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The paradox of obligate sex: The roles of sexual conflict and mate scarcity in transitions to facultative and obligate asexuality

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Abstract

The maintenance of obligate sex in animals is a long-standing evolutionary paradox. To solve this puzzle, evolutionary models need to explain why obligately sexual populations consistently resist invasion by facultative strategies that combine the benefits of both sexual and asexual reproduction. Sexual antagonism and mate availability are thought to shape the occurrence of reproductive modes in facultative systems. But it is unclear how such factors interact with each other to influence facultative invasions and transitions to obligate asexuality. Using individual-based models, we clarify how sexually antagonistic coevolution and mate availability affect the likelihood that a mutant allele that gives virgin females the ability to reproduce parthenogenetically will invade an obligately sexual population. We show that male coercion cannot stop the allele from spreading because mutants generally benefit by producing at least some offspring asexually prior to encountering males. We find that effects of sexual conflict can lead to positive frequency-dependent dynamics, where the spread of the allele is promoted by effective (no-cost) resistance when males are common, and by mate limitation when sex ratios are female-biased. However, once the mutant allele fixes, effective coercion prevents the complete loss of sex unless linkage disequilibrium can build up between the allele and alleles for effective resistance. Our findings clarify how limitations of female resistance imposed by the genetic architecture of sexual antagonism can promote the maintenance of sexual reproduction. At the same time, our finding of widespread obligate sex when costs of parthenogenesis are high suggests that developmental constraints could contribute to the rarity of facultative reproductive strategies in nature.

KEYWORDS

developmental costs, evolution of sex, facultative parthenogenesis, individual-based model, invasion dynamics, loss of sex, mate limitation, paradox of obligate sex, sexually antagonistic coevolution

1 | INTRODUCTION

The continued maintenance of costly obligate sexual reproduction in some groups of organisms, especially animals, represents a paradox. Theory suggests that facultative strategies that incorporate both sexual and asexual reproduction provide all the genetic advantages of obligate sex but with much lower costs (D'Souza & Michiels, 2010; Green & Noakes, 1995). For example, facultative sex/asex is as effective as obligate sex at enhancing purifying selection (Lynch & Gabriel, 1983; Wagner & Gabriel, 1990), creating advantageous allele combinations (Bell, 1988; Hurst & Peck, 1996; Kondrashov, 1984), promoting adaptation (Lynch & Gabriel, 1983; Sasaki & Iwasa, 1987) and facilitating evolutionary escape from coevolving parasites (Flatt, Maire, & Doebeli, 2001; Yamauchi, 1999; Yamauchi & Kamite, 2003). Thus, to explain the paradox of obligate sex, theory must account for the capacity of obligately sexual populations to resist invasions by facultatively asexual mutants (Burke & Bonduriansky, 2017).

Although most primitive eukaryotes are capable of both sexual and asexual reproduction (Dunthorn & Katz, 2010; Lahr, Parfrey, Mitchell, Katz, & Lara, 2011; Speijer, Lukeš, & Eliáš, 2015), facultative strategies are very rare in higher eukaryotes. For some metazoans—especially mammals and birds—the capacity to reproduce asexually at all is likely permanently impeded by genetic or developmental constraints, such as genomic imprinting, diploidy restoration and female heterogamety, that interfere with parthenogenetic development (Engelstadter, 2008; Neiman, Sharbel, & Schwander, 2014). Nevertheless, the vast majority of animal groups—including amphibians, annelids, arthropods, bryozoans, cnidarians, echinoderms, fishes, molluscs, nematodes, platyhelminths, reptiles and rotifers—possess at least some asexual taxa (Bell, 1982), demonstrating an in-principle capacity to overcome constraints on parthenogenesis. Thus, the widespread occurrence of obligate sex in lineages for which parthenogenesis is possible suggests the existence of additional mechanisms or dynamics that act as persistent impediments to evolutionary transitions from obligate sex to facultative or obligate asexuality.

One possible mechanism that could impede such transitions is coercion by males (Burke & Bonduriansky, 2017; Burke, Crean, & Bonduriansky, 2015; Gerber & Kokko, 2016; Kawatsu, 2013a, 2013b, 2015). Males typically have a higher optimum mating rate than females, and selection therefore favours male strategies that coerce females into mating, even if it results in reduced female fecundity or longevity (Arnqvist & Rowe, 2005; Maklakov, Bilde, & Lubin, 2005; Martin & Hosken, 2003a; Parker, 1979). In a sexual population, a mutant allele that makes parthenogenetic reproduction possible may be expected to flourish due to the demographic advantage of producing all-female offspring (Maynard Smith, 1978), as well as the ecological and physiological advantages of reproduction without costs of mating (Williams, 1975). However, coercive males could directly inhibit the spread of parthenogenesis by forcing facultative mutants to reproduce sexually, since in many facultatively asexual diploid animals only virgin females are capable of parthenogenesis (Bell, 1982). Parthenogenesis may also be impeded if facultative mutants lose more fitness than wild-type females after encountering males (Burke et al., 2015; Kawatsu, 2013b; Schartl et al., 1997). However, male coercion can select for female resistance, potentially setting off a sexual "arms race" (Gavrilets, 2000; Holland & Rice, 1998; Rice & Holland, 1997). A number of recent studies suggest that females' capacity for effective resistance could play an important role in counteracting the suppressive effect of coercion on parthenogenetic reproduction (Burke & Bonduriansky, 2017; Burke et al., 2015; Gerber & Kokko, 2016; Kawatsu, 2013a). However, important alternative sources of selection that favour parthenogenesis should also be considered.

Conditions that lead to mate scarcity—such as low density or skewed sex ratio—can strongly favour parthenogenetic strategies because of the reproductive assurance that asexual reproduction provides when females fail to mate (Brittain, 1982; Gerritsen, 1980; Johnson, 1994; Markow, 2013; Schwander & Crespi, 2009). The ability to reproduce prior to encountering males could modulate effects of sexual conflict on parthenogenetic reproduction, but it is unclear how these factors interact. A recent theoretical analysis of the effect of density on sexual antagonism in facultative systems found that resistance can favour parthenogenesis at low density (i.e., low conflict) because females pay fewer costs of resistance when mates are infrequently encountered (Gerber & Kokko, 2016). But this analysis did not explicitly distinguish between parthenogenesis occurring before versus after mate encounters, and therefore the roles of mate scarcity and sexual antagonism were not fully disentangled. A subsequent model incorporating environmental gradients suggested that female resistance and mate scarcity could work in tandem to shape local distributions of reproductive mode in facultative systems (Burke & Bonduriansky, 2018b). However, a key question that remains unanswered is how sexual antagonism and mate scarcity interact to promote or prevent facultative strategies invading (ancestral) obligately sexual populations in the first place.

The emergence of a facultative mutant in a genetic background of high resistance could feasibly lead to the spread of alleles for facultative parthenogenesis through the resistance-mediated production of daughter parthenogens, causing a female skew to the sex ratio. With fewer males around to initiate mating attempts, parthenogenesis might then increasingly occur via mate limitation rather than resistance, potentially driving males extinct (see Burke & Bonduriansky, 2018b). However, in the absence of effective resistance, facultative mutants would be able to reproduce parthenogenetically only if they failed to mate by chance (i.e., due to localized mate scarcity), thereby limiting the spread of alleles for facultative parthenogenesis. These dynamics are likely to be modulated by the economics of resistance and parthenogenesis, and by ecological factors like density that influence rates of inter-sexual encounter.

To test these predictions, we used individual-based models (IBMs) to determine whether (a) the potential for evolution of effective female resistance under sexual conflict (i.e., high genetic variance for resistance alleles that can allow females to avoid costly mating despite encountering males) can promote successful invasion

of facultative strategies; (b) the ability of facultative mutants to reproduce without encountering males (i.e., under conditions of mate scarcity) can also promote invasion; and (c) these processes can interact. Furthermore, we asked under what conditions invasion of facultative mutants can lead to the complete loss of sex through the extinction of males. We addressed these aims by varying sexual conflict intensity (i.e., the cost of mating for females), the timing of mutant introduction (i.e., mutants arising while genetic variance for female resistance was high versus after genetic variance was depleted), the cost of resistance, the relative fecundity achieved via parthenogenetic reproduction and population density (which determines the probability of male encounter).

2 | MATERIALS AND METHODS

2.1 | Overview of IBMs

We employed a series of individual-based simulation models (IBMs) in the program NetLogo (Wilensky, 1999). We consider a finite population of diploid organisms with overlapping generations inhabiting a gridded environment of square patches in a torus-shaped world. This explicit spatial structure enabled us to create high and low population densities—which are known to affect selection on reproductive modes in facultative systems (Gerber & Kokko, 2016). We did this by setting the number of patches to low (11 × 11) and high (51 × 51), respectively. The low-density setting simulates conditions that organisms at high risk of mating failure are likely to experience in wild populations (Greenway, Dougherty, & Shuker, 2015), and for which, in principle, facultative parthenogenesis would be strongly beneficial for reproductive assurance (Gerritsen, 1980), whereas the high-density setting generates high rates of male-female encounter, which reduces mating failure but promotes sexual conflict (e.g., see Lauer, Sih, & Krupa, 1996; Martin & Hosken, 2003b).

The sexes in our models experience sexual conflict over mating rate, such that mating more than once is costly for females but mating multiply is beneficial for males—a scenario typical of many arthropods (Arnqvist & Nilsson, 2000; Arnqvist & Rowe, 2005; Maklakov & Lubin, 2004; Maklakov et al., 2005). We model sexual antagonism in two ways. First, we define 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. This means that coercion genotypes *cc*, *Cc* and *CC* are only expressed phenotypically in males, and resistance genotypes *rr*, *Rr* and *RR* are only expressed phenotypically in females. This "discrete-trait" model is set up as an evolutionary game where the outcome of a sexual interaction (mating or not mating) is determined by the phenotypic strength of the male's coercion genotype compared to the female's resistance genotype. Because either males or females can have an advantage in such inter-locus sexual conflict, we consider two scenarios where either males or females can gain the upper hand over the other sex. These scenarios differ in the phenotypic effects of coercion and resistance alleles: in the first scenario (hereafter "coercion" model), the phenotypic strength of coercion alleles is higher than that of resistance alleles, allowing for males to evolve to always successfully coerce matings, whereas in the second scenario (hereafter "resistance" model), the phenotypic expression of coercion alleles is relatively weaker than that of resistance alleles and so females have the potential to evolve to always successfully resist male coercion (see Figure 1). Sexually antagonistic coevolution in these "discrete-trait" models occurs via selection on standing genetic variation at resistance and coercion loci (i.e., we assume that there is no mutation at these loci), allowing populations to stabilize at coercion-dominated or resistance-dominated states. Second, in a separate model ("continuous-trait" model), we simulate an escalating arms race where coercion and resistance are treated as continuously distributed values (representing a large mutational target) and allowed to coevolve without limit via selection on both standing and mutational variation (see Supporting Information).

We assume that a single sex-limited autosomal locus with two alleles, *p* and *P*, controls reproductive mode. Wild-type *pp* individuals are capable only of sexual reproduction, whereas *P* is a dominant mutant allele that allows females to reproduce prior to mating (i.e., via facultative parthenogenesis). This architecture is motivated by known genetic architectures of asexual reproduction in plants and animals (reviewed in ref. Neiman et al., 2014). The *P* allele is introduced either at time-step 0 before the sexes can coevolve, or in a one-off mutation event at time-step 10,001 after sexually antagonistic selection has altered coercion and resistance allele frequencies. We hereafter refer to "discrete-trait" models set up this way as incorporating "no prior sexual coevolution" versus "prior sexual coevolution", respectively. The *P* allele arises in a random sample of individuals, with 1% of males and 1% of females becoming *PP*, and 1% of males and 1% of females becoming *Pp*. This frequency of *P* alleles limits the extinction of facultative mutants due to drift.

(a) "Coercion" model

Male genotype \ Female genotype	*RR*	*Rr*	*rr*
CC	Mating	Mating	Mating
Cc	No mating	Mating	Mating
cc	No mating	No mating	Mating

(b) "Resistance" model

Male genotype \ Female genotype	*RR*	*Rr*	*rr*
CC	No mating	Mating	Mating
Cc	No mating	No mating	Mating
cc	No mating	No mating	No mating

FIGURE 1 Mating outcomes when males and females of different antagonistic genotypes encounter each other in the discrete-trait "coercion" model (a) and "resistance" model (b). Mating is determined by the relative phenotypic strength of male and female antagonistic genotypes, which differs between "coercion" and "resistance" models. For instance, all male genotypes can mate with at least one female genotype in the "coercion" model, whereas all female genotypes can resist mating with at least one male genotype in "resistance" models

2.2 | Initialization of simulations

Simulations start with 500 females and 500 males randomly distributed across patches, each with a fixed lifespan of 100 time-steps and an age of 0 that increases by 1 every time-step. In "discrete-trait" models, males and females are both allocated coercion and resistance genotypes according to Hardy–Weinberg probabilities and linkage equilibrium, with an allele frequency of 0.5. The carrying capacity of the environment (i.e., maximum population size) is 2,500.

2.3 | Life cycle

During each time-step, individuals perform tasks in four ordered phases: moving, mating, reproducing and dying. In the moving phase, individuals turn to face a new direction between 0 and 90° relative to their current direction, which is decided by drawing a random number from a uniform distribution with limits 0 and 90. Individuals 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 within a time-step by more than one male if successive mating attempts in a time-step are unsuccessful. Thus, females can mate only once per time-step, but can mate multiply over their lifetime. Mating occurs when a male's coercion genotype beats the resistance genotype of the female (see Figure 1).

Since the economics of resistance can have profound effects on how selection operates on mating strategies (Rowe, 1992; Thornhill & Alcock, 1983), we examine the potential influence of these effects on the spread of parthenogenesis by modelling resistance as either costly or noncostly 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. Following convention (e.g., Härdling & Kaitala, 2005), we assume that females store enough sperm from one mating to fertilize all their eggs until the end of their life. We also assume that a female's last mate sires her subsequent offspring, as occurs in many insects (reviewed in ref. Parker, 1970).

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. Mating more than once is costly for wild-type females when female mating cost is >0, whereas male fitness increases with each additional mating regardless of the cost to males. Cost of mating to females therefore reflects the intensity of sexual conflict, such that the risk of death increases linearly with each additional mating. Such costs could be mediated by increased exposure to venereal diseases (Knell & Mary Webberley, 2004; Thrall, 1996), greater somatic damage during copulation (Reinhardt, Anthes, & Lange, 2015) or higher risk of predation (Rowe, 1994).

Reproduction is modelled as a lottery that occurs every time-step if the current population size is less than the carrying capacity. Reductions in population size occur due to mortality in the previous time-step. Females that win the lottery reproduce sequentially (i.e., one random female after the other). The lottery stops if the number of offspring produced causes the population size to equal the carrying capacity, and the remaining lottery winners do not get to reproduce. If the lottery is triggered, each female capable of reproducing is allotted a random number from a uniform distribution between 0 and 1. 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 globally set at either 0.1 (as for previously mated females), 0.05 or 0.005. The last two probabilities represent 50% and 95% reduced parthenogenetic fecundity, respectively, reflecting a low and high genetic/developmental constraint on asexuality (e.g., see Corley, Blankenship, Moore, & Moore, 1999; Corley & Moore, 1999; Engelstadter, 2008; Lamb & Willey, 1979). Females can win the reproductive lottery multiple times but only produce one offspring per reproductive bout, resulting in continuous reproduction over the lifetime. Mated females produce daughters and sons with equal probability, whereas parthenogenesis results in daughters only. As occurs in many facultatively parthenogenetic taxa (Bell, 1982), females that mate reproduce sexually thereafter, even if they carry the *P* allele. We assume no cost of switching from one reproductive mode to the other. Although sexual recombination can provide long-term genetic advantages (Hamilton, 1980; Kondrashov, 1988; Otto & Barton, 1997; Peck & Waxman, 2000), we ignore these potential benefits to focus solely on short-term invasion dynamics.

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; Simon, Delmotte, Rispe, & Crease, 2003), and therefore inherit their mothers' complete genotype. Sexually produced offspring inherit parental alleles following Mendelian rules of segregation.

Following reproduction, an individual's survival value, *S*, is determined as:

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

where *a* is an individual's current age in time-steps, *b* 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 $S \leq 0$.

2.4 | Analysis

We performed 25 simulation runs for each unique parameter combination of "discrete-trait" and "continuous-trait" models 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 (male extinction) or population extinction. In one additional run, we collected data every time-step on population size, sex ratio, mean lifetime mating costs, coercion and resistance genotype frequencies,

and number of offspring to inform our interpretations. All simulation runs lasted 20,000 time-steps following the emergence of the *P* allele, except in cases of prior population extinction. Because outcomes for both "discrete-trait" and "continuous-trait" models were broadly similar, we focus below on "discrete-trait" models and briefly compare results for those models with outcomes from the "continuous-trait" model. A detailed description of and full results for the "continuous-trait" model are provided in the Supporting Information. A list of all model parameters used in "discrete-trait" models is provided in Table 1.

In "discrete-trait" models prior to the introduction of the *P* allele, relative costs of mating for each sex had consistent demographic effects, with higher costs for one sex generating strongly biased sex ratios. Since large deviations from equal sex ratio are likely to represent extreme cases, we focus below on simulations where male and female costs of mating are balanced, and sex ratios therefore remain initially approximately equal. Nonetheless, we report *P* allele and reproductive mode outcomes for all mating cost combinations in Figure S1.

3 | RESULTS

3.1 | Conditions for the invasion of the *P* allele

We find that the *P* allele spreads via two interacting 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 versions of the "discrete-trait" model. Even in the "coercion" models where males can evolve to coerce any female to mate, mate scarcity promotes the spread of the *P* allele because at least some virgin females reproduce parthenogenetically prior to encountering males. Separate analyses (not shown) confirm that the *P* allele spreads because of this general fecundity advantage and not due to drift. This suggests that, under our assumptions, sexual conflict mediated by male coercion cannot impede the invasion of a facultative strategy.

Nonetheless, sexual antagonism has dynamic effects on invasions. In resistance models with no prior coevolution, selection for resistance generates strong epistasis for fitness in unmated females (whereby the *R* allele enhances female fitness in the presence of the *P* allele, but not otherwise), which leads to the build-up of positive linkage disequilibrium between the *R* allele and the *P* allele (especially when resistance is cost-free; Figure 2), creating a feedback loop of resistance-mediated parthenogenesis. Both resistance and mate scarcity contribute to this feedback loop in important complementary ways: resistance promotes the spread of parthenogenesis during initial stages of invasion (evidenced by the higher proportion of offspring produced after than before encounters with males in early time-steps in Figure 3b), whereas mate scarcity is the dominant driver as more and more parthenogens are produced (see the higher proportion of offspring produced before than after encounters in later time-steps in Figure 3b). These patterns are indicative of positive frequency-dependent selection, such that resistance favours parthenogenesis when males are common, whereas mate scarcity favours parthenogenesis when females are more abundant. Assessing the "coercion" model with no prior sexual coevolution reveals a slightly different invasion dynamic. Resistance partially contributes to the spread of the *P* allele immediately following introduction, as some females can still resist matings and reproduce parthenogenetically before coercion completely gains the upper hand (Figure 3a). However, once the *C* allele eventually fixes (thereby rendering

TABLE 1 Parameters used in simulations of the "discrete-trait" model

Parameter	Description	Parameter levels investigated
Number of patches	The number of patches that make up the world, which determines population density	High (2,601 patches; "low density") Low (121 patches; "high density")
Cost per mating for males	The number of time-steps deducted from male lifespan per mating	0, 5, 10, 15, 20
Cost per mating for females	The number of time-steps deducted from female lifespan per mating	0, 5, 10, 15, 20
Relative efficacy of coercion and resistance	The trait that has the upper hand in sexual encounters	"Coercion can evolve to always beat resistance" "Resistance can evolve to always beat coercion"
Cost of resistance	The number of time-steps deducted from the lifespan of females that successfully resist	0 ("resistance not costly") 10 ("resistance costly")
Timing of *P* allele introduction	The time-step at which mutants carrying the *P* allele are introduced into the population, which determines whether populations experience sexual coevolution prior to the mutants' introduction	0 ("no prior sexual coevolution") 10,000 ("prior sexual coevolution")
Cost of asexual reproduction	The proportion of fecundity lost by parthenogenetic females relative to sexual females	0 ("parthenogenetic fecundity is the same as sexual fecundity") 0.5 ("parthenogenetic fecundity is 50% lower than sexual fecundity") 0.95 ("parthenogenetic fecundity is 95% lower than sexual fecundity")

resistance impossible), mate scarcity then becomes the sole mechanism by which the *P* allele spreads. Together, these patterns suggest that mate scarcity can drive invasions of the *P* allele regardless of the effectiveness of coercion, but that sexually antagonistic coevolution can greatly accelerate and extend the parameter space of successful invasion if alleles for effective resistance are present in the population.

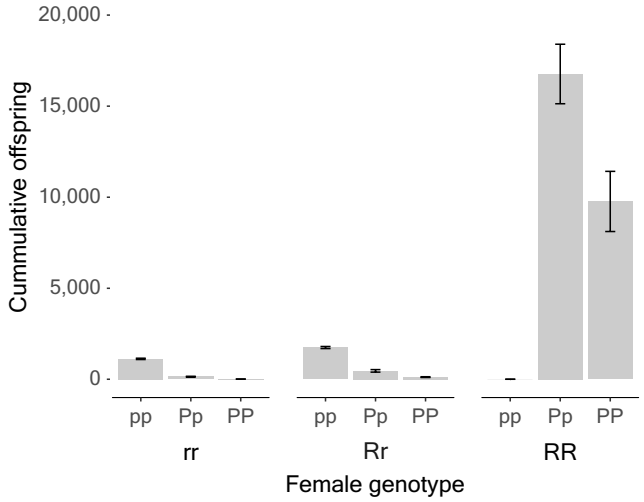

FIGURE 2 Mean cumulative sum ± *SE* of offspring produced by female genotypes. Data are obtained from the first 1,200 time-steps of simulations of "resistance" models where the *P* allele is introduced without prior sexual coevolution, allowing linkage disequilibrium to build up between *R* and *P*. 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

This also highlights the importance of standing genetic variation for invasion outcomes. The timing of the *P* allele's introduction in our simulations (either pre- or post-sexual coevolution) determines whether or not populations possess standing genetic variation in antagonistic traits and therefore how invasion proceeds. In resistance models *with* prior coevolution, linkage disequilibrium between the *P* and *R* alleles is unable to build up because females with high resistance fail to mate and reproduce, and selection rapidly eliminates the *R* allele from the population before facultative mutants finally arise (Figure S2B). However, in resistance models *without* prior sexual coevolution, genetic variation for resistance is available, and thus the *P* allele can rapidly associate with high resistance genotypes and invade rapidly over a large range of the parameter space, especially when resistance is cost-free (Figure 4b). By contrast, timing of introduction has less effect on the *P* allele's spread in the "coercion" model (Figure 4a) because high coercion rapidly evolves to beat resistance irrespective of prior sexual coevolution (Figure S2A,C).

The intensity of sexual conflict, reflecting costs of mating for females, also plays an important role in determining the success of *P* allele invasions. When there is no sexual conflict over mating rate (i.e., female cost per mating = 0), extinction of the *P* allele via drift is common (see Figure 4a). However, as the cost per mating increases, sexual conflict enhances the invasion probability of the *P* allele (see Figure 4a,b). In high-density populations where mating

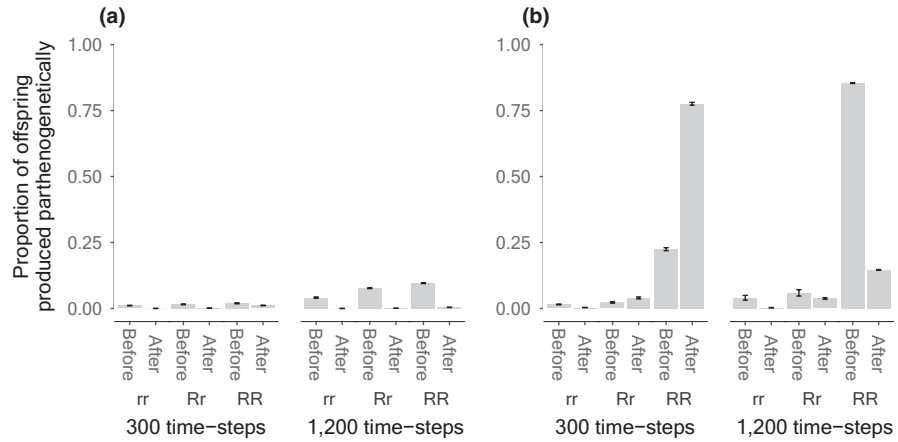

FIGURE 3 Mean proportion ± *SE* of offspring produced parthenogenetically by females of different resistance genotypes during the first 300 and 1,200 time-steps of simulation runs before and after initial sexual encounters, providing a fine-scale snapshot of how the frequency of parthenogenetic reproduction and the mechanism that promotes it change depending on which sex "wins" the sexual conflict. In the "coercion" model where sexual encounters always end in mating after the *C* allele spreads [a], most parthenogenetic reproduction occurs before females encounter males (i.e., due to mate scarcity) because only those females that by chance fail to encounter a male have the opportunity to reproduce asexually, and this proportion increases (and eventually stabilizes) as time progresses from 300 to 1200 time-steps. Conversely, in the "resistance" model [b], parthenogenetic reproduction initially occurs more frequently *after* mate encounters (i.e., via resistance) because as linkage disequilibrium builds up between the *R* and *P* alleles more and more females resist mating and are able to reproduce asexually instead. However, by 1,200 time-steps, as the *R* and *P* alleles approach fixation, parthenogenetic reproduction becomes far more frequent *before* mate encounters (i.e., due to mate scarcity) because even though most females at this stage have the ability to resist any mating attempt, the low production of sons means they almost never encounter a male. (In these simulations, *P* is introduced without prior sexual coevolution, with other parameters as in Figure 2)

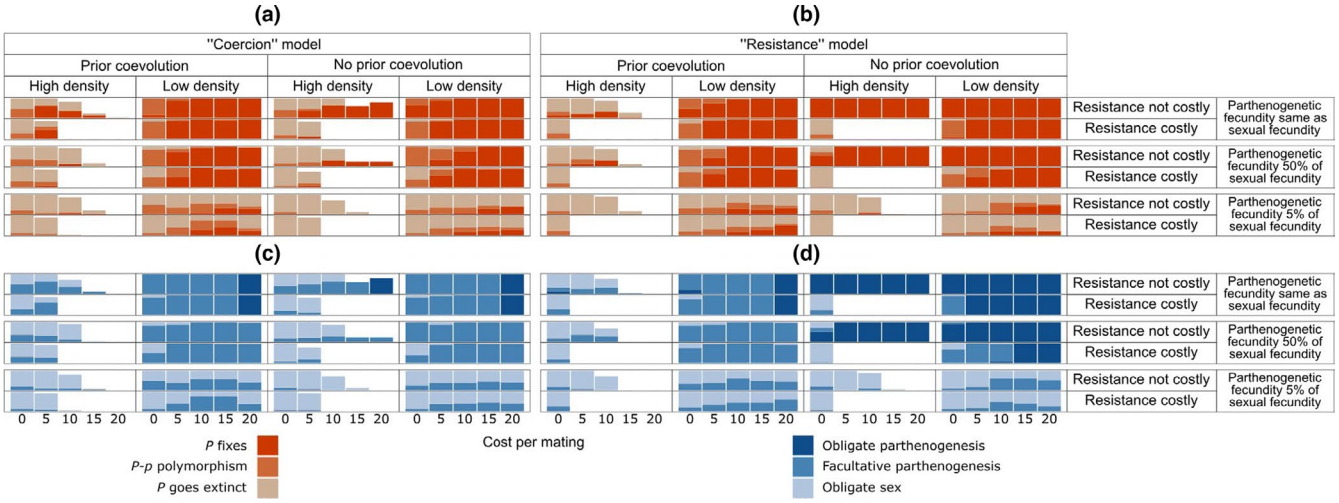

FIGURE 4 Evolutionary outcomes at the end of 20,000 time-steps following the introduction of the *P* allele for "discrete-trait" models where the cost per mating is equivalent for each sex. Increasing costs per mating for females reflects intensifying sexual conflict. The cost per mating for males is set at the same value as for females to maintain near-equal sex ratios. Left-hand graphs ([a] and [c]) are from the "coercion" model; right-hand graphs ([b] and [d]) are from the "resistance" model. Upper graphs ([a] and [b]) show the proportion of simulations that ended in different *P* allele outcomes; lower graphs ([c] and [d]) show the proportion of simulations that ended in different reproductive mode outcomes. White regions indicate parameter spaces where population extinction occurred before simulations ended. The likelihood of *P* allele fixation (dark orange) increases in panels [a] and [b] as costs per mating (and the consequent intensity of sexual conflict) increase along the *x*-axis. The probability of simulations ending in obligate parthenogenesis (dark blue in panels [c] and [d]) also increases as costs per mating increase. The *P* allele easily fixes in panel [a], especially when population density is low, because fecundity selection favours the production of parthenogens when mates are scarce, even though males in these simulations can coerce any female to mate. However, the success of the *P* allele in panel [a] does not translate to widespread obligate parthenogenesis in panel [c] because coercion ensures the continued production of males when females cannot resist effectively. Instead, obligate sex and facultative parthenogenesis are the most common outcomes in panel [c]. The *P* allele fixes more often in panel [b] than in panel [a] because resistance can beat coercion in these simulations. Transitions to obligate parthenogenesis are also more extensive in panel [d] than panel [c] because linkage disequilibrium can build up between the *P* allele and alleles for high resistance when resistance beats coercion and when there is no prior sexual coevolution. However, resistance costs greatly limit the success of parthenogenetic strategies in panels [b] and [d] when population density (and therefore sexual conflict) is high. Fixation of the *P* allele and transitions to facultative and obligate parthenogenesis are reduced across the board when asexual fecundity is extremely low. *N* = 25 simulations for each parameter combination

rates per female are very high, female lifespan and opportunities to reproduce decrease with increasing costs of mating, sending populations on a downward spiral to extinction (Figure 4a,b). Resistance costs exacerbate rates of extinction because high density elevates the frequency of resistance, which further reduces female lifespan. These conditions of declining population size are associated with increased rates of *P* allele fixation because the capacity to reproduce without mating is strongly favoured as densities plummet. Together, these results suggest that increased intensity of sexual conflict can promote facultative parthenogenesis and thereby avert population extinction if resistance costs are not too high.

Our results also show that low asexual fecundity can constrain the evolution of facultative parthenogenesis and inhibit transitions to obligate asexuality. In simulations where females are 95% less fecund when reproducing asexually than sexually, the *P* allele rarely spreads, and obligate sex is the predominant outcome. Even in the "resistance" model, where parthenogenesis is favoured under higher fecundity settings, the *P* allele mostly dies out when asexual fecundity is greatly reduced, and linkage disequilibrium fails to build-up between the *P* and *R* alleles. Similarly, low rates of invasion occur in low-density scenarios. However, whereas sex is never lost when parthenogenetic fecundity is very low, facultative parthenogenesis is still able to evolve in a number of simulations where density is low and costs of mating are high. Given that these last two parameter settings reduce encounter rates, our results suggest that facultative invasions may still proceed despite high costs of parthenogenesis if mate limitation is great enough.

3.2 | Conditions for the establishment of obligate parthenogenesis

We find that the introduction of *P* allele-carrying mutants into obligately sexual populations leads to one of three distinct evolutionary outcomes: (a) the *P* allele dies out, leaving populations to reproduce via obligate sex; (b) the *P* allele spreads either to an intermediate frequency or to fixation, with males able to persist in the population thereby allowing sex and parthenogenesis to coexist between and/or within individuals (i.e., facultative parthenogenesis); (c) the *P* allele spreads to fixation and parthenogenesis becomes obligate as a result of the complete extinction of males (Figure 2c,d). Facultative parthenogenesis and obligate sex are the most common evolutionary outcomes in the "coercion" model because highly coercive males ensure the continued production of male offspring by fertilizing eggs

of at least some facultatively parthenogenetic females (Figure 4c,d). By contrast, obligate and facultative parthenogenesis are the most common results in the "resistance" model. Rapid transitions to obligate parthenogenesis occur across a broad range of parameter space when the *R* and *P* alleles can become linked (i.e., in the "resistance" model without prior sexual coevolution), but only if costs of resistance are not too high (Figure 4d). However, in the "resistance" model with prior sexual coevolution, outcomes for reproductive mode closely resemble those for the "coercion" model (compare Figure 4c,d). This is because the high resistance allele *R* (and the potential for linkage disequilibrium) is lost whenever there is prior sexual coevolution (Figure S1A,B), and coercion consequently ensures the continued production of males.

3.3 | "Continuous-trait" model

In the "continuous-trait" model, where coercion and resistance can escalate in a coevolutionary arms race without limit, we find that *P* allele frequencies and reproductive mode outcomes are intermediate between those of "coercion" and "resistance" discrete-trait models. This occurs because increases in coercion are rapidly counteracted by increases in resistance and vice versa. Thus, linkage disequilibrium between the *P* allele and alleles for high resistance is less likely to result in male extinction because males quickly counter-evolve more effective coercion. Conversely, coercion is less effective at constraining transitions from facultative to obligate parthenogenesis because females quickly counter-evolve more effective resistance which readily becomes linked with the *P* allele. Nonetheless, as with "discrete-trait" models, *P* allele fixation becomes more likely as the intensity of sexual conflict increases. Detailed results for the "continuous-trait" model are reported in Supporting Information and Figure S3.

4 | DISCUSSION

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 role of each mechanism in the spread of facultative parthenogenesis and transitions to obligate parthenogenesis. We found that the *P* allele invaded successfully and displaced alleles for obligate sex across most of the realized parameter space due largely to the mechanism of mate scarcity. Even when all matings could be 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 initial 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. However, these effects were strongly mediated by asexual fecundity: high costs of asexual reproduction greatly reduced the probability of invasion even when females had the capacity to resist effectively.

The introduction of the *P* allele into obligately sexual populations led to one of three distinct evolutionary outcomes: obligate sex (*P* allele extinction), facultative parthenogenesis (*P* allele spread) or obligate parthenogenesis (extinction of males and consequent loss of sex). The distribution and frequency of each of these reproductive strategies was strongly determined by both the state of sexual antagonism at the time of the *P* allele's emergence and population density. Facultative parthenogenesis and obligate sex were the predominant outcomes when male coercion was able to permanently suppress female resistance and when asexual fecundity was severely penalized, whereas obligate parthenogenesis via male extinction was the most common outcome when standing genetic variation for effective resistance was present in the population at the introduction of the *P* allele. Moreover, male extinction was most frequent in low density when resistance was cost-free, whereas obligate sex was most frequent in high density when resisting was costlier than accepting matings.

These results are broadly consistent with previous findings, but extend them in important ways. First, our study confirms the effect of density from Gerber and Kokko's model (2016), but highlights different dynamics in the context of facultative invasions: high density in our simulations frequently resulted in obligate sex, but not because resistance costs made parthenogenesis less likely, as Gerber and Kokko (2016) found; rather, high density elevated female mortality and therefore increased the risk of the *P* allele being lost by chance. Second, our model recapitulates the effect of positive feedback on reproductive mode (see Burke & Bonduriansky, 2018b) by showing that selection for effective resistance following fixation of the *P* allele can generate epistasis for fitness (i.e., interacting phenotypic effects of *R* and *P* alleles on fitness) in females that do not mate, leading to the build-up of linkage disequilibrium between alleles for resistance and alleles for parthenogenesis. Importantly, however, our results go further to show that whereas this linkage disequilibrium enhances facultative invasions, it is not necessary for such outcomes, as the ability to produce offspring prior to encountering males generates sufficient positive selection on the *P* allele for fixation to occur. In addition, we show for the first time that the capacity for linkage disequilibrium to drive the loss of sex is dependent on the extent of genetic variation for effective resistance as determined by the genetic architecture of antagonistic traits, with chase-away sexual coevolution providing greater scope for the build-up of linkage disequilibrium than coevolution resolved in favour of coercion. This last finding suggests that factors that inhibit

optimal linkage disequilibrium could play a key role in determining evolutionary transitions to parthenogenesis in animal systems.

Indeed, our analysis suggests that the nature of resistance itself could be a particularly important limitation in this regard. In some species, males appear to "win" sexual arms races due to intense and persistent selection for effective coercion, whether by mechanically overpowering females to force matings (Rowe, Arnqvist, Sih, & Krupa, 1994), by chemical manipulation (Andersson, Borg-Karlson, & Wiklund, 2004; Chapman, Liddle, Kalb, Wolfner, & Partridge, 1995) or by precopulatory exploitation of sensory biases (Holland & Rice, 1998; Ryan & Rand, 1993). Genotypes that completely resist mating attempts may be rare or absent from many populations due to strong selection against mating failure (Kokko & Mappes, 2013), or due to selection favouring convenience polyandry when costs of resistance are high (Rowe, 1992). Moreover, many resistance behaviours are plastic, with virgin females often the least resistant to mating (Ringo, 1996); but see Hosken, Martin, Born, & Huber, 2003), whereas fixed strategies of high resistance are probably uncommon in natural populations. Thus, the absence of genetic variation for effective resistance may severely inhibit spontaneous transitions to asexuality by ensuring the continued production of sons, unless parthenogens can arise fully reproductively isolated, such as via inter-species hybridization (Avise, Quattro, & Vrijenhoek, 1992; Simon et al., 2003).

Increased mating costs in our simulations enhanced the spread of facultative parthenogenesis and promoted male extinction, whereas costs of resistance constrained the parameter space in which populations persisted. These costs are both involved in sexual conflict, but whereas mating costs drive inter-locus sexual conflict and select for female resistance to mating (Parker, 1979), resistance costs can limit the scope for the evolution of such female counter-adaptations (Arnqvist & Rowe, 2005). The different effects of these costs in our model highlight the importance of accounting for and distinguishing between them. Although we do not consider plastic resistance, future work could usefully investigate how the landscape of evolutionary outcomes might change if females have the capacity for facultative adjustment of resistance.

Theory suggests that facultative strategies should outcompete obligate sex (Green & Noakes, 1995; Yamauchi & Kamite, 2003). Indeed, our simulations show that facultative parthenogenesis can spread via mate scarcity under a broad range of ecological and genetic conditions. What, then, explains the rarity of facultative strategies in animal systems? Our analysis suggests that the nature of parthenogenetic mutants themselves could be the answer. First, natural populations probably give rise to facultatively parthenogenetic mutants at very low rates because the complex cytological changes required for parthenogenetic reproduction usually involve simultaneous mutations at multiple loci (Engelstadter, 2008; Neiman et al., 2014). Second, even when they do arise, facultative mutants may be far less fecund than wild-type females (Corley & Moore, 1999; Corley et al., 1999; Lamb & Willey, 1979), especially if the mechanism of parthenogenesis is meiotic (Levitis, Zimmerman, & Pringle, 2017). Third, in many facultatively asexual diploid animals, mated females appear to be unable to prevent their eggs from being fertilized. Indeed, cases of post-copulatory parthenogenesis are extremely rare (e.g., see Arbuthnott, Crespi, & Schwander, 2015; Chang, Ting, Chang, Fang, & Chang, 2014), which suggests that coerced fertilization of eggs by sperm may further limit parthenogenetic reproduction. Fourth, fitness costs associated with switching from a parthenogenetic to sexual mode of reproduction could be an additional hurdle (Burke & Bonduriansky, 2018a; Burke et al., 2015). Such constraints on parthenogenetic reproduction could therefore play key roles in preventing facultative mutants from invading obligately sexual populations. Our simulation results support this idea by showing that, when parthenogenetic fecundity is very low, facultative mutants typically fail to invade obligately sexual populations. However, our model did not allow for parthenogenetic capacity to evolve. Selection could enhance asexual fecundity if females show genetic variation for parthenogenetic ability (Carson, 1967) and regularly experience mate scarcity (Schwander, Vuilleumier, Dubman, & Crespi, 2010). Likewise, it is possible that enhanced parthenogenetic fecundity could evolve in combination with effective female resistance to mating.

Animal lineages that offer the best opportunities to test our model include those—such as stick insects, harvestmen and mayflies—that are broadly immune to developmental constraints and have evolved a diversity of asexual strategies numerous times. Such systems could be used, for example, to investigate the phylogenetic relationship between resistance and reproductive mode. Our model predicts that if linkage disequilibrium between alleles for resistance and alleles for parthenogenesis can drive males extinct, many obligate parthenogens of nonhybrid origin may show signs of past selection for high resistance. Stronger rejection behaviours in obligate parthenogens compared to sexual relatives, or sharper selection-driven decay in traits facilitating courtship, copulation and/or fertilization, would be suggestive of this link. Such patterns already appear to occur in a number of obligately asexual taxa (van der Kooi & Schwander, 2014; Schwander, Crespi, Gries, & Gries, 2013), but further investigations would be highly valuable.

Finally, if male coercion inhibits the evolution of obligate parthenogenesis, then taxa with greater potential for coercion may be less likely to exhibit obligately asexual forms. At a broad phylogenetic scale, the rarity of obligate asexuality in animals compared to both plants (Otto & Whitton, 2000) and microbial eukaryotes (Lahr et al., 2011; Speijer et al., 2015) may reflect the greater range of opportunities in animal systems for behavioural and chemical coercion (Andersson et al., 2004; den Hollander & Gwynne, 2009; Rowe et al., 1994; Wigby & Chapman, 2005). Conversely, mate scarcity could play a more important role than sexual conflict in plants, given their immobility, which may account for the high frequency of facultative asexuality via vegetative reproduction and selfing in many plant lineages (Holsinger, 2000). Comparative studies testing these predictions on a finer taxonomic scale may shed light on variation in reproductive strategies within eukaryotic lineages. Such studies will need to quantify actual rates of parthenogenetic reproduction, the incidence and costs of resistance, the costs of mating and the relative fecundity of sexual versus parthenogenetic reproduction

in natural populations of facultative organisms, all of which remain poorly known.

CONFLICT OF INTEREST

The authors have no conflict of interest in the production of this work.

DATA AVAILABILITY STATEMENT

Data will be available through the Dryad Digital Repository (datadryad.org). https://doi.org/10.5061/dryad.mh1m9j4.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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