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4 **Sexual selection, phenotypic plasticity, and female reproductive output**
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19 **Summary**
20

21 In a rapidly changing environment, does sexual selection on males elevate a population's
22 reproductive output? If so, does phenotypic plasticity enhance or diminish any such effect?
23 We outline two routes by which sexual selection can influence the reproductive output of a
24 population: a genetic correlation between male sexual competitiveness and female lifetime
25 reproductive success; and direct effects of males on females' breeding success. We then
26 discuss how phenotypic plasticity of sexually selected male traits and/or female responses
27 (e.g. plasticity in mate choice), as the environment changes, might influence how sexual
28 selection affects a population's reproductive output. Two key points emerge. First, condition-
29 dependent expression of male sexual traits makes it likely that sexual selection increases
30 female fitness *if* reproductively successful males disproportionately transfer genes that are
31 under natural selection in both sexes, such as genes for foraging efficiency. Condition-
32 dependence is a form of phenotypic plasticity if some of the variation in net resource
33 acquisition and assimilation is attributable to the environment rather than solely genetic in
34 origin. Second, the optimal allocation of resources into different condition-dependent traits
35 depends on their marginal fitness gains. As male condition improves, this can therefore
36 increase or, though rarely highlighted, actually decrease the expression of sexually selected
37 traits. It is therefore crucial to understand how condition determines male allocation of
38 resources to different sexually selected traits that vary in their immediate effects on female
39 reproductive output (e.g. ornaments versus coercive behaviour). In addition, changes in the
40 distribution of condition among males as the environment shifts could reduce phenotypic
41 variance in certain male traits, thereby reducing the strength of sexual selection imposed by
42 females. Studies of adaptive evolution under rapid environmental change should consider the
43 possibility that phenotypic plasticity of sexually selected male traits, even if it elevates male
44 fitness, could have a negative effect on female reproductive output, thereby increasing the
45 risk of population extinction.
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1. Introduction

Sexual selection favours traits that are often exclusively expressed or only exaggerated in males, such as weapons and ornaments, which increase mating or fertilization success when there is competition for mates or fertilization opportunities. In contrast, natural selection favours economically efficient traits that are usually similarly expressed in both sexes, which improve foraging ability, predator evasion, disease resistance and so on. In general, therefore, natural and sexual selection are in opposition. There is a trade-off between a longer life or a faster mating rate (but see [1,2]). It might therefore seem slightly paradoxical that researchers have asked whether sexual selection on males can increase the rate at which females adapt to a novel environment [3-7]. This is akin to asking if sexual selection on *males* elevates the mean absolute lifetime reproductive output of *females* (i.e. population mean fitness [definition modified from 8; see also 9]), thereby increasing the maximum population growth rate and decreasing the likelihood of population extinction. In a similar vein, researchers studying phenotypic plasticity, especially those motivated by conservation concerns arising from climate change, industrial-scale agriculture and urbanization, have asked whether plastic responses to rapid environmental change reduce the likelihood of population extinction ('plastic rescue' *sensu* [8]) because phenotypic plasticity increases population mean fitness [10].

Surprisingly, these two research questions are rarely combined. Researchers studying plastic rescue mostly ask whether plastic responses of naturally selected traits to a changing environment are broadly adaptive (i.e. elevate male and female absolute fitness). It is rare for them to instead ask whether adaptive plasticity of sexually selected traits in *males* (i.e. those that increase relative mating or fertilization success) will increase the mean absolute lifetime

75   reproductive output of *females*. Before proceeding further, we should acknowledge that mean

76   *female* lifetime reproductive success (LRS) is an imperfect proxy for the realised growth of a

77   population and its effective population size (the two key demographic parameters that

78   influence extinction risk [review: 11; see also 12]). We are essentially assuming there is

79   'hard' rather than 'soft' selection on female LRS [see 13] such that *absolute* differences in

80   female LRS between a population with and without sexual selection translate into differential

81   recruitment rates. This is a simplification, but one that is widely used when investigating so-

82   called 'population fitness' [e.g. 8].

83

84   Many factors select for different levels of expression of sexually selected traits by males

85   [review: 14]. For example, the sonic and light environment affect the transmission of acoustic

86   and visual courtship signals respectively [review: 15]; and the local predator and parasite

87   community determine the costs of investing in attractive traits that increase a male's

88   vulnerability to predators, or capacity to tolerate parasites. The benefits of investing in

89   sexually rather than naturally selected traits also depend on the strength of sexual selection,

90   which can covary with the operational sex ratio, density of competitors and mate encounter

91   rate [16-18]. Perhaps the most important and widespread form of phenotypic plasticity in

92   sexually selected traits relates to the availability of resources. Many sexually selected traits

93   show 'condition-dependent' expression, being smaller when food is limited. All of these

94   factors should select for males that detect appropriate environmental cues and show an

95   adaptive plastic response in their investment into sexually selected traits.

96

97   In this review, we explore how plastic responses by males to a changing/novel environment

98   could influence the mean absolute lifetime reproductive success (LRS) of females, hence the

99   likelihood of population extinction. We focus on plasticity in males rather than females

100   because theory suggests that sexual selection mainly acts on males [19-21]. This claim is

101   widely supported empirically by greater male weaponry and ornamentation [22-25], and by a

102   stronger relationship in males than females between mating and reproductive success [26; but

103   see 27-28] (for examples of sexual selection in females see [29, 30]).

104

105   (Terminology: We define *phenotypic plasticity* as a genotype producing different phenotypes

106   depending on the environment in which it is expressed. This is broadly synonymous with

107   individuals (whose genotype is constant) showing a *plastic response*. The response is

108   adaptive if it increases fitness compared to continued expression of the phenotype produced

109   prior to the environmental change of interest. When referring to the degree of plasticity

110   expressed by a genotype we refer to its *reaction norm* (the function relating the expression of

111   the focal trait to an environmental parameter). Selection for an adaptive plastic response is

112   synonymous with selection for an appropriately shaped reaction norm. Evolution of plasticity

113   can only occur if there is additive genetic variation in reaction norms (i.e. gene-by-

114   environment [GxE] effects). We should note that individual plasticity is not strictly

115   synonymous with GxE, despite individuals having different genotypes, because individuals

116   might vary phenotypically across focal environments for purely non-genetic reasons (e.g. a

117   good start in life might increase their ability to adjust their phenotype [permanent

118   environment effects: see 31]).

119

120   **2. How can sexual selection affect female reproductive output?**

121

122   Regardless of whether or not phenotypic plasticity in sexually selected traits is adaptive for

123   males, it seems unlikely on the face of it to affect the likelihood of population extinction in a

124   rapidly changing environment. This is because the expression of sexually selected traits

125 simply determines which males mate. *Does this have any bearing on how many females there*

126 *are, how often they breed, and the success of each breeding attempt?* Sexual selection on

127 males will only influence population extinction if it affects these three key demographic

128 parameters. We therefore first address the role of sexual selection in determining female LRS

129 before we consider additional compounding effects of male plasticity. Naïvely we might

130 assume that males cannot affect mean female LRS because females are rarely limited in their

131 ability to acquire a mate, but this conclusion would be wrong [32]. Males can affect mean

132 female LRS for three main reasons.

133

134 (a) There is a positive genetic correlation because successful males transfer genes that elevate

135 their daughters' LRS (r_G) [33,34]. This is most likely to occur when there is additive genetic

136 variation for naturally selected genes that determine condition, and male sexual traits are

137 condition-dependent (see §4). Conversely, there could be a negative correlation if successful

138 males transfer sexually antagonistic genes that elevate their sons' mating success but lower

139 their daughters' LRS [35,36]. A negative inter-sex genetic correlation (r_{MF}) for fitness has

140 been documented in several species [37-41], but this is not strictly equivalent to a negative

141 genetic correlation between male sexual competitiveness and female LRS. For example, even

142 in the absence of sexual selection, a negative r_{MF} could arise if natural selection favours

143 different genotypes in each sex, which is likely given sex-specific life histories [e.g. 42-44].

144 Strictly speaking it is necessary to measure the genetic correlation (r_G) between male and

145 female fitness that is attributable to sexual and natural selection respectively. This correlation

146 is likely to vary predictably depending on the environment in which it is measured

147 [45,46](see §5). It should also be noted that a positive r_G is not equivalent to female choice

148 for genetic quality ('good genes'), as this refers to a sire's effect on mean offspring fitness

149 (i.e. daughters *and* sons) [47-49].

150

151 (b) There is a phenotypic correlation (r_P), between a male's mating success and his mate's

152 LRS because male sexual competitiveness covaries with: (i) traits that determine the level of

153 sexual conflict over mating and sperm use (e.g. seminal toxins, traumatic damage to females)

154 [50-52]; (ii) the likelihood he passes on sexually transmitted infections [53,54]; (iii) the

155 quantity and/or quality of resources transferred (e.g. parental care, nuptial gifts) that improve

156 a female's ability to rear viable offspring [55-57]; and, (iv) his daughters' LRS due to his rate

157 of 'offspring provisioning' (e.g. food intake when young, or access to breeding resources as

158 an adult) [e.g. 58,59]. When calculating the contribution of successful males to a population's

159 reproductive output we need to determine how many daughters they sire compared to the

160 average male, and if their daughters are of above average fecundity [60]. In general, however,

161 there is only weak empirical evidence that sire attractiveness affects the offspring sex ratio

162 [61].

163

164 (c) Even if we ignore the issues of which males mate, male-male competition leads to the

165 coevolution of sexually selected male traits and corresponding female traits (e.g. mate choice,

166 mating resistance), that generally reduce female LRS below the level that would occur in

167 their absence [62]. First, investment into sexual traits lowers males' parental investment,

168 reducing the mean output per breeding event [21]. Second, intense sperm competition can

169 cause sperm depletion that lowers fertilization success, reducing the output per breeding

170 event. This is most common when only a subset of males acquire mates [63,64]. Third, sexual

171 conflict that arises when female evade and resist males tends to increase the interval between

172 breeding events, and lowers female fecundity due to energetic costs, lost foraging time and

173 allocation of resources to defensive traits instead of offspring [65-67]. Sexual conflict can

174 also kill females, reducing the number of breeding females in a population [68,69].

175

3. The net effect of sexual selection on mean female reproductive output

177

For all of the scenarios covered in §2a,b there are both theoretical models and empirical data suggesting that mating with more successful (i.e. competitive) males can have either a positive or negative effect on mean female LRS, depending on contingent factors. For example, the proportion of genes with sexually antagonistic effects tends to be lower when populations are in a novel or changing environment [e.g. 70-72] (§5). Consequently, there is no consensus as to how variation in male mating success due to sexual selection affects the likelihood of population extinction. In contrast, all of the sexually selected processes in §2c reduce mean female LRS. The net effect of sexual selection on mean female LRS, hence population extinction risk, is therefore uncertain [3-7], although it seems on balance to be beneficial.

188

First, sexual selection is positively correlated with lineage diversification (speciation minus extinction rates) across many taxa [73,74; but see 75,76]. If this relationship is partly driven by lower extinction rates, it is plausible that sexual selection has a beneficial effect on mean female LRS. Second, a recent study of ostracods found that persistence in the fossil record (i.e. time to extinction) was shorter for species assumed to have more intense sexual selection on males [77]. Third, numerous experimental evolution studies have created breeding lines in which sexual selection is either present (females have access to many males) or absent (enforced monogamy). The two types of lines often evolve differences in female fecundity, lifespan, offspring viability and other traits [review: 78]. Sexual selection clearly elevates components of female LRS in some studies [e.g. 79-81] but not others [e.g. 82-84].

199 Intriguingly, a few studies have directly shown that sexual selection lowers the rate of line

200 extinction [85-88].

201

202 **4. Environmental drivers of plasticity in sexually selected male traits**

203

204 In §2 we noted that sexually selected male traits can vary in the costs they impose on female

205 LRS (e.g. ornaments versus seminal toxins). A key challenge in understanding how plasticity

206 affects population extinction risk is therefore to predict how males plastically allocate

207 resources into different sexually selected traits as the environment changes. We defer

208 discussion of this topic to §6. In this section, we simply introduce three key factors that

209 induce plasticity in sexually selected traits: environment-dependent resource availability, the

210 social environment, and the signalling environment. We emphasise the benefits to males of

211 these plastic responses with the implicit understanding that whether they are adaptive or not

212 also depends on the costs of developmental/cognitive mechanisms that allow for plasticity,

213 the capacity to detect environmental cues, and the likelihood of misinterpreting these cues

214 [89-92]. See [10] for a more complete discussion of the costs of plasticity in the context of

215 adaptation to novel environments.

216

217 *Condition-dependence traits: a plastic response to resource availability*

218

219 'Condition' is defined as the acquired resources that an individual can strategically allocate to

220 life history traits [93]. Condition is a simple concept invoked in numerous sexual selection

221 models [49; 94,95], but it is notoriously difficult to measure [96,97]. Nonetheless, it is often

222 stated that most sexually selected traits are strongly condition-dependent [98-100]. This claim

223 is based on trait expression positively covarying with environmental variation in resource

224   availability, and this covariation being stronger for sexually than naturally selected traits

225   [97,101] (e.g. a greater change in sexually than naturally selected traits when diet is

226   manipulated). It remains unclear to us whether other key life history traits (e.g.

227   immunocompetence, female fecundity) are, in fact, less condition-dependent than sexually

228   selected male traits [review by 98; but see 99,102]. Nonetheless, phenotypic plasticity in

229   sexually selected traits attributable to environmental variation in resource availability is often

230   high. This is consistent with a zero-sum game in which success at competing for mates and

231   eggs is largely determined by a male's relative investment in attractiveness, fighting ability

232   and sperm competitiveness [103,104].

233

234   Variation in condition among individuals arises due to contingent external factors (e.g. season

235   of birth that affects resource availability in the environment) and direct effects of many

236   naturally selected traits that determine the ability to acquire or assimilate resources (e.g.

237   foraging ability, immune function). Strictly speaking we cannot treat condition-dependence

238   as synonymous with phenotypic plasticity. Why? Plasticity involves a change in trait

239   expression for a given genotype due to the environment. In contrast, condition-dependence

240   could reflect differences in the resources that can be allocated to a trait that arise solely from

241   genetic differences among individuals rather than environmental factors. However, we think

242   it is biological sensible to assume that phenotypic variation in condition-dependence traits

243   arises due to both genetic and environmental variation. In addition, we assume that condition-

244   dependence is almost always associated with GxE interactions (hence additive genetic

245   variation in reaction norms) when considering large environmental changes because when the

246   environment changes in unexpected directions it seems likely that only some of the existing

247   standing genetic variation will yield phenotypes that improve an individual's fit to the

248   environment and thereby increase condition.

249 Crucially, variation in condition among males is 'revealed' in condition-dependent, sexually

250 selected traits. So male mating success is potentially correlated with additive genetic

251 variation for naturally selected traits that benefit females, thereby making r_G positive if

252 condition-enhancing genes elevate both male mating success and female LRS. Phrased

253 slightly differently, condition-dependent traits provide a mechanism whereby sexual selection

254 can eliminate deleterious alleles from a population, regardless of whether they arise due to

255 mutations, gene flow between locally adapted populations [13,105,106], or mismatch due to

256 environmental change [5,107; but see 108]. The existence of condition-dependent, sexually

257 selected male traits might therefore seem likely to elevate mean female LRS because of the

258 genetic benefits to females of mating with males in good condition. Unfortunately, this

259 conclusion is premature because many condition-dependent traits also damage females as a

260 by-product of conferring an advantage to males when there is sexual conflict over mating

261 (e.g. [109]). This makes it crucial to know how males allocate resources to different

262 condition-dependent traits as resource availability changes due to the environment (see §6).

263

264 ***The social environment: the response to cues of sexual competition***

265

266 Males could benefit from plastic responses of sexually selected traits to the number of

267 competitors, the sex ratio, and other social factors that affect the compound probability of

268 obtaining a mate and their sperm achieving fertilization. The most common plastic responses

269 are shifts in sperm production, ejaculate size, and rates of courtship or aggression [63, 110-

270 114]. Studies that examine plastic responses to the social environment by males rarely

271 quantify the effect on female reproductive output [115,116; but see 117]. Instead, researchers

272 usually extrapolate from effects of male traits on females in other studies to predict how male

273 plasticity will alter female LRS. For example, male *Drosophila* that perceive higher rates of

274   sperm competition mate for longer and stimulate higher rates of egg laying [118]. All else

275   being equal, this implies that male plasticity might elevate female LRS, but this is obviously

276   contingent on the mortality costs to females of a male-induced increase in productivity [e.g.

277   119]. In other studies, male plasticity seems likely to reduce female LRS. For example,

278   dominant males in domestic fowl mate more often and produce more sperm than subordinates

279   but, unlike subordinates, ejaculate quality decreases over successive copulations [120].

280   Greater investment into sperm in a more competitive social environment could therefore

281   lower female LRS if it reduces egg fertilization rates.

282

283   Our understanding of how plastic response of males to social cues affect female LRS is

284   limited. In some cases, we can use theory to reliably predict plastic responses in specific male

285   traits (e.g. strategic ejaculation [113]). In other cases, the plastic response is not in the

286   predicted direction. For example, there were no consistent effects of perceived future mating

287   opportunities on investment into either pre or post-copulatory sexual traits by guppies [121];

288   nor did male mice adjust their ejaculates to the number of potential mating opportunities,

289   although they did so in response to the perceived risk of sperm competition [122]. These

290   anomalies might arise because the marginal benefits of allocating resources to different

291   sexually selected traits depend on the level of mating and fertilization competition [114].

292   Again, this means it is crucial to be able to predict how males allocate resources to different

293   traits if we want to relate male plasticity to female LRS (§6).

294

295   ***The signaling environment***

296

297   There is good evidence, especially in species where males call to attract females, that males

298   adjust their signals to the transmission properties of the environment. These are often

299 textbook examples of adaptive plasticity. For example, studies show that anthropogenic

300 factors, such as urban noise and artificial lighting, impose direct selection on sexually

301 selected male traits [123,124]. Numerous studies have further reported differences between

302 urban and rural populations in sexually selected traits, such as bird song [125,126; review:

303 127]. Many of these differences are in the direction predicted by functional considerations

304 about signal transmission efficacy [128]. It seems improbable that selection on male genetic

305 variation in song imposed by urban noise is responsible for urban-rural population differences

306 [but see 129]. Given the recent origin of cities, these differences instead implicate plastic

307 responses due to learning, and even cultural evolution. In general, it seems unlikely that male

308 plasticity in response to the signaling environment will affect mean female LRS. It might,

309 however, reduce female mate search costs by increasing males' conspicuousness; and it could

310 make it easier to discriminate between potential mates, which would increase the strength of

311 sexual selection which can then affect female LRS (§5).

312

313 **5. Male plasticity and female reproductive output due to the genetic correlation (r_G)**

314 So far, we have broadly discussed how sexual selection might affect female LRS (§2,3), and

315 then described the main types of plastic responses of male sexual traits (§4). Next, we ask

316 how male plasticity affects mean female LRS, hence population extinction (§1), driven by the

317 genetic correlation (r_G) between non-random male mating success due to the expression of

318 sexually selected traits and female LRS. We mainly emphasise the role of condition-

319 dependence (i.e. plasticity when due to the environment) in male sexually selected traits.

320 In general terms the observed phenotypic response to selection (R) of a trait in a two-trait

321 system is:

322 $$R_x = h_x^2 S_x + r_{xy} h_x h_y S_y \qquad (1)$$

323

324   where $h^2 = V_A/V_{Phenotype}$ = heritability, S = selection differential

325

326 Here we can think of x = female LRS, y = male mating success, so $r_{xy} = r_G$ [equations 11.6

327 and 19.3 in 130]. If the genetic correlation (r_G) between female LRS and male sexual

328 competitiveness is positive then non-random mating due to sexual selection on males hastens

329 the fixation of genes that improve female LRS above that due to natural selection on female

330 LRS. The magnitude of r_G depends on the additive genetic variation (V_A) in male mating

331 success and female LRS and their covariation (r = covar(x,y)/sqrt[var(x)var(y)]), while the

332 correlated response to selection on male mating success on female LRS due to a non-zero r_G

333 also depends on the heritability of male mating success. If sexual selection is weak (i.e.

334 variation in mating success is mainly due to chance) then there is little difference between

335 mean male mating success and the mating success of those males that breed, so S for mating

336 success is small; and the heritability of male mating success is also low because there is no

337 effect of genetic variation in sexual competitiveness on male mating success. In the absence

338 of sexual selection, a positive r_G has no effect on female LRS. Simply put, if females mate

339 randomly they do not disproportionately mate with males with genes that elevate female

340 LRS, even if $r_G = 1$.

341

342 Given condition-dependent expression of sexually selected male traits, theory suggests that r_G

343 is more positive in a novel or rapidly changing environment, as both sexes tend to have

344 phenotypes that are similarly displaced from their selected optima (Fig. 1a). Genes under

345 natural selection in males are therefore likely to benefit females because they will equally

346 move females towards their new optimum. If so, the inter-sex genetic correlation for fitness

347 (r_{MF}) is positive [45,46]. More specifically for r_G, some of the V_A in condition-dependent,

348 sexually selected male traits that determine male success is due to genes that otherwise

349 improve naturally selected traits (§4). As such, more competitive males carry genes that tend

350 to elevate mean female LRS if natural selection acts concordantly on both sexes, hence $r_G >$

351 0. In contrast, in a stable environment, genes that are under consistent selection in both males

352 and females (e.g. genes for condition) tend to reach fixation. The V_A in condition is then

353 reduced so that a greater proportion of the standing additive genetic variation in LRS and

354 male mating success is attributable to sexually antagonistic genes, hence $r_G < 0$. Studies that

355 compare r_{MF} (often, but not always, identical to r_G; see §2) between populations which are

356 either well or poorly adapted to the local environment suggest that r_{MF} is more positive in

357 novel environments [34,70,131]; but see [132-134], although a full meta-analysis is still

358 needed. Of course, several key assumptions underlie the claim that r_G is more often positive

359 in novel environments [34,46,108].

360

361 First, if additive genetic variance changes due to gene-by-environment (GxE) interactions

362 [134] this can affect r_G or r_{MF} in unexpected ways. To take an extreme case, $r_G = 0$ if there is

363 no additive genetic variation in male mating success in a new environment where chance

364 alone determines which males mate. For example, consider what happens in the case of a

365 sexually dichromatic cichlid fish with female mate-choice based on male colour that lives in

366 clear water if the environment becomes highly turbid [135-137]. Even if condition still

367 determines male colouration, bright males do not have higher mating success and the link

368 between condition, which still elevates female LRS, and male mating success is broken.

369 Second, sex-specific optima in a novel environment might be associated with greater intra-

370 locus sexual conflict. For example, consider a population with a mean phenotype for a

371 naturally selected trait that is intermediate between the male and female optima. A standard

372 assumption is that, in the novel environment, the trait optima are displaced in the same

373 direction for both sexes [138] (Fig. 1a). If, however, they are displaced in opposite directions

374   then the potential for intra-locus sexual conflict will increase (Fig. 1b; see also Fig.1 in 134]).

375   Even if the new sex-specific optima are displaced in the same direction, if they are further

376   apart in the novel environment then r_G will tend to be more negative once the population

377   mean trait exceeds the new optimum of one sex (Fig. 1c). Third, even if sex-specific optima

378   are minimally displaced, there could be greater sexual antagonism in a novel environment

379   due to sex-specific GxE interactions. For example, a genotype beneficial to both sexes in the

380   original environment could produce a phenotype that is displaced much further from the

381   female than male optimum in the novel environment. This is plausible given that a novel

382   environment might affect sex-specific life histories (i.e. the sexes differ more in the particular

383   traits that increase their condition due to, for example, greater sex differences in the available

384   prey types). The interested reader is referred to [108] for a useful summary of other ways in

385   which r_{MF}, r_G (and S) might be affected by a changing environment.

386

387   So what role does male plasticity play in increasing the extent to which sexual selection on

388   males increases female LRS in a novel environment? Unfortunately, most theoretical studies

389   of how sexual selection facilitates adaptation implicitly assume that sexually selected traits

390   are condition-dependent. This is because it is the only obvious mechanism to link the process

391   of females disproportionately mating with males with greater investment in sexually selected

392   traits (usually modelled assuming female choice) to genetic benefits that elevate female

393   reproductive success [13,45,46,106]. However, this approach precludes answering the

394   broader question of whether r_G is more positive, sexual trait heritability ($h^2 = V_A/V_P$) is higher,

395   or S is larger in a novel environment if sexually selected traits are phenotypically plastic

396   instead of fixed in expression (i.e. whether they increase the value of $r_{xy}h_xh_yS_y$ in equation 1).

397   We can, however, still ask how sexually selected male traits being condition-dependent might

398   affect the values of these three key parameters when the environmental changes. It is worth

399   noting here that each of these terms incorporates elements of the other so they are not

400   independent (e.g. V_A affects the value of r_G and $h^2_{male\ mating}$; and $h^2_{male\ mating}$ incorporates an

401   element of S, i.e. if $S = 0$ then $h^2_{male\ mating} = 0$)

402

403   *Plasticity and the heritability of male mating success*

404

405   Condition-dependence implies that the environment affects phenotypic variation in sexually

406   selected male traits, hence sexual competitiveness, and mating success. The degree of

407   phenotypic displacement of the average male from the naturally selected optimum in a novel

408   environment is likely to affect the distribution of male condition, hence V_P [102, Fig.1 in

409   134]. Males will generally be in poorer condition, and the resultant decline in mean condition

410   is likely to be associated with greater variation in condition [see 108,139]. This implies that

411   male mating success has lower heritability in a novel environment due to the larger V_P, but

412   heritability (V_A/V_P) also depends on V_A. Additive genetic variation in condition, hence sexual

413   trait expression, is likely to change in unpredictable ways in a novel environment simply

414   because of GxE interactions. This makes it unlikely that we can predict how condition-

415   dependence will affect heritability. There is, however, some evidence from meta-analyses

416   that heritability is lower in less favourable environments, although this is contingent on the

417   type of trait being measured [140]. One explanation for lower heritability of condition in less

418   favourable environments (i.e. when extractable resource availability is lower due to

419   maladaptation) is that there is a minimum threshold below which individuals die, which

420   reduces V_A for condition among surviving males.

421

422   *Plasticity and the strength of sexual selection*

423

424   The strength of sexual selection affects both S and h for mating success in equation (1). The

425   heritability of male mating success depends on non-random variation in mating success due

426   to sexual selection on males (because this creates the necessary link between V_A in male

427   sexually selected traits and mating success). We therefore need to know how a novel

428   environment changes the types of males that females choose, and what factors determine

429   which males win fights, or have greater sperm competitiveness.

430

431   Initially, greater V_P for male condition in a novel environment seems likely to increase the

432   strength of selection S because the contrast between high and low condition males is

433   exacerbated. But this need not be the case. For example, the extent to which choosy females

434   discriminate between males based on ornament size might decline when the mean ornament

435   size is smaller due to males being maladapted and in poorer condition. This could occur if

436   females use size-based threshold rules to determine which males are suitable mates: if most

437   males fall below the threshold, they will be equally (un)attractive as mates. More generally,

438   phenotypic variance in traits depends on how resources are allocated to different sexually and

439   naturally selected traits. The relationship between the mean condition of males and how

440   males allocate resources to different condition-dependent traits is hard to predict (§6). Plastic

441   shifts in allocation, even if only among sexually selected traits, could lead to unexpected

442   outcomes. These include males in better condition being less successful because plastic

443   responses are maladaptive in the novel environment. This is plausible because these

444   responses have evolved based on females' behaviour in the original environment. For

445   example, greater investment into ejaculate size by males in better condition might be

446   disadvantageous if females in a novel environment do not mate multiply. In sum, condition-

447   dependent changes in allocation could alter V_P in key sexually selected traits in ways that

448   change the proportion of variation in mating and fertilization success that is attributable to V_A

449   in condition, thereby reducing the variation in male mating success that also increases female

450   LRS. Similar adverse outcomes for female LRS could arise when males plastically respond to

451   cues about the social, or even signalling, environment that alter covariation between V_A in

452   condition and male mating success.

453

454   Another way that male plasticity could weaken sexual selection is if males respond to social

455   cues by 'specialising' in increasing their success at certain stages of reproduction (e.g. mate

456   acquisition versus fertilization). Here we note that, for ease, we previously treated sexual

457   selection as synonymous with variation in mating success in equation 1. Strictly speaking we

458   should refer to "variation in fertilization success which arises from the combined effects of

459   female mate choice, cryptic choice, the intensity of sperm competition, and how winning

460   male-male contests elevates mating and fertilisation success". For brevity we do not.

461   Specialization can reduce variation in male reproductive success under sexual competition if

462   males make the 'best of a bad job' (e.g. small males or those in low condition sneak rather

463   than court [141]). More generally, when males plastically adjust their investment in sexually

464   selected traits to take advantage of information about individual females, this can reduce

465   variation in male fitness. For example, males can plastically adjust ejaculate size based on

466   cues about a female's previous mating history or the likelihood that she will re-mate [113].

467

468   Conversely, plasticity could increase V_P in male reproductive success under sexual

469   competition. For example, a lack of detectable variation among males in one trait in a novel

470   environment could favour females that shift their attention to assessing males using another

471   trait [135; see also 142]. If males plastically adjust their investment into sexual traits that are

472   still detectable by females [143] this could increase (or decrease) the variation in

473   attractiveness among males depending on the ease with which females can discriminate

474   among males for different trait-environment combinations. In general, although many studies

475   have documented that plastic responses affect which males mate or sire offspring, far fewer

476   studies have quantified how this affects the net strength of sexual selection on different male

477   traits.

478

479   ***Plasticity and r_G***

480

481   To recap, r_G depends on V_A in male success under sexual competition, V_A in female LRS, and

482   their covariation. We have already discussed how condition-dependence might affect V_A in

483   male success via the heritability (V_A/V_P) of male success. However, we glossed over the

484   possibility that the proportion of V_A in male success attributable to condition changes across

485   environments. This will affect the covariation between male success and female LRS. For

486   example, if most V_A in male success is due to sexually antagonistic genes then r_G will be

487   negative. A major consideration is therefore how male plasticity, other than that due to

488   condition-dependence, effects the proportion of V_A in sexually selected traits attributable to

489   sexually antagonistic genes. To our knowledge, few empirical or theoretical studies have

490   explored this question. For example, does plastic expression by males of sexually selected

491   traits in response to changes in social cues, such as lower population density in a novel

492   environment, decrease the likelihood that male sexual traits are associated with genes that

493   elevate female LRS?

494

495   **6. Direct effects of males on female reproductive output**

496   Males with greater expression of certain sexually selected traits can either elevate or depress

497   the LRS of their mates (via r_P) (§2a,b). For this to affect mean female LRS there must be

498   sexual selection so that some males have higher mating success than others. More generally,

499 sexual competition among males can affect female LRS irrespective of which males end up

500 mating (§2c). Any effect of male plasticity on mean female LRS therefore depends on how it

501 affects the strength of sexual selection and which male traits increase males' success (i.e. are

502 they those that increase or decrease female LRS?). We have already discussed the strength of

503 selection in §5 so we now focus on plastic changes in selected male traits.

504

505 First, it seems self-evident that the mixture of plastic responses by males to social cues, the

506 signalling environment and the total resources they acquire and assimilate (i.e. condition) due

507 to being in a different environment makes it almost impossible to predict how resources will

508 be allocated to different sexually and naturally selected traits. Less obvious, however, is the

509 fact that it is still difficult to make predictions even if we only consider adaptive allocation of

510 resources to different condition-dependent traits [95]. The adaptive response to an increase in

511 condition driven by greater resource acquisition is to allocate these additional resources to the

512 trait with the greatest marginal fitness gains. (In a novel environment, where fewer resources

513 are available, we can treat this as a question of reduced investment into the trait where there

514 will be the smallest marginal decrease in fitness.) This suggests that additional resources will

515 be allocated exclusively to a single trait with the highest gain, such that only a single trait

516 exhibits positive condition-dependence. There are, however, general reasons to believe that

517 marginal fitness gains will not consistently differ among traits as a male's condition changes.

518 First, investment into a trait often yields diminishing fitness gains. For example, whenever a

519 trait increases the probability of a particular outcome, such as detection by potential mates, it

520 cannot be increased beyond its maximum value of 1. Second, the marginal fitness gains from

521 different traits are rarely independent. Fitness gains depend on the current values of other

522 traits, and traits tend to function most efficiently if they are 'balanced' so that an individual

523 operates as an integrated unit. For example, a longer tail ornament might be favoured by

524   female choice, but it will eventually become so long that investment into larger wings to

525   maintain the ability to fly is likely to be more advantageous than a further increase in tail size.

526   This should lead to plastic responses with increased expression of multiple traits in

527   environments where males have access to more resources. Third, some traits might become

528   more efficient (hence have greater fitness gains) when expressed at a higher absolute level

529   [e.g. 144]. If so, greater condition could induce a shift in allocation that manifests as an

530   increase in the focal trait, alongside a *decrease* in other (fitness-enhancing) traits [95]. This is

531   one reason why both acquisition and allocation are themselves sometimes described as

532   condition-dependent [e.g. 145].

533

534   Clearly, the sheer number of possible plastic responses by males to a change in condition that

535   arise from being in a novel environment, make general predictions about plasticity in specific

536   sexually selected traits problematic. There is no guarantee that greater condition leads to

537   equal increases in all condition-dependent sexually (or naturally) selected traits. Broadly

538   speaking, optimal condition-dependent allocation depends on the shape of the multivariate

539   function that links traits to fitness. This function depends on species-specific details, such as

540   morphological integration, the ecological context and, in the case of sexually selected traits,

541   how the intensity and type of sexual selection (e.g. mating versus sperm competition) change

542   with the environmental availability of resources that affect male and female condition.

543   Consequently, when sexually selected traits vary in their effects on female LRS (e.g. a

544   reduction in male song rate is unlikely to damage a female, while investment into seminal

545   toxins is likely to induce female mortality), it is hard to determine whether condition-

546   dependent plasticity will elevate or lower female LRS when males are in a novel (usually

547   more stressful) environment.

548

549   When there are social cues about the level of mating or sperm competition there is often a

550   clear theoretical prediction about how male investment will change for specific traits; and

551   empirical studies typically report plastic responses in the predicted direction (i.e. greater

552   investment in ejaculates as sperm competition increases) [review: 111]. However, as noted

553   for condition-dependence, it is a challenge to predict the adaptive response when sexual

554   selection acts on multiple traits. Specifically, the social setting could cause the marginal

555   benefits of investment into different traits to change because of shifts in the relative

556   importance of different sexual selection processes (e.g. courtship versus sperm competition).

557   Even when models make predictions about optimal investment into testes versus

558   weapons/ornaments in different social contexts [e.g. 94], they are hard to test because: (a)

559   there are simplifying assumptions about the constancy of natural selection which do not apply

560   if the social setting affects naturally selected traits; (b) most models predict evolution due to

561   changes in gene frequencies, rather than the optimal plastic response, but the two outcomes

562   are not necessarily in agreement (§8); (c) there is within-population variation in condition.

563   All these factors makes it harder to predict the optimal plastic response for each individual

564   [for a similar problem see 95].

565

566   Given no clear prediction about how males will allocate resources to different traits

567   depending on their condition, determining the allocation patterns that are likely to arise in

568   nature is chiefly an empirical matter. Even then, the relative amount of variation in

569   acquisition versus allocation among individuals affects the observed population level

570   correlations between traits [146; review: 145]. The two main areas with relevant data are: (a)

571   whether condition-dependent male sexual signals are honest indicators of parental care, and

572   (b) whether males with greater investment into sexually selected traits (preferred males, or

573 males that win fights for access to females), benefit or harm their mating partners compared

574 to the average male.

575

576 (a) A 'good parent' model suggests that condition-dependent sexual traits honestly signal

577 parental care, while 'differential allocation' models predict that sexual selection on males

578 lowers parental care due to the resource trade-offs that males face [147-149]. This is why the

579 relationship between condition-dependent male sexual signal and paternal care is unclear, and

580 both outcomes seem possible [150]. However, the fact that female mating preferences might

581 evolve in response to the direction of the relationship would appear to favour males being

582 'good parents', which could even lead to the evolution of male-only care [151]. But the

583 enduring challenge is to explain why attractive males provide more care when mating

584 precedes caring. In general, there must be inherent constraints on preferred males, perhaps

585 due to the social setting (e.g. strict monogamy [149], or because early mate desertion by

586 females increases the value of male care [152]), such that males gain more by providing the

587 'advertised' care than redirecting resources to pursue additional mating opportunities [see

588 also 153]. The extent to which such constraints are associated with plastic male responses to

589 condition due to environmental variation is an open question, but it is one way in which

590 plasticity could facilitate the process of sexual selection increasing mean female LRS. In

591 general, there is high variation among species in the link between male sexual trait expression

592 and how it effects female LRS through parental care, fertilization success, or other factors

593 that influence female fecundity (e.g. nuptial gifts) [55].

594

595 (b) It seems unlikely that females would prefer males that lower their LRS, but this occurs in

596 some species, and presumably reflects an on-going 'arms race' between seduction and

597 resistance that males are currently 'winning' [e.g. 50,51,154]. Mating with males who are

598 more sexually competitive can still increase a female's fitness even if it lowers her LRS if the

599 costs of mating resistance are higher than accepting such males as mates. However, selection

600 on females could lead to the evolution of the ability of females to reduce mating costs [155].

601 The net effect is that sexual selection on males can lower mean female LRS. But, to what

602 extent does male plasticity influence this process? First, condition-dependent expression of

603 damaging male traits might magnify the harmful effects of mating with more sexually

604 competitive males. Intriguingly, however, we know of no systematic review that determines

605 the extent to which, for example, experimental manipulation of male condition is associated

606 with increased expression of specific male traits that appear to harm females such as seminal

607 toxins and genital structures [but see 156]. There is, however, evidence that social cues of the

608 intensity of sperm competition lead to plastic responses in ejaculates (e.g. protein content and

609 sperm count) that lower female LRS [122,157]. In addition, recent studies suggest that greater

610 relatedness between competing males can result in phenotypic responses that reduce the

611 extent to which males lower female LRS when competing for fertilizations [e.g. 158,159].

612

613 **7. Plasticity and females**

614 We have emphasized sexual selection on males but, of course, sexual selection also acts on

615 females (e.g. female-female competition for breeding opportunities and male mate choice)

616 [reviews: 30,160,161]. What effect do sexually selected female traits have on mean female

617 LRS? Clearly, mean female LRS must decline if there is any fitness trade-off with naturally

618 selected traits [162]. If females simply used a lottery rather than expend resources on

619 competition to determine contested breeding opportunities, then the 'winning' female could

620 invest more in reproduction. Of course, the same is true for males, but a key difference is that

621 the mean LRS of breeders is likely to be more strongly affected by which females, rather than

622 males, breed. This claim is based on the assumption that there is greater variation in female

623   fecundity and parental investment than in direct male effects on female LRS (§2b). A more

624   interesting question is: to what extent does plasticity in sexually selected female traits

625   increase the realised fecundity of breeders when breeding sites and/or male mates are a

626   limited resource? If female investment in sexually selected traits is condition-dependent, but

627   the proportion of resources invested is smaller for females in better condition (so that they

628   remain more fecund), then plasticity might increase the mean LRS relative to that observed if

629   females stochastically acquired breeding opportunities. To our knowledge, the circumstances

630   where condition-dependence of female sexually selected traits elevates mean female LRS

631   have not been formally modelled. We refer the reader to [162] for an extensive review of

632   female ornament evolution.

633

634   Female plasticity is mainly studied by asking how it affects male-imposed costs, or how it

635   allows a female to choose males that increase her LRS or the fitness of her offspring. We

636   consider both. First, recent models examine in detail how plasticity affects sexually

637   antagonistic selection [163,164]. Specifically, they ask how it affects the conflict load (fitness

638   reduction compared to a hypothetical best-case scenario) of individuals involved in pairwise

639   interactions, when each party controls an antagonistic trait that decreases the other party's

640   fitness. The focus is on a situation where plasticity is unilateral, i.e. only one party shows a

641   plastic response, while the other's strategy evolves due to differential success of genotypes.

642   An illustrative case in which females are the plastic party is post-copulatory sexual conflict,

643   where males commit to a strategy by transferring seminal fluid proteins (SFPs) that females

644   then respond to plastically. The general finding is that plasticity, compared to neither party

645   showing plasticity, always reduces the conflict load of the non-plastic party, but that of the

646   other party can either increase or decrease [163,164]. The intuitive reason is as follows. There

647   are two directions in which an individual of party P (for 'plastic') might adjust its

648 antagonistic trait p when faced with a mutant of party N (for 'non-plastic') with a slightly

649 deviant antagonistic trait n. If p is adjusted in the same direction as the change in n (i.e. less

650 antagonistic mutants elicit a less antagonistic response), then plasticity selects for lower

651 antagonism in N. By contrast, if p is adjusted such that more antagonistic mutants elicit a less

652 antagonistic response, then plasticity selects for greater antagonism in N. Thus, depending on

653 the direction of the plastic response, plasticity either selects for more or less antagonism in N,

654 either increasing or reducing P's conflict load. In contrast, N's conflict load always decreases

655 because N always evolves in the direction that elicits a less antagonistic response. This is an

656 intriguing result, but its applicability to post-copulation sexual conflict probably depends on

657 biological details. For example, if SFPs elevate the oviposition rate, but females can restore a

658 nearly optimal rate with a plastic response, the evolution of more SFPs need not increase the

659 conflict load for females. Instead, regardless of the absolute amount of SFPs transferred, the

660 females' conflict load might reflect only the extent to which they are actually manipulated.

661 Similarly, regardless of the absolute magnitude of a 'female resistance trait', a male's conflict

662 load might reflect only the extent to which his mate's oviposition rate deviates from his

663 optimum. There is no compelling reason why this deviation will necessarily be smaller when

664 females exhibit a plastic rather than an evolved response.

665

666 Second, many studies have investigated plasticity in female mate choice. Empirical studies

667 have shown that choice is plastically adjusted to external factors, such as the energetic costs

668 of mate sampling, and that shifts in the threshold for acceptable mates occur based on the

669 type and rate at which prospective males are encountered [165]. There is also good empirical

670 evidence that female mate choice is often condition-dependent [166]. It is reasonable to

671 assume initially that these are mainly cases of adaptive plasticity because the inherent costs of

672 mate choice suggest that selection favours random mating if choosiness provides no benefits

673 [49]. The genetic benefits of choosing certain males as mates are small or absent in many

674 species [167], so adaptive mate choice implies that plasticity is likely to elevate female LRS.

675 It should be noted, however, that while plasticity might increase female LRS in the short-

676 term, it could favour the evolution of male traits that lower female LRS. An obvious example

677 is that greater mean female choosiness due to plasticity selects more strongly for coercive

678 male traits that tend to lower females' fecundity or longevity [62].

679

680 If natural selection acts similarly in both sexes there is a scenario in which condition-

681 dependent female choice can elevate mean female LRS. The opening premise is that local

682 adaptation is reduced when natural selection differs among populations and there is gene flow

683 (migration). The rate of local adaptation is increased if females prefer locally adapted males,

684 thereby reducing gene flow. In general, female mating preferences lead to local adaptation if

685 they favour males in good condition (i.e. locally adapted) [but see 13 for complexities]. This

686 general idea was modelled by [168] who developed a simple model with two patches that

687 differ ecologically and two evolving traits: an ecological trait and a female mating preference.

688 The strength of the preference for males in good condition was contingent on the female's

689 ecological fit to the local patch (i.e. her condition). In this case, condition-dependent female

690 preferences facilitate local adaptation: the costs of choice tend to slow the spread of a mating

691 preference, but with condition dependence these costs are disproportionately born by poorly

692 adapted females (who are in worse condition) thereby lowering their fitness relative to that of

693 better adapted females.

694

695 **8. Do adaptive plastic responses mirror the direction of evolution?**

696 It is tempting to assume that adaptive plasticity will produce a phenotypic shift in the same

697 direction as selection on genotypes for fixed traits. This assumption is not universally

698   justified. For example, Kahn et al. [169] modelled sex allocation decisions where mothers can

699   re-allocate parental resources to produce more offspring when some die during the period of

700   parental investment. They examined the effect of environmental stress that increases the

701   mortality of sons during the period of parental investment. Although the adaptive plastic

702   response of mothers is to produce *fewer* sons when only some mothers experience this stress,

703   the population as a whole will evolve to produce *more* sons when the stress applies globally.

704   This pattern arises because a locally-favoured trait (i.e. producing daughters) faces negative

705   frequency-dependent selection at the population level, so it is not universally advantageous.

706   Opposing directions of adaptive and evolved responses could occur in many other frequency-

707   dependent selection scenarios.

708

709   More generally, game theory often predicts the coexistence of alternative phenotypes under

710   negative frequency-dependent selection in a mixed Evolutionarily Stable Strategy (ESS)

711   [170]. A mixed ESS can manifest either as a genetic polymorphism or probabilistic

712   expression of phenotypes (at the same frequencies as fixed phenotypes). However, if

713   heterogeneity in local factors makes one phenotype slightly advantageous, then selection

714   might favour a plastic response to produce the locally favoured phenotype. For example, in

715   some spiders a mixed ESS is predicted whereby males are either monogynous (mate with one

716   female only) or bigynous (mate with two females) [171]. If the mortality risk of mate-

717   searching varies among males, then males with a below-average risk should plastically

718   exhibit bigyny [172]. But, depending on the adult sex ratio, greater mortality costs of mate

719   searching at the population level can either increase or decrease the frequency of bigyny

720   [171]. Whether or not adaptive plastic responses match the direction of evolution of fixed

721   differences in response to the same environmental cues depends on details that do not readily

722   permit generalisations. However, mismatch hinges on negative frequency-dependent

723 selection, and many adaptations are frequency-independent (e.g. temperature tolerance). If

724 selection on a trait is frequency-independent, we suggest that it will usually be true that,

725 following an environmental change, adaptive phenotypic plasticity and selection on mean

726 trait values will shape phenotypes to evolve in the same direction.

727

728 **9. Summary**

729

730 Whether sexual selection hastens female adaptation to environmental change, and thereby

731 reduces the likelihood of population extinction, is unresolved [5,11,13]. The extent to which

732 male phenotypic plasticity further enhances or diminishes the effect of sexual selection is

733 even harder to discern. We focussed on female LRS rather than, as is standard in sexual

734 selection models, mean offspring fitness. We mainly concentrated on a few ideas. First,

735 sexual selection changes the likelihood of population extinction if it affects which males mate

736 *and* this influences how many females breed and their mean LRS. Second, although a range

737 of environmental cues induce plastic responses in sexually selected male traits, the condition-

738 dependence of these traits is the factor most likely to affect female LRS in a changing

739 environment. This is because, under such circumstances, sexually competitive males are more

740 likely to transfer genes that elevate female LRS than to have sexually antagonistic effects.

741 Third, condition-dependence is important when the environment changes because it can alter

742 the strength of sexual selection, affect who mates, and change the allocation of resources to

743 different sexually selected traits that vary in the extent to which they benefit or harm females.

744

745 We conclude that there are no general rules determining whether plasticity of sexually

746 selected traits will reduce or elevate the risk of population extinction. This unsatisfying, but

747 almost inevitable, conclusion concurs with inferences draw about the effects of phenotypic

748 plasticity on eco-evolutionary dynamics [173]. There is, however, a glimmer of hope. Recent

749 theoretical models of sexual conflict over mating [163,164], offspring sex ratio adjustment

750 based on sire attractiveness [60,174], and whether plastic maternal effects are more likely

751 than plastic responses by offspring to generate adaptive outcomes [175] all show that there is

752 the potential to make predictions about the extent to which different forms of phenotypic

753 plasticity in sexually selected and allied traits facilitate adaptive evolution. The challenge

754 now is to produce models that explicitly incorporate phenotypic plasticity, in order to ask

755 questions about the role of sexual selection in facilitating population persistence in the face of

756 rapid environmental change [see 176].

757

765

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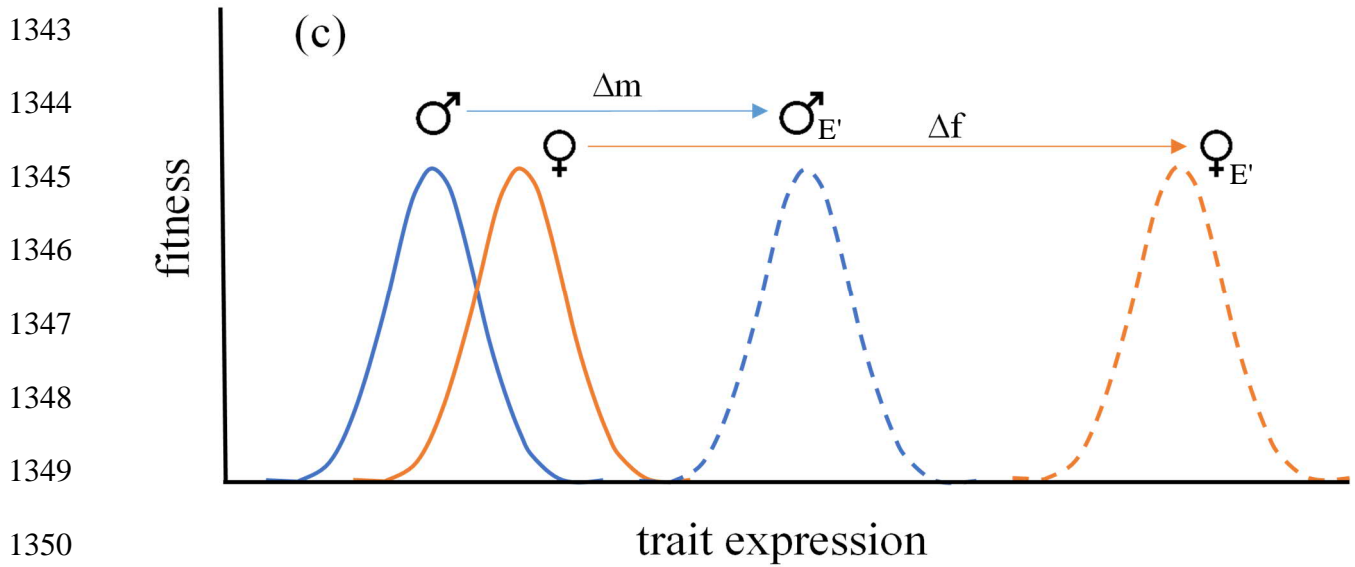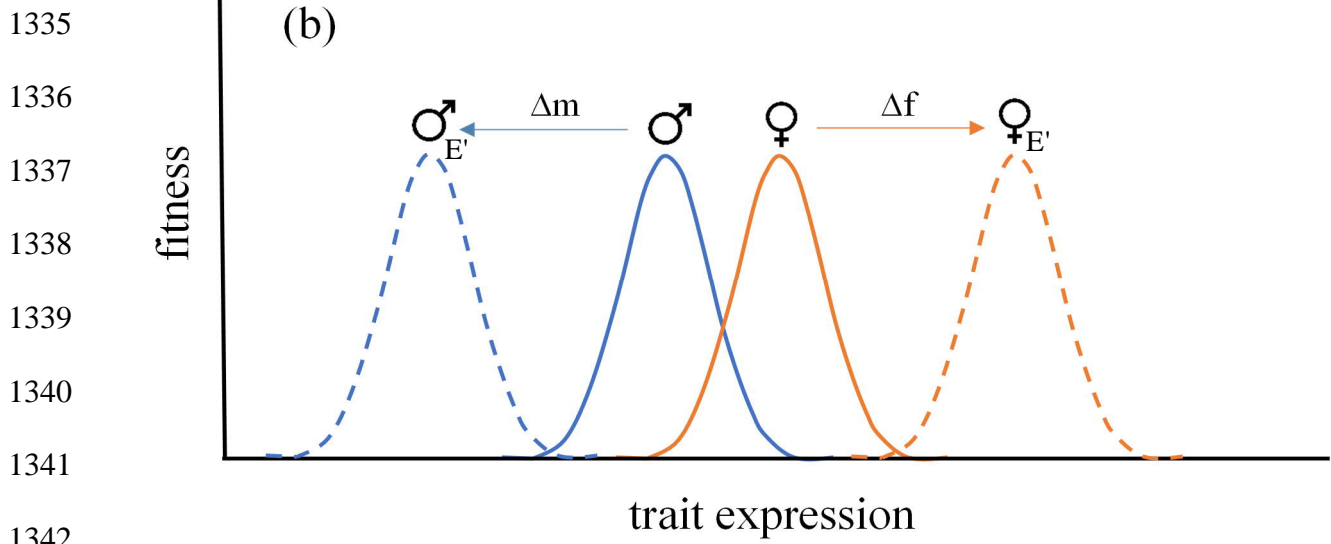1318   and sexual selection. *Evolution* **67**, 673-678.
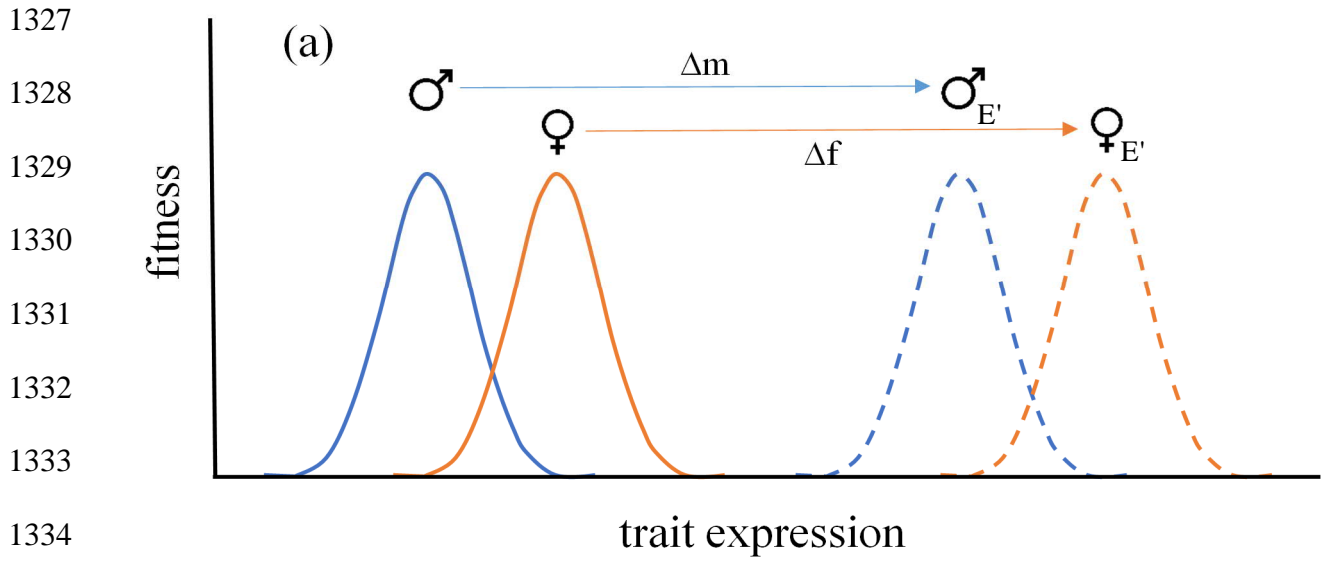
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1351 Figure 1: The likely change in the intersex genetic correlation for fitness (r_{MF}) in a changed

1352 environment. In the original environment, phenotypic values for males (solid blue) and

1353 females (solid orange) are distributed around sex-specific fitness optima. Due to opposing

1354 selection, and in the absence of sex-limited gene expression, the mean phenotype is likely to

1355 lie between the two optima. In a novel environment (E'), trait optima for each sex shift and,

1356 following selection, phenotypic values are eventually distributed around the new optima for

1357 males (dotted blue) and females (dotted orange). (a) shows the 'classic' case in which both

1358 sexes are displaced in the same direction and by the same amount ($\Delta m = \Delta f$). Genes under

1359 natural selection in males are therefore likely to benefit females and the inter-sex genetic

1360 correlation for fitness (r_{MF}) is positive. In (b) the novel environment causes the new trait

1361 optima for each sex to shift in opposite directions (here $\Delta m = \Delta f$, but with opposite signs),

1362 resulting in greater intra-locus sexual conflict i.e. r_{MF} is negative. Finally, (c) represents a

1363 case in which the new sex-specific optima are displaced in the same direction, but by

1364 different amounts (here $\Delta m < \Delta f$), such that r_{MF}, while briefly positive, becomes more

1365 negative the further the mean trait value in the population surpasses the new male optimum.