

1 **Male coercion, female resistance and the** 2 **evolutionary trap of sexual reproduction**

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22 **ABSTRACT**

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

40

41 **INTRODUCTION**

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

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

51 It is widely assumed that sex is more common than parthenogenesis because it provides
52 benefits that outweigh the large costs (Maynard Smith 1978; Lehtonen et al. 2012). Genetic
53 recombination is thought to provide most of these benefits by promoting the elimination of
54 deleterious alleles (Muller 1964; Kondrashov 1993; Agrawal 2001; Siller 2001) and creating
55 beneficial and novel gene combinations (Fisher 1930; Muller 1932) that augment adaptive
56 responses to environmental or ecological change (Williams 1975; Maynard Smith 1978;
57 Hamilton 1980; Bell 1982; Ladle 1992; Agrawal 2006) and enhance “evolutionary potential”
58 of populations (Weismann 1889; Burt 2000; Roze 2012). However, because the benefits of
59 recombination generally apply to populations rather than individuals, hypotheses based on
60 indirect genetic benefits tend to treat sex as a group-selected trait (Kondrashov 1993; Green
61 and Noakes 1995). Moreover, the hypothesized benefits of sexual reproduction typically
62 apply within a limited subset of ecological parameter space (Bell 1982; Suomalainen et al.
63 1987; Barton 1995; West et al. 1999; Burt 2000; Rice 2002; Otto 2003, 2009; de Visser and
64 Elena 2007; Kouyos et al. 2007; Sloan and Panjeti 2010), whereas the widespread
65 taxonomic and ecological distribution of sexual reproduction in nature suggests that a very
66 general mechanism may be involved.

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

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

82 It has been suggested that costly obligate sex could be maintained by physiological
83 constraints. For example, limited diploidy restoration (Suomalainen et al. 1987), failed
84 activation of unfertilised eggs (Stricker 1999), and genomic imprinting (Reik and Walter
85 2001), may impede the evolution of parthenogenesis in many sexual animals (Engelstadter
86 2008). Such constraints can limit asexual invasions by disrupting embryonic development,
87 suppressing asexual egg production, or reducing offspring viability (Enghoff 1976; Lamb and
88 Willey 1979; Lynch 1984). Likewise, physiological dependence on sexual stimulation could
89 constrain the spread of spontaneously-derived parthenogenesis (Neiman 2004). However, if
90 asexual reproduction is advantageous, long-term evolution may be expected to circumvent
91 such constraints in many lineages. The widespread occurrence of obligate sexuality
92 therefore suggests the existence of taxonomically widespread mechanisms or dynamics that
93 not only prevent asexual reproduction from occurring in existing sexual populations, but also
94 act as persistent impediments to evolutionary transitions to asexuality in diverse sexual
95 lineages, turning sex into an evolutionary trap.

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

102 that coerce females into mating and reduce female fecundity (Maklakov et al. 2005) or
103 longevity (Martin and Hosken 2003). Given the potentially large costs that females can suffer
104 due to male harassment, sexual coercion (and sexual conflict more generally) has been
105 proposed as an additional cost of sex (Rankin et al. 2007). However, it has also been
106 suggested that sexual coercion could promote the persistence of sex (Dagg 2006; Kawatsu
107 2013a, 2013b, 2015; Burke et al. 2015; Gerber and Kokko 2016). In a sexual population, a
108 mutant allele that makes parthenogenetic reproduction possible may be expected to flourish
109 and spread due to the demographic advantage of producing all-female offspring (Maynard
110 Smith 1978), as well as the ecological and physiological advantages of reproduction without
111 mating. However, if asexual mutants suffer greater costs than sexual females as a
112 consequence of encountering coercive males, parthenogenesis may fail to spread (Kawatsu
113 2013b). Such differential costs could manifest as coercion-induced declines in female
114 fecundity or survival (Burke et al. 2015). Moreover, coercion could inhibit the spread of
115 parthenogenesis by forcing females to reproduce sexually because, in many facultatively
116 asexual animals, only virgin females are able to reproduce parthenogenetically (Bell 1982;
117 Normark and Kirkendall 2009).

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

129 Another theoretical analysis suggests that female resistance to mating may be more
130 effective in promoting parthenogenesis when mate encounter rate is low (Gerber and Kokko
131 2016). However, this analysis did not fully disentangle the relative roles of resistance versus
132 mate scarcity in promoting evolutionary transitions to asexuality. Disentangling these
133 mechanisms requires assessment of the timing of parthenogenetic reproduction in relation to
134 mating attempts: mate scarcity can select for parthenogenesis whenever facultatively
135 parthenogenetic virgins reproduce asexually prior to encountering a mate; whereas
136 resistance can select for parthenogenesis whenever virgin females reproduce asexually after
137 resisting sexual encounters. The contribution of each of these mechanisms to the invasive
138 potential of facultative asexuality therefore remains unclear (although see Burke et al. 2015).

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

148

149 **METHODS**

150 ***Overview of IBMs***

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

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

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

168 The sexes in our models experience sexual conflict over mating rate such that multiple
169 matings always benefit males but can be costly to females. Although species vary in the
170 number of matings females can obtain before additional matings become costly (Parker and
171 Birkhead 2013), for simplicity, our models follow the formulation of Arnqvist and Rowe
172 (Arnqvist and Rowe 2005) and assume that any more than one mating is costly for females.

173 The sexes in our models experience antagonistic sexual coevolution, a predicted
174 consequence of sexual conflict (7). We model coercion and resistance as discrete traits,
175 each controlled by a diploid autosomal locus with two alleles, c and C , and r and R ,
176 respectively, with additive, sex-limited effects. We model resistance and coercion this way
177 because we wanted to investigate cases where sexually antagonistic selection on standing
178 genetic variation could reach a stable end point, at which either male coercion or female
179 resistance gains the upper hand. In a separate set of models that simulate an escalating
180 arms race, we allow coercion and resistance to coevolve without constraint by treating them
181 as continuously distributed values (see Online Appendix).

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

189 The mutant P allele is introduced either at time-step 0 (i.e., at initialization), or at time-step
190 10,001 (i.e. after simulations have run for 10,000 time-steps, allowing for sexually
191 antagonistic selection to alter allele frequencies and for genetic associations to build-up in
192 the population). We hereafter refer to models setup this way as ‘non-equilibrated’ and
193 ‘equilibrated’, respectively.

194 ***Initialization of simulations***

195 At the start of each simulation and before the mutant P allele is introduced, 500 females and
196 500 males are randomly distributed throughout the environment, each facing a randomized
197 direction. Each individual is allocated a maximum lifespan of 100 time-steps, and an age of 0
198 time-steps that increases by 1 every time-step. Males and females are both allocated
199 coercion and resistance genotypes according to Hardy-Weinberg probabilities (i.e.,
200 individuals have a probability of 0.5 of obtaining heterozygous genotypes and a probability of
201 0.25 of obtaining either of the alternative homozygous genotypes). The carrying capacity of
202 the environment (i.e., maximum population size) is fixed at 2,500.

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

207 **Life cycle**

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

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

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

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

233 a mating attempt (i.e., if mating is attempted but does not take place). Successful matings
234 result in the transfer of paternal alleles via male gametes (sperm) which females store
235 internally. Following other models of sexual conflict (e.g., 72), we assume that females store
236 enough sperm from one mating to reproduce sexually their whole life. Last male precedence,
237 which is widespread in a diversity of animal taxa (Smith 1984), is assumed in all models.
238 Males and females incur sex-specific survival costs of mating, applied as penalties of 0, 5,
239 10, 15 or 20 time-steps deducted from an individual's remaining lifespan. This generates
240 sexual conflict over mating rate: when the female mating cost is > 0 , a single mating per
241 lifetime is optimal for females, whereas male fitness increases with each additional mating
242 regardless of the cost.

243 The mating phase is followed by the reproducing phase. Reproduction is a lottery that occurs
244 every time-step as long as the current population size is less than the fixed carrying
245 capacity. Each female capable of reproducing (i.e., any previously mated female, and any
246 virgin female carrying at least one copy of the *P* allele) is allotted a random number between
247 0 and 1 every time-step. Previously mated females with a random number < 0.1 reproduce
248 sexually. For virgin females that carry the *P* allele, reproduction probability per time-step is
249 set at either 0.05 or 0.1, representing either 50%-reduced parthenogenetic fecundity (i.e., a
250 genetic/physiological constraint on asexuality; 49,71) or equal fecundity of parthenogenetic
251 reproduction relative to sexual reproduction. A female can win the reproductive lottery
252 multiple times during her life, and produces one offspring each time. Females that reproduce
253 parthenogenetically produce only daughters, while females that reproduce sexually produce
254 daughters and sons with equal probability. As occurs in many facultatively parthenogenetic
255 taxa (Bell 1982), females that mate reproduce sexually thereafter, even if they are capable of
256 parthenogenesis (i.e., even if they carry the *P* allele). There are no costs of gamete or
257 offspring production.

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

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

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

$$266 \quad S = 1 - \left(\frac{a + cm}{L} \right)$$

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

270 **Analysis**

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

279

280

281 **RESULTS**

282 ***Sexual conflict as a function of differential costs of mating***

283 Relative male and female costs of mating have consistent and important demographic
284 effects prior to the introduction of the *P* allele. When the cost per mating is set higher for
285 females than for males, male-biased sex ratios are generated because of reduced female
286 longevity; whereas when the cost per mating is set higher for males than for females,
287 female-biased sex ratios are generated because of reduced male longevity. This pattern can
288 be seen in the first 10,000 time-steps of Figures 1 *A* and *B* and: male sex ratios are less than
289 0.5 when male costs are higher than female costs, but are greater than 0.5 when female
290 costs are greater than male costs. A male-biased sex ratio increases sexual encounter rates
291 and mean female mating rates, elevating female mortality. This demographic pattern is
292 observable in the mean lifespan penalties incurred by mated females during the first 10,000
293 time-steps of Figures 1 *C* and *D*. The consequences of these dynamics are discussed
294 below.

295 ***Conditions for the invasion of the *P* allele***

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

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

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

318

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

331

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

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

344 ***Conditions for the establishment of obligate parthenogenesis***

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

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

372 High resistance in non-equilibrated resistance models releases females from the constraints
373 of high coercion and facilitates rapid transitions to obligate parthenogenesis across a very
374 broad parameter space, but only if costs incurred due to resistance are not too high (Figure
375 2 D). By contrast, the number of simulations ending in obligate parthenogenesis in
376 equilibrated resistance models closely resembles the outcome for equilibrated coercion
377 models (compare Figures 2 C and D). This similarity is due to sexually antagonistic selection
378 favouring the high coercion *C* allele and suppressing the high resistance *R* allele in both
379 equilibrated models (Figures 5 A and B). Selection always promotes the *C* allele due to the
380 universal benefit to males of mating multiply (Figure 5); whereas the *R* allele is strongly
381 selected against in the absence of the *P* allele because *RR* females successfully resist all
382 mating attempts and produce no progeny when parthenogenesis is not possible. This effect
383 is particularly evident in equilibrated resistance models where *R* is highly disadvantageous in
384 the absence of *P*, and the least resistive allele *r* becomes fixed before mutants carrying the
385 *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

413 **DISCUSSION**

414 Previous studies have suggested that male coercion can promote the maintenance of sexual
415 reproduction in facultatively asexual systems (Kawatsu 2013a, 2013b, 2015; Burke et al.
416 2015; Gerber and Kokko 2016). Results from these studies suggest that sexual conflict could
417 play a key role in the maintenance of sexual reproduction, and thus contribute to a resolution
418 of the 'paradox of sex'. However, sexual conflict gives rise to sexually antagonistic
419 coevolution—a process whose dynamics are likely to reflect both ecological parameters
420 (such as costs of mating, sexual encounter rates, and resistance) and the genetic
421 architecture of sexually antagonistic traits. Understanding the role of sexual conflict in the
422 maintenance of sex requires elucidating how these factors and resulting coevolutionary
423 dynamics promote/inhibit transitions from sexual to asexual reproduction. Moreover,
424 previous studies have not clearly differentiated the role of sexual conflict from the role of
425 mate scarcity in the evolution of reproductive mode. We used individual-based models with
426 varying ecological and genetic parameters to investigate the conditions whereby an asexual
427 (parthenogenetic) strategy can invade sexual populations undergoing sexually antagonistic
428 coevolution.

429 Our results indicate two mechanisms by which sexually coevolving populations can be
430 invaded by facultative parthenogenesis: mate scarcity and female resistance. It has long
431 been suggested that mate scarcity favours the spread of facultative parthenogenesis in small
432 or female-biased populations where mating is either difficult or impossible (Stalker 1956;
433 Kramer and Templeton 2001; Schwander et al. 2010), and might therefore account for the
434 evolution of some facultative systems, including some Phasmatids (Schwander and Crespi
435 2009), Ephemeropterids (Brittain 1982), and Dipterans (Markow 2013). The mate scarcity
436 mechanism applies to any situation where virgin females have an opportunity to reproduce
437 prior to initial sexual encounters. Our analysis allowed us to distinguish between instances of
438 parthenogenetic reproduction facilitated by mate scarcity (i.e., parthenogenesis before initial
439 mating attempts) and by resistance (i.e., parthenogenesis after initial mating attempts), and

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

454 The introduction of the *P* allele into obligately sexual populations led to one of three distinct
455 evolutionary outcomes: obligate sex, facultative parthenogenesis, or obligate
456 parthenogenesis (loss of sex). The distribution and frequency of each of these reproductive
457 modes was strongly determined by the genetic architecture of sexual antagonism at the time
458 of the *P* allele's emergence. When males successfully evolved the capacity to coerce any
459 female to mate, facultative parthenogenesis was the predominant outcome, and male
460 extinction rarely occurred. By contrast, when females evolved effective resistance, male
461 extinction (and therefore obligate parthenogenesis) was the most common result.

462 Importantly, this finding occurred both when coercion and resistance were modelled as traits
463 determined by single loci (and therefore when evolution occurred via selection on standing
464 genetic variation), and when coercion and resistance were modelled as multi-locus traits
465 representing a large mutational target (and therefore when sexual arms races could escalate
466 without limit; see Online Appendix) This suggests that when effective resistance cannot

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

471 Our finding that resistance can be a key factor in transitions to facultative and obligate
472 parthenogenesis is consistent with results of previous studies (Kawatsu 2013a; Burke et al.
473 2015; Gerber and Kokko 2016). However, our analysis provides a number of additional
474 insights. In particular, we show that the potential for invasion of facultatively parthenogenetic
475 mutants is strongly dependent on the relative costs of mating for males and females and the
476 dynamics of sexual coevolution. Consistent with previous findings (Kawatsu 2013a), we
477 found that the evolution of obligate parthenogenesis depended on whether alleles for
478 effective female resistance were present in the population at the time of the *P* allele's
479 introduction, and therefore whether positive epistatic associations between resistance and
480 parthenogenesis could build up. In addition, our analysis showed that the evolution of
481 obligate parthenogenesis was contingent on the weight of resistance costs: populations
482 evolved to be obligately asexual only to the extent that females were able to bear the cost of
483 resisting mating attempts. This result supports previous findings that facultatively
484 parthenogenetic females should resist only at low population densities where encounter
485 rates are low (Gerber and Kokko 2016). Our analysis also extends previous work by
486 clarifying the roles of both mate scarcity and sexual conflict, and the consequences of
487 fecundity costs of parthenogenetic reproduction, in evolutionary transitions to facultative and
488 obligate parthenogenesis.

489 The limitations of resistance highlighted by our analysis have important implications for
490 understanding the incidence of sex and parthenogenesis in nature. In many species, males
491 tend to “win” sexual arms races due to intense and persistent selection for effective coercion,
492 whether by mechanically overpowering females to force matings (Rowe et al. 1994), by
493 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 (Avisé 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; Gavrillets 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 2013a; 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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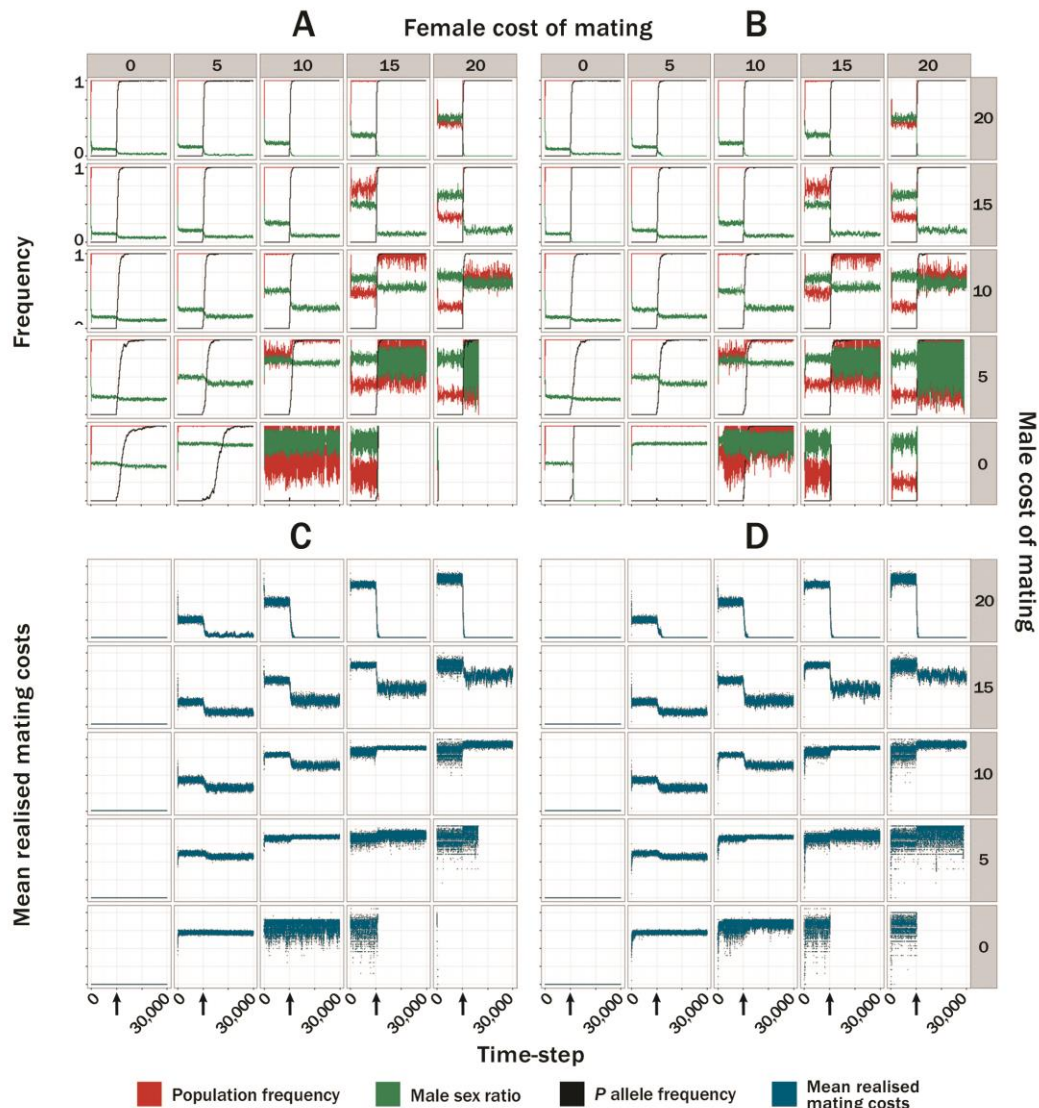
Table 1
Parameters used in simulations

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 <i>P</i> allele introduction	The time-step at which mutants carrying the <i>P</i> 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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643 **Figure 1**
 644 **Time-dependent changes in population statistics (A and B) and mean realised costs**
 645 **of mating (C and D) for a single simulation per mating-cost combination of**
 646 **equilibrated models.**

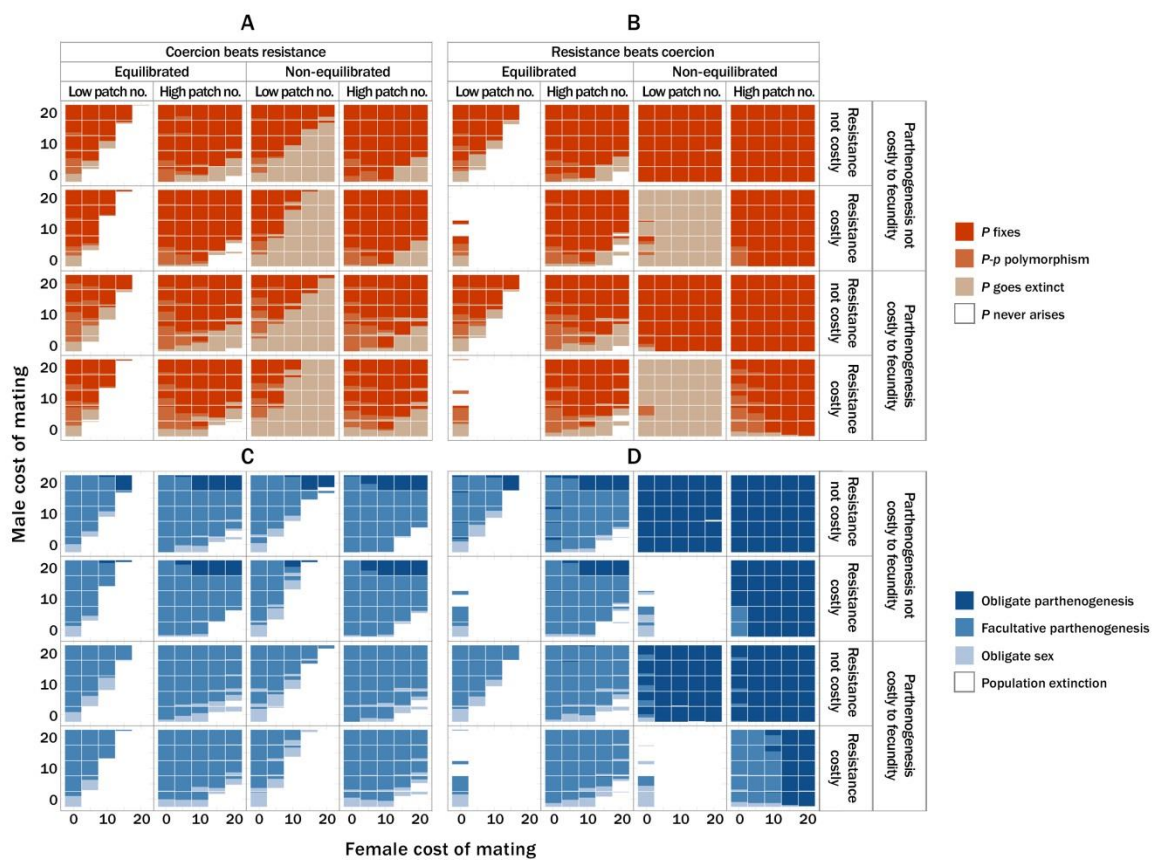
647 Left-hand graphs (A and C) are from coercion models; right-hand graphs (B and D) are from
 648 resistance models. In upper graphs (A and B), population frequency (in red) is the population
 649 size divided by the carrying capacity, with extinction indicated by a frequency of 0. Male sex
 650 ratio (in green) is the number of males divided by the population size, with sex ratios above
 651 0.5 indicating male bias. *P* allele frequency (in black) is the number of individuals carrying
 652 the *P* allele divided by the population size. In lower graphs (C and D), realised cost of mating
 653 is the mean lifespan penalty due to mating accumulated by females over their lifetime.
 654 Arrows indicate the time-step at which the *P* allele is introduced. Other parameter settings
 655 are: number of patches: high; cost of resistance: 0; *P* allele timing: 10,000 (i.e., with
 656 equilibration); cost of parthenogenesis: 0.



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658 **Figure 2**
 659 **Evolutionary outcomes following the introduction of the *P* allele.**

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



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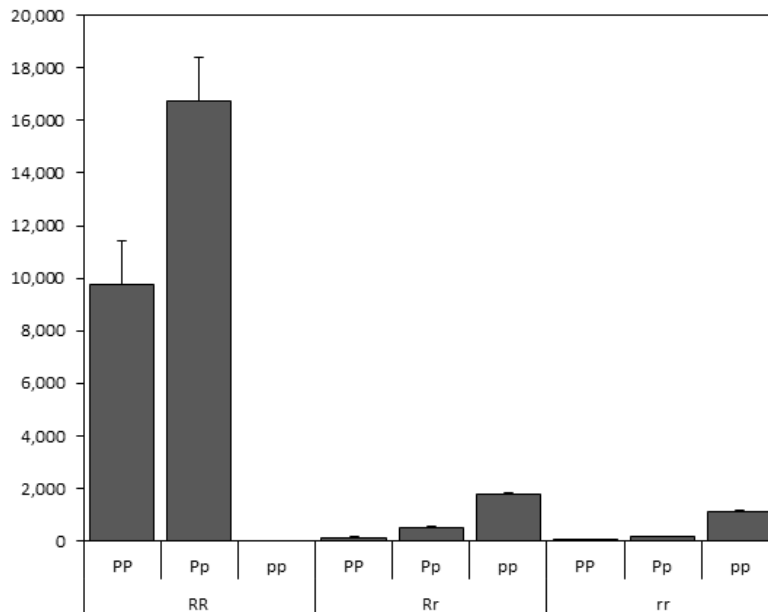
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677 **Figure 3**

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

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



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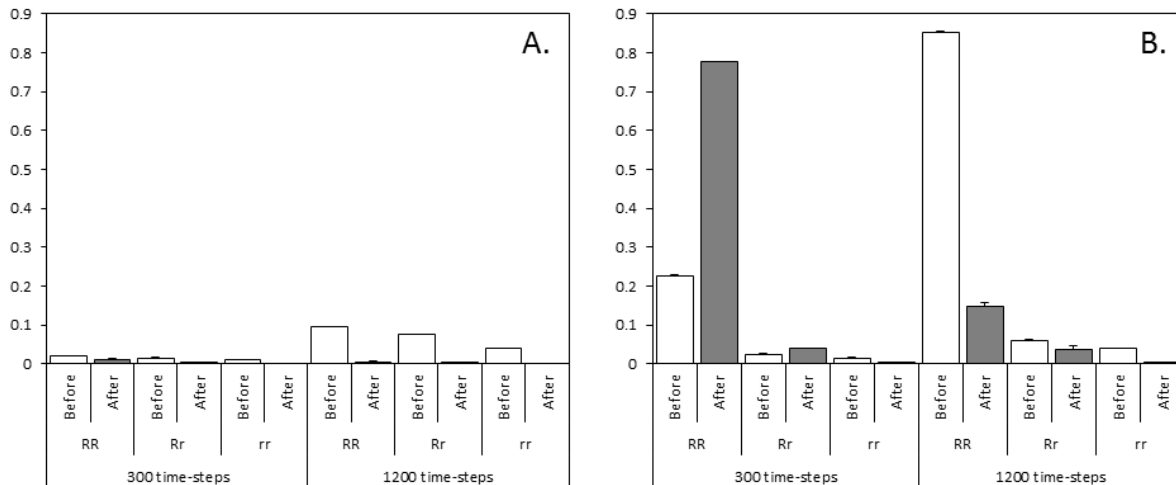
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698 **Figure 4**
699 **Mean proportion (+ SE) of offspring produced parthenogenetically by females of**
700 **different resistance genotypes during the first 300 and 1,200 time-steps of simulation**
701 **runs before and after initial sexual encounters.**

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



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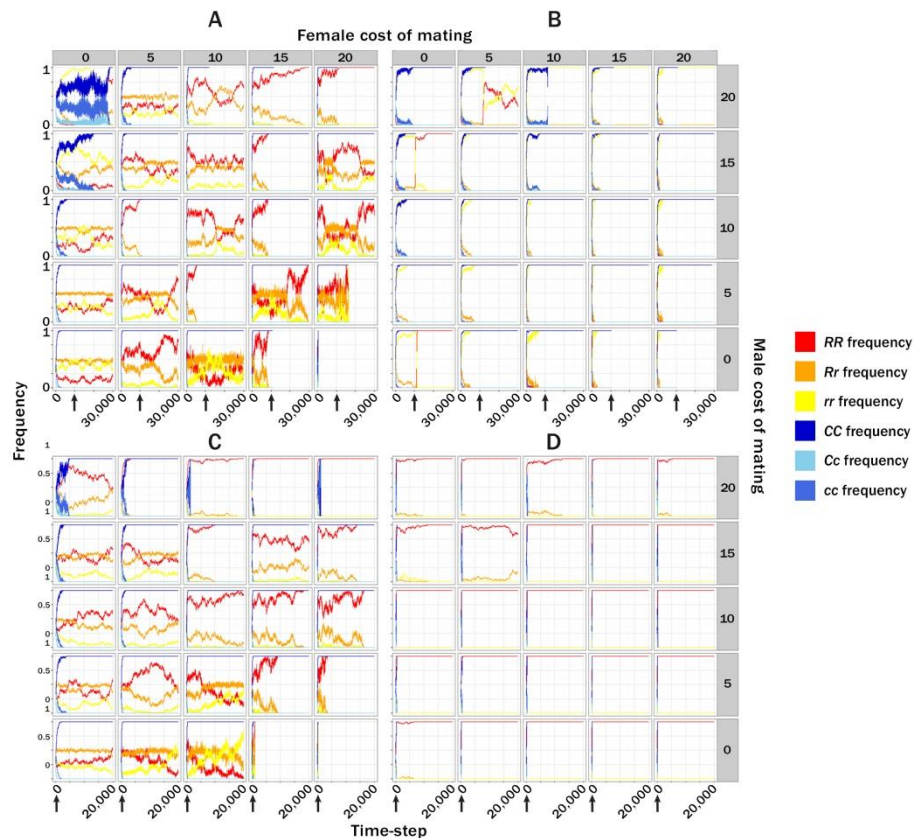
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724 **Figure 5**
725 **Time-dependent changes in coercion and resistance genotypes for a single**
726 **simulation per mating-cost combination.**

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



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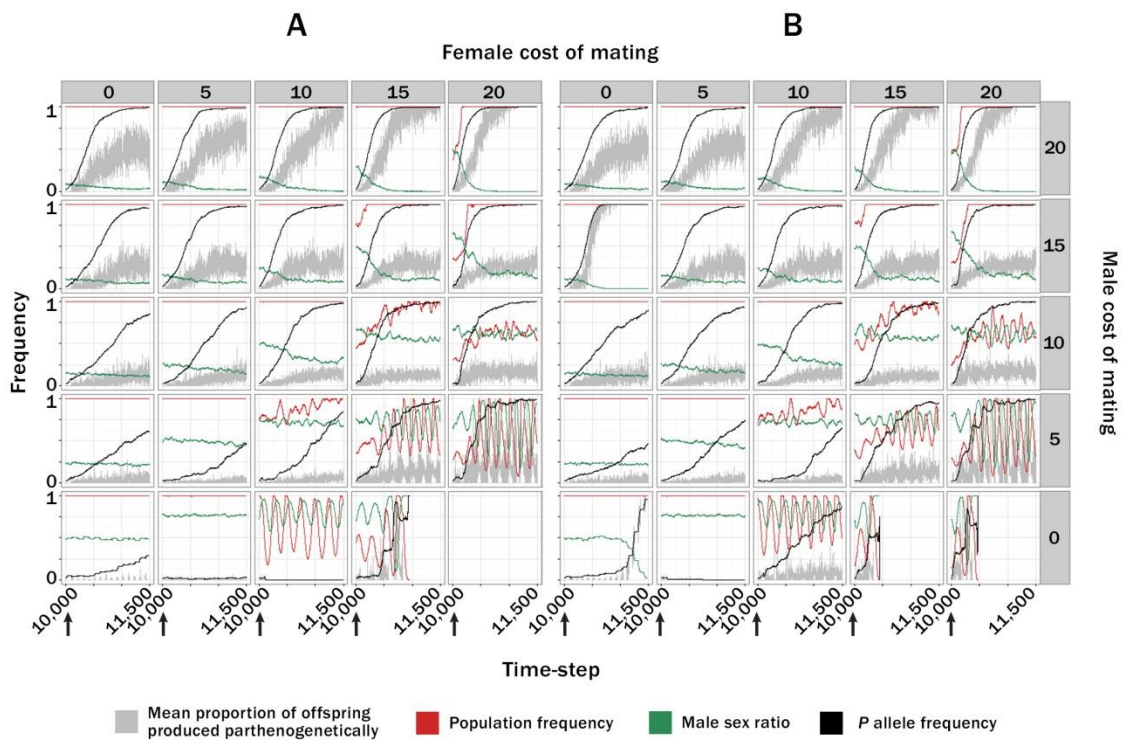
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743 **Figure 6**
744 **Snapshot of time-dependent changes in population statistics after equilibration for a**
745 **single simulation per mating-cost combination.**

746 Each graph is a snapshot of Figures 2 A and B, respectively, between time-steps 10,000 to
747 11,500. Graph A shows results from coercion models; graph B from resistance models.
748 Population frequency (in red), male sex ratio (in green), and *P* allele frequency (in black)
749 are calculated the same as in Figure 2. The mean proportion of parthenogenetic offspring (in
750 grey) is calculated as the total number of offspring produced parthenogenetically divided by
751 the total number of offspring produced sexually and parthenogenetically. Arrows indicate the
752 time-step at which the *P* allele is introduced. Other parameter settings are the same as in
753 Figure 1. Each graph shows distinct cycling dynamics in population size, sex ratio and
754 realised parthenogenetic reproduction when female mating costs are greater than male
755 mating costs.



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765

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970 **APPENDIX**

971 **Continuous-trait model**

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

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

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

996 and A1 A). The proportion of simulations ending in obligate parthenogenesis was also
997 intermediate (compare and contrast Figures 2 C, 2 D and A1 B). These intermediate results
998 emerged because of the dynamics of chase-away sexual coevolution: every coevolutionary
999 step by one sex further escalated the arms race and selected for counter-adaptation in the
1000 other sex, such that coercion was never able to completely subdue resistance, or vice versa.
1001 As in discrete-trait models, the timing of the *P* allele's introduction in the continuous-trait
1002 model was an important determiner of evolutionary outcomes, with frequent transitions to
1003 obligate parthenogenesis occurring in non-equilibrated versions of the model when high
1004 resistance genotypes were able to associate with the *P* allele, and especially when
1005 parthenogenesis bore no cost (Figure A1 B). However, the negative effect of costly
1006 resistance on extinction rates was not as severe in the continuous-trait model as in the
1007 resistance model (contrast Figures 2 D and A1 B). This is because escalating sexual
1008 coevolution produced more diverse resistance and coercion genotypes than did sexual
1009 coevolution resolved in favour of females. This greater variation in female ability to resist
1010 also explains why *P* allele invasions and transitions to obligate parthenogenesis were less
1011 frequent in the continuous-trait model compared to the resistance model (contrast Figures 2
1012 B and D with Figures A1 A and B, respectively): escalating arms races prevented fixation of
1013 permanently high resistance genotypes by facilitating the continuous production of ever-
1014 more coercive males. With females unable to decisively gain the upper hand, the ability of
1015 linkage disequilibrium between resistance and the *P* allele to drive males extinct was
1016 reduced.

1017 In equilibrated versions of the continuous-trait model, sexual conflict always decreased after
1018 the introduction of the *P* allele, unless the allele died out (see the drop in mean realised
1019 costs of mating for females after the introduction of *P* in Figure A2 B). At any given time-
1020 step, chase-away antagonistic coevolution generated at least some females with high
1021 resistance that could resist some matings, and the *P* allele allowed some of these high-
1022 resistance females to reproduce parthenogenetically. Hence, invasions of facultative

1023 parthenogenesis in the continuous-trait model universally reduced sexual conflict, regardless
1024 of the relative cost of mating for females, because effective resistance could always evolve
1025 to reduce mating rates.

1026 These results suggest that the genetic architecture of sexual coevolution has qualitatively
1027 similar effects on the invasion success of facultatively parthenogenetic mutants regardless of
1028 whether sexual coevolution is resolved in favour of one of the sexes or is unresolved in a
1029 continuously escalating arms race.

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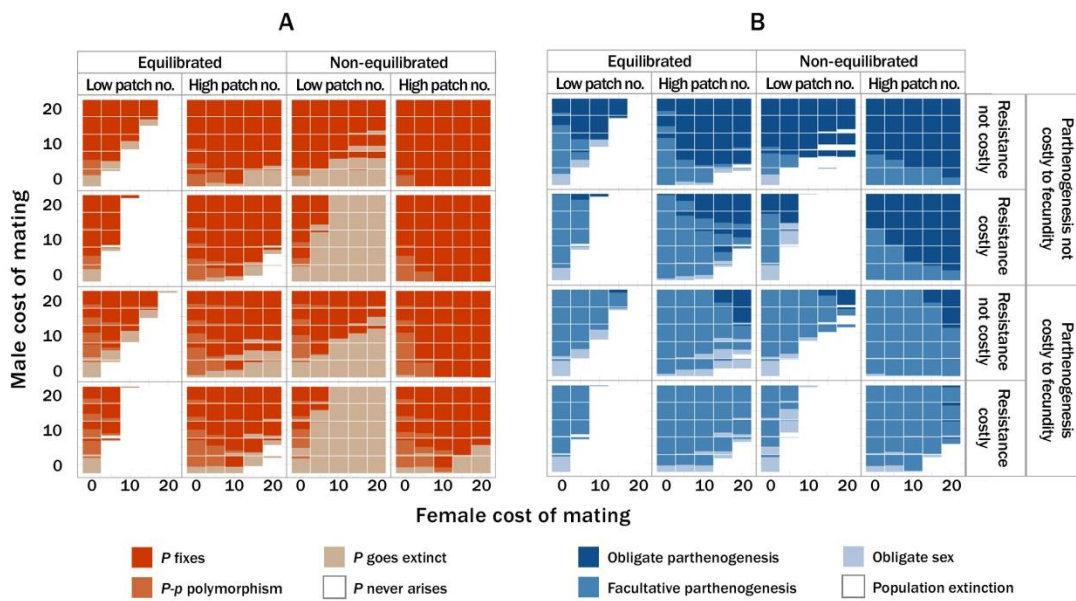
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1044 **Figure A1**
 1045 **Evolutionary outcomes of the continuous-trait model following the introduction of the**
 1046 ***P* allele.**

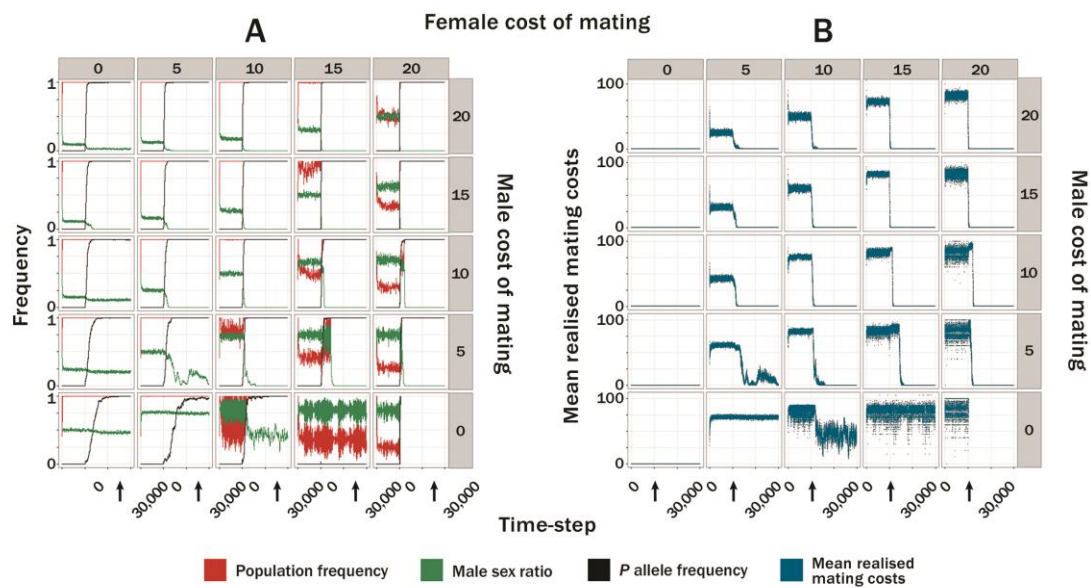
1047 Graph A shows *P* allele outcomes; graph B shows reproductive mode outcomes. The small
 1048 coloured squares in graph A show the proportion of 25 simulation runs per parameter
 1049 combination ending in *P* fixation (dark orange), *P-p* polymorphism (light orange), *P* extinction
 1050 (beige), and *P* never arising (white). Note that the *P* allele is always set to arise in non-
 1051 equilibrated simulations (see *Initialization of simulations* in Methods for details). Coloured
 1052 squares in graph B show the proportion of 25 simulation runs per parameter combination
 1053 ending in obligate parthenogenesis (dark blue), facultative parthenogenesis (medium blue),
 1054 obligate sex (light blue), and population extinction (white).



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1065 **Figure A2**
1066 **Time-dependent changes in population statistics (A) and mean realised costs of**
1067 **mating (B) for a single simulation per mating-cost combination of the equilibrated**
1068 **version of the continuous-trait model.**

1069 Graph A shows population frequency (in red), male sex ratio (in green), and *P* allele
1070 frequency (in black). Graph B shows the mean lifespan penalty due to mating accumulated
1071 by females over their lifetime. Arrows indicate the time-step at which the *P* allele is
1072 introduced. Other parameter settings are: number of patches: high; cost of resistance: 0; *P*
1073 allele timing: 10,000 (i.e., with equilibration); cost of parthenogenesis: 0.



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