-trait model and the discrete-trait (i.e., coercion and resistance) models being in the way sexually antagonistic traits are modelled and inherited. At the start of continuous-trait model simulations, individuals of each sex are given random trait values for coercion and resistance obtained from a normal distribution of mean 0 and standard deviation 1. Offspring inherit the averaged coercion and resistance values of their parents' genotypes at the relevant locus +/- a mutational deviation: offspring genotype is determined by drawing a random value from a normal distribution centred at the mean of the parental genotypic values with a standard deviation of 1. This method of modelling mutation assumes that genetic variance is not diminished by persistent selection, and is therefore not realistic for a moderate number of loci. However, we used this approach in order to maximize evolvability of male coercion and female resistance traits, thereby allowing for a sexual arms race unconstrained by short-term limitations on genetic variance.

We ran 25 simulations of each of 400 unique parameter combinations of the continuous-trait model, and collected the same data as described for discrete-trait models. In an additional run, we collected mean antagonistic trait values for each sex along with the other statistics described in Methods.

We found that, in continuous-trait simulations, *P* allele invasion was intermediate in frequency compared to the two discrete-trait models (compare and contrast Figures 2 *A*, 2 *B*

and A1 *A*). The proportion of simulations ending in obligate parthenogenesis was also intermediate (compare and contrast Figures 2 *C*, 2 *D* and A1 *B*). These intermediate results emerged because of the dynamics of chase-away sexual coevolution: every coevolutionary step by one sex further escalated the arms race and selected for counter-adaptation in the other sex, such that coercion was never able to completely subdue resistance, or vice versa.

As in discrete-trait models, the timing of the *P* allele's introduction in the continuous-trait model was an important determiner of evolutionary outcomes, with frequent transitions to obligate parthenogenesis occurring in non-equilibrated versions of the model when high resistance genotypes were able to associate with the *P* allele, and especially when parthenogenesis bore no cost (Figure A1 *B*). However, the negative effect of costly resistance on extinction rates was not as severe in the continuous-trait model as in the resistance model (contrast Figures 2 *D* and A1 *B*). This is because escalating sexual coevolution produced more diverse resistance and coercion genotypes than did sexual coevolution resolved in favour of females. This greater variation in female ability to resist also explains why *P* allele invasions and transitions to obligate parthenogenesis were less frequent in the continuous-trait model compared to the resistance model (contrast Figures 2 *B* and *D* with Figures A1 *A* and *B*, respectively): escalating arms races prevented fixation of permanently high resistance genotypes by facilitating the continuous production of ever-more coercive males. With females unable to decisively gain the upper hand, the ability of linkage disequilibrium between resistance and the *P* allele to drive males extinct was reduced.

In equilibrated versions of the continuous-trait model, sexual conflict always decreased after the introduction of the *P* allele, unless the allele died out (see the drop in mean realised costs of mating for females after the introduction of *P* in Figure A2 *B*). At any given time-step, chase-away antagonistic coevolution generated at least some females with high resistance that could resist some matings, and the *P* allele allowed some of these high-resistance females to reproduce parthenogenetically. Hence, invasions of facultative

parthenogenesis in the continuous-trait model universally reduced sexual conflict, regardless of the relative cost of mating for females, because effective resistance could always evolve to reduce mating rates.

These results suggest that the genetic architecture of sexual coevolution has qualitatively similar effects on the invasion success of facultatively parthenogenetic mutants regardless of whether sexual coevolution is resolved in favour of one of the sexes or is unresolved in a continuously escalating arms race.

Figure A1

Evolutionary outcomes of the continuous-trait model following the introduction of the *P* allele.

Graph A shows *P* allele outcomes; graph B shows reproductive mode outcomes. The small coloured squares in graph A show the proportion of 25 simulation runs per parameter combination ending in *P* fixation (dark orange), *P-p* polymorphism (light orange), *P* extinction (beige), and *P* never arising (white). Note that the *P* allele is always set to arise in non-equilibrated simulations (see *Initialization of simulations* in Methods for details). Coloured squares in graph B show the proportion of 25 simulation runs per parameter combination ending in obligate parthenogenesis (dark blue), facultative parthenogenesis (medium blue), obligate sex (light blue), and population extinction (white).

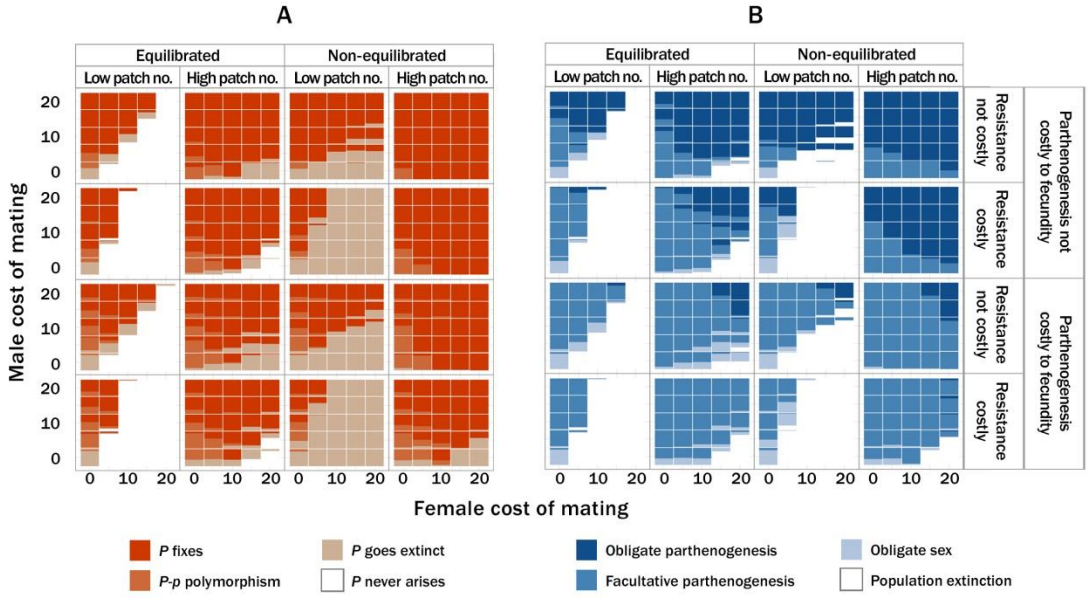

Figure A2

Time-dependent changes in population statistics (A) and mean realised costs of mating (B) for a single simulation per mating-cost combination of the equilibrated version of the continuous-trait model.

Graph A shows population frequency (in red), male sex ratio (in green), and *P* allele frequency (in black). Graph B shows the mean lifespan penalty due to mating accumulated by females over their lifetime. Arrows indicate the time-step at which the *P* allele is introduced. Other parameter settings are: number of patches: high; cost of resistance: 0; P allele timing: 10,000 (i.e., with equilibration); cost of parthenogenesis: 0.