1   **Sexual conflict and STIs: coevolution of sexually antagonistic host traits with a**
2   **sexually transmitted infection**

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18 **Abstract Word Count:** 197
19 **Main Text Word Count:** 6271
20 **References:** 44
21 **Figures:** 4
22 **Tables:** 1

23

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34 **Contributions:** AMW and AFA conceived the idea, designed the model, and wrote the paper.
35 AMW performed the analysis and wrote the simulations.
36 **Data:** Should the manuscript be accepted, Mathematica and Python code will be archived in
37 Dryad and the data DOI will be included in the article.

Abstract

In many taxa, there is a conflict between the sexes over mating rate. The outcome of sexually antagonistic coevolution depends on the costs of mating and natural selection against sexually antagonistic traits. A sexually transmitted infection (STI) changes the relative strength of these costs. We study the three-way evolutionary interaction between male persistence, female resistance, and STI virulence for two types of STIs: a viability-reducing STI and a reproduction-reducing STI. A viability-reducing STI escalates conflict between the sexes. This leads to increased STI virulence (i.e., full coevolution) if the costs of sexually antagonistic traits occur through viability but not if the costs occur through reproduction. In contrast, a reproduction-reducing STI de-escalates the sexual conflict but STI virulence does not coevolve in response. We also investigated the establishment probability of STIs under different combinations of evolvability. Successful invasion by a viability-reducing STI becomes less likely if hosts (but not parasite) are evolvable, especially if only the female trait can evolve. A reproduction-reducing STI can almost always invade because it does not kill its host. We discuss how the evolution of host and parasite traits in a system with sexual conflict differ from a system with female mate choice.

Introduction

Sexual conflict over mating rate arises when male reproductive success increases with mating rate while female reproductive success is maximized at some intermediate rate (Bateman, 1948; Arnqvist and Rowe, 2005). Males evolve persistence traits to increase their mating or fertilization rate that often cause harm to females physically or physiologically. Females, in turn, evolve resistance traits that deter males or offset the physiological harm. This conflict gives rise to sexually antagonistic coevolution between male persistence traits and female resistance traits, the outcome of which determines the mating rate. Sexually transmitted infections (STIs) are, by definition, transmitted during mating and thus may play an important role in the evolution of host mating strategies. Here, we explore the interplay between STI virulence evolution and the evolution of host traits mediating sexual conflict over mating rate.

64   The virulence of STIs that affect host mortality are predicted to evolve in much the same way
65 as that of an ordinary infectious disease, i.e., evolutionary stable virulence is proportional to the
66 natural host mortality rate and depends on the shape of the trade-off between transmission and
67 virulence (Knell, 1999). Surprisingly, evolutionarily stable virulence of an STI does not depend on
68 the mating rate, though its spread and infection prevalence does (Lipsitch and Nowak, 1995; Knell,
69 1999). STIs, in turn, are known to affect the evolution of mating strategies. Theory has focused
70 on STIs in hosts with conventional sexual selection involving female mate choice. Traditionally,
71 STIs were thought to select for monogamy (Immerman, 1986; Immerman and Mackey, 1997) but
72 subsequent models showed that STIs can maintain both monogamy and promiscuity in a population,
73 as well as select for risky female choice (Thrall et al., 1997; Boots and Knell, 2002; Kokko et al.,
74 2002). (Female choosiness based on attractiveness of males is considered a risky strategy because
75 the most popular males have the highest mating rate and are most likely to be infected.)

76   By altering the mating system, the STI changes its own ecological landscape, possibly setting
77 the stage for coevolution. The outcome of coevolution can be hard to intuit. Indeed, there are
78 several examples in the host-parasite literature of coevolution leading to different or unexpected
79 outcomes compared to the evolution of either host or parasite in isolation (Gandon et al., 2002;
80 Day and Burns, 2003; Best and White, 2009). Of particular interest is a model that considers the
81 coevolution of host mate choosiness with virulence of an STI that affects host fecundity (Ashby
82 and Boots, 2015). An investigation of STI virulence in the absence of host coevolution showed
83 that a parasite that reduces the mating success of its host should evolve to be less virulent. Knell
84 (1999) suggested that hosts would subsequently lose disease-avoidance behaviours such as mate
85 choice based on the degree of parasitism of potential mates. However, when the level of host
86 choosiness based on parasitism was allowed to coevolve with STI virulence, Ashby and Boots
87 (2015) found that intermediate levels of disease-avoidance behaviour and virulence could evolve,
88 and that coevolutionary cycling could occur between host choosiness and STI virulence. These
89 unexpected results emphasize the importance of considering possible coevolutionary feedbacks of
90 an STI with host mating system.

91   Over the last 25 years, it has become clear that, in many systems, sexual conflict over mating

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92   rate plays at least as large a role in shaping the evolution of male-female interactions as conventional

93   sexual selection processes (Rice and Holland, 1997; Arnqvist and Rowe, 2005). In the absence of

94   an STI, there are several possible outcomes of sexually antagonistic coevolution depending on the

95   biology of the system. If male persistence and female resistance carry no inherent cost, traits will

96   continually escalate in an evolutionary arms race (Gavrilets and Hayashi, 2006). Incorporating

97   natural selection against persistence and resistance traits prevents runaway evolution (Gavrilets

98   et al., 2001) and allowing for the evolution of female sensitivity can lead to female indifference

99   to male traits, halting the coevolutionary process (Rowe et al., 2005). In all of these cases, only

100   females suffer the cost of mating. Given that a sexually transmitted infection increases the cost

101   of mating to both males and females it is not obvious how an STI will affect sexually antagonistic

102   host interactions. Though classic theory indicates that the ESS virulence of an STI is unaffected

103   by mating rates, there remains the potential for host-parasite coevolution. Because the traits

104   mediating sexual conflict are themselves costly, evolutionary changes in these traits driven by the

105   emergence of an STI might create epidemiological feedbacks that drive subsequent STI evolution.

106   We model an STI in a host system with sexual conflict over mating rate., i.e., the STI can

107   coevolve with sexually antagonistic host traits (male persistence and female resistance). We examine

108   how an STI changes the escalation of sexually antagonistic traits in the host, as well as how

109   the evolution of these host traits affects whether an STI can establish and, if so, the evolution

110   of STI virulence. The fitness of a host is determined by both its viability and its reproductive

111   output whereas these two fitness components of the host are not equally important to the fitness

112   of a sexually (horizontally) transmitted infection. For this reason, we consider separately cases

113   where the fitness costs of the host's sexually antagonistic traits occur through viability versus

114   reproduction. In addition, we separately examine an STI that reduces host fitness via mortality and

115   one that reduces sexual fitness (i.e., female fecundity and male mating success); we refer to these as

116   "viability-reducing STI" and "reproduction-reducing STI" models, respectively. We highlight how

117   the evolutionary outcomes that occur in cases where costs of sexually antagonistic traits and/or

118   STI virulence manifest through host mortality contrast with cases where they manifest through

119   host reproduction.

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Model Setup

121 We take as our focal case the model with a viability-reducing STI where the costs of sexually

122 antagonistic (SA) traits are manifest through reductions in viability (hereafter "viability-reducing

123 STI" and "viability costs for SA traits"). Other cases are subsequently described with respect to

124 how they differ from this focal case. The results presented are from individual-based simulations.

125 Numerical solutions of an analytical model of the focal case are similar (Supplementary Material).

Host Life Cycle without the STI

127 We model an interlocus sexual conflict over mating rate by assuming sex-limited expression of male

128 and female traits (each controlled by a single additive diploid locus). A male is characterized by

129 his persistence trait and a female her resistance trait; these traits may be morphological (e.g., male

130 grasping and female anti-grasping traits of water striders; Arnqvist and Rowe 2002) or behavioural

131 (e.g., harassment by males or vigorous struggles by females in water striders; Arnqvist 1989).

132 Resistance (x) and persistence (y) levels expressed by a host are calculated as the average of the

133 trait values from each chromosome (e.g., $x = (x_1 + x_2)/2$ where x_1 is the resistance allele on

134 chromosome one and x_2 is the resistance allele on chromosome two). Definitions of key parameters

135 are provided in Table 1.

136 Each breeding season, a female encounters a certain number of males, randomly drawn from

137 a Poisson distribution with mean α. The probability of mating during an encounter between a

138 male with persistence y and a female with resistance x is an increasing function of the difference

139 $u = y - x$ as given by $\phi[u] = 1/(1 + e^{-u})$ (following Gavrilets et al. 2001 and Rowe et al. 2005).

140 The mating rate $\phi[u]$ plays several roles in the model. Importantly, it affects female mortality as

141 females pay a viability cost d per mating. The mating rate also affects female fecundity in that a

142 given female can have zero fitness if she remains unmated (at low mating rates) by the end of the

143 breeding season. A mated female has maximum fecundity b (per breeding season) that is decreased

144 by density-dependence if population size exceeds the carrying capacity K.

145 Mortality occurs after mating but prior to offspring production. Males and females suffer

146 baseline mortality rates, μ_m and μ_f, respectively, and face nonlinearly increasing costs of expressing

147 their respective sexually antagonistic traits. A male pays a cost for his persistence trait; the strength

148 of this cost is ce^y where c is the "persistence cost" parameter. Likewise, a female pays a mortality

149 cost for her resistance trait given by δe^y where δ is the "resistance cost" parameter. Viability

150 selection against these sexually antagonistic traits, together with the cost of mating experienced by

151 females, results in three costs incurred by hosts (persistence costs to males as well as resistance and

152 mating costs to females). After mating (but before giving birth), adult females die with probability

153 $(1 - e^{-(\mu_f + \delta e^x + dm)})$ and adult males die with probability $(1 - e^{-(\mu_m + ce^y)})$.

154   Surviving females produce offspring. The number of offspring born to a mated female is drawn

155 from a Poisson distribution with mean $b(1 - (M + F)/K)$ where M and F are the number of

156 adult males and females in the population. If a female has mated with multiple males (let m be

157 the number of males she mated with), a given male sires an average of $1/m$ of her offspring, with

158 the actual sire of each offspring chosen at random from the m males. Gametes are formed with

159 free recombination between loci and alleles experience mutation with probability U_{hosts} per locus.

160 Effect size of a mutation is chosen from a normal distribution with a mean of zero and standard

161 deviation of 0.1. If the mutational step yields a negative trait value, the trait value is assumed to

162 be zero. The surviving adults and newborn offspring make up the next generation, whose mean

163 and variance in host trait values are recorded before undergoing another round of selection, mating,

164 and mutation.

165

166 **Inclusion of a Viability-Reducing STI**

167 If an STI is present in the population, it can be transmitted from an infected individual to an

168 uninfected ("susceptible") individual with probability $\beta[v]$, given a mating between them. $\beta[v]$

169 is a function of STI virulence, v, and takes the standard form $\beta[v] = v/(w + v)$, where w de-

170 termines the shape of the trade-off between transmission and virulence (see Otto and Day, 2007,

171 Chapter 12). An infected host suffers additional mortality v, e.g., adult males die with probability

172 $(1 - e^{-(\mu_m + ce^y + v)})$. A newly infected host cannot infect other hosts during the same breeding period

173 in which it became infected itself, i.e., the newly infected host must survive to the next breeding

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Table 1: Parameters and Variables

Parameter	Definition
y	male persistence trait
x	female resistance trait
$u = y - x$	mating rate metric
$\phi[u]$	mating probability function
c	persistence cost to males
δ	resistance cost to females
d	mating cost to females
α	number of males encountered
b	maximum fecundity
K	carrying capacity
M	number of males in the population
F	number of females in the population
m	number of males a given female mates with
μ_i	baseline mortality coefficient of sex i
v	virulence of the STI
$\beta[v]$	STI transmission function
w	transmission-virulence trade-off parameter

174 period before it is infectious. During this latent period, the STI undergoes mutation with proba-

175 bility U_{STI}. Effect size of a mutation is chosen from a normal distribution with a mean of zero and

176 standard deviation of 0.01. If the mutational step yields negative virulence, virulence is assumed to

177 be zero. At the start of each generation, the population mean and variance in virulence are recorded.

178

Reproduction Costs for SA Traits

180 Simulations proceed as above except the cost of sexually antagonistic traits affects aspects of re-

181 production instead of viability. Higher resistance trait values reduce female fecundity such that

182 the number of offspring born to a mated female is drawn from a Poisson distribution with mean

183 $e^{-\delta e^x} b(1 - (M + F)/K)$. Higher persistence trait values reduce male fertility by decreasing siring

184 success (i.e., the male's persistence trait increases his expected number of matings but decreases his

185 postcopulatory competitive ability). Specifically, having mated with a given female, a male sires an

186 average of $e^{-ce^y}/\sum_{i=1}^{m} e^{-ce^{y_i}}$ of her offspring, where m is the total number of males she mated with

187 and y_i is the persistence trait of male i.

Reproduction-Reducing STI

189 Simulations with a reproduction-reducing STI proceed as described for a viability-reducing STI

190 except disease virulence affects host fitness through aspects of reproduction. Female fecundity is

191 reduced by infection such that the number of offspring born to a mated female is drawn from a

192 Poisson distribution with mean $e^{-v}b(1 - (M + F)/K)$. To prevent continually escalating virulence

193 evolution, a reproduction-reducing STI faces a transmission-virulence trade-off in males. Infected

194 males have a lower probability of mating with an encountered female than an uninfected male with

195 the same persistence trait value; infected males can be considered as exhibiting lower mating effort

196 than uninfected males. Specifically, the probability of mating with an infected male is $e^{-v}\phi[u]$.

197 Note that like a viability-reducing STI, there is a latent period before an individual is infectious.

Initial Conditions

199 Individual-based simulations were carried out in Python (available upon request) for $50,000$ gen-

200 erations, well after the trait values appeared to reach evolutionary equilibrium. To initiate the

201 population, diploid hosts are assigned trait values at the resistance and persistence loci by drawing

202 random values from a normal distribution with mean $\bar{x}$ and $\bar{y}$, respectively, and standard deviation

203 0.5. In host populations infected with a sexually transmitted infection (STI), the STI is introduced

204 into 5% of hosts with the virulence of each infection drawn from a normal distribution with mean

205 $\bar{v}$ and standard deviation 0.1. Values from the last 1000 generations were averaged and reported in

206 Figs. 1 and 4. Rare stochastic extinctions of the host population are excluded from the average.

8

Results

Evolution of Host Traits without an STI

To understand how a coevolving STI affects the outcome of sexually antagonistic coevolution we first consider how the host evolves in the absence of an STI (which has been previously studied for 'mortality costs' of sexually antagonistic (SA) host traits, e.g., Gavrilets et al. 2001). Rather than reporting the SA traits directly, it is more useful to consider two other metrics instead. The average of the two trait values, $(y+x)/2$, is an indicator of the degree of escalation in the SA traits (hereafter, "conflict escalation") and is plotted in Fig. 1A-C. Second, we plot the difference between male persistence and female resistance, $u = y - x$ (hereafter, "mating rate metric") because the mating rate is an increasing function of this difference (Fig. 1D-F). The evolutionary equilibrium trait values presented in Fig. 1 and all subsequent figures are stable to different initial values.

For the range of parameters shown in Fig. 1, male persistence y always exceeds female resistance x, so the mating rate metric u is positive and mating rates are not too low. The extent of conflict escalation, as reflected by average trait values, increases substantially as the mating cost to females increases (Fig. 1A). This is driven by the female trait increasing to reduce the mating rate, while the male trait increases only slightly in response because of the nonlinearly increasing cost of natural selection. Because female resistance increases more than male persistence, the mating rate metric u declines (Fig. 1D) with increased mating cost. Conflict escalation does not change drastically with increasing cost of male persistence (Fig. 1B) but there is a reduction in the mating rate (Fig. 1E). In contrast, increasing the cost of female resistance leads to a decline in conflict escalation (Fig. 1C) accompanied by an increase in the mating rate metric (Fig. 1F). At high resistance costs, females with lower resistance better balance the costs of mating with the costs of their SA trait. Qualitatively similar patterns are observed when the costs for sexually antagonistic traits occur through reproduction (Fig. S1) rather than viability (Fig. 1), although there is a noticeable decrease in the mating rate metric.
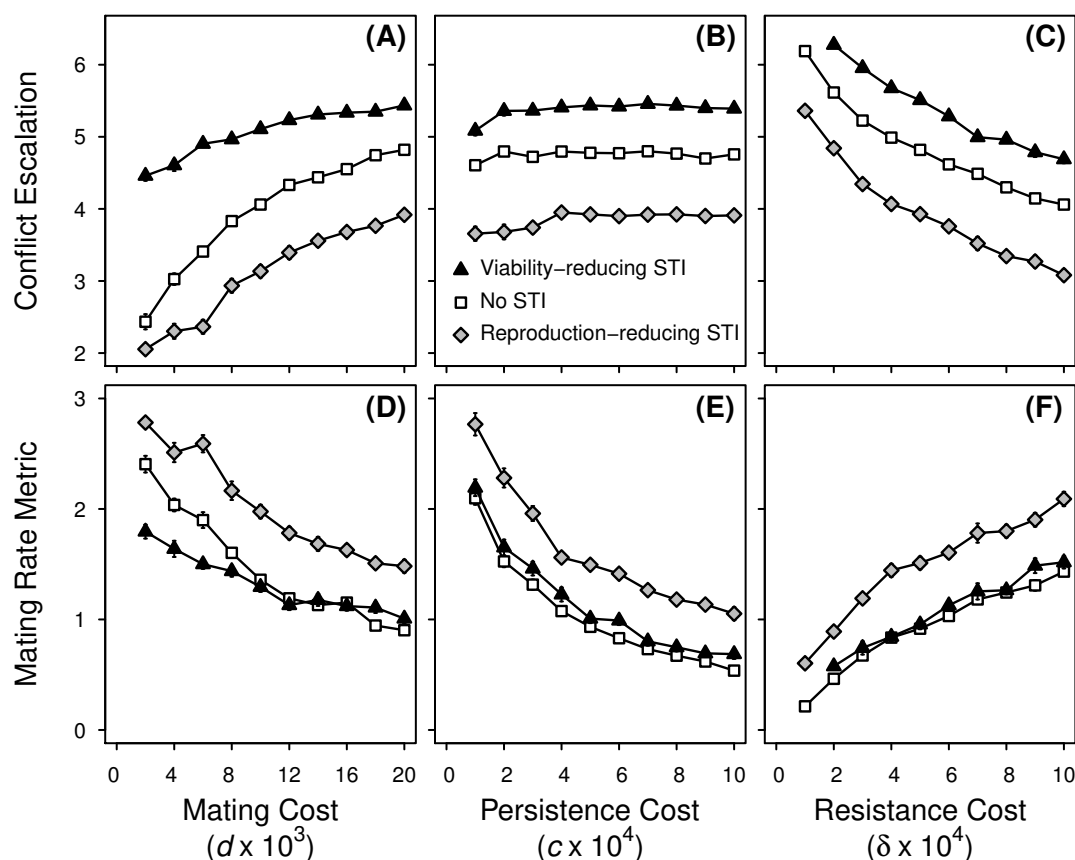
Figure 1: The outcome of coevolution between males and females in the absence of an STI and in the presence of two types of evolving STIs. Both sexes pay viability costs for expressing sexually antagonistic traits. Shown here are the degree of escalation in sexual conflict, measured as the average of y and x (A - C), and the difference between persistence, y, and resistance, x (D - F); values are at evolutionary equilibrium. The mating rate is an increasing function of the difference between male persistence and female resistance $(y - x)$; thus, we label this difference the 'mating rate metric'. Simulations with an evolving STI began at the STI-absent host trait values and the results for a reproduction-reducing STI (diamonds) and a viability-reducing STI (triangles) are a three-way evolutionary equilibrium with the STI at evolutionary equilibrium virulence v (see Fig. 4A). Parameter values (unless shown otherwise): $\alpha = 10$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, with $c = 0.0005$, $\delta = 0.0005$, $d = 0.02$. Each point represents the average of 20 independent simulations +/- standard error. Simulations in which the disease went extinct are excluded from calculating the average. At very low resistance costs, a viability-reducing STI went extinct in 100% of simulations (see Fig. 3) and there is no data for the three-way evolutionary equilibrium. Qualitatively similar patterns are observed when hosts pay reproduction costs for expressing sexually antagonistic traits (see Supplementary Material Fig. S1).

10

Evolution of Host Traits with an Evolving STI

Viability-Reducing STI

We now consider the evolution of host traits in the presence of an evolving sexually transmitted infection and then later discuss virulence of the STI. We begin by examining a viability-reducing STI in a host where the costs of SA traits occur through viability effects. The introduction of an evolving viability-reducing STI escalates the conflict between the sexes (Fig. 1). (A viability-reducing STI also causes an increase in conflict escalation if costs of SA traits occur through reproduction, though the increase is smaller; Fig. S1.) This increase is driven by females. If only the females are allowed to evolve (not shown), female resistance will evolve to be higher than male persistence to reduce the additional cost of mating arising from the risk of STI infection. In this case, $u = y - x$ is negative and the mating rate drops to low levels. A viability-reducing STI cannot persist at low mating mates. In contrast, if only the males are allowed to evolve (not shown), male persistence increases from its STI-absent equilibrium, presumably because males are selected to more quickly obtain additional mates in the face of higher mortality rates due to infection (though the change is smaller compared to the case where females evolve). The STI does not go extinct because mating rates remain high (as u increases). When females and males can both evolve, the increase in female resistance invokes a subsequent increase in male persistence so males ensure mating (Fig. 2A). As such, the net effect of coevolutionary feedback tends to be increased average trait values (i.e., increased conflict escalation). It is worth emphasizing that the evolution of male persistence in response to the escalation in female resistance allows the STI to remain in the system. If a lack of genetic variation in the male trait prevented its coevolution, the STI would go extinct after female resistance increased and caused a decrease in mating rates.

Reproduction-Reducing STI

A reproduction-reducing STI has qualitatively different consequences for the outcome of sexual conflict. Again, this outcome can be understood by first thinking about the evolution of each sex in isolation. If only females are allowed to evolve (not shown), female resistance decreases in the presence of a reproduction-reducing STI. A prevalent STI results in less mating pressure on females

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259 because infected males exhibit reduced mating effort compared to uninfected males. Consequently,

260 females do not need to invest as much in costly resistance traits. If only males are allowed to evolve,

261 there is no observed change in the male persistence trait. When males and females can both evolve,

262 the decrease in female resistance allows male persistence to decrease slightly (although to a lesser

263 extent). Thus, in contrast to a viability-reducing STI, a reproduction-reducing STI decreases the

264 degree of conflict escalation (Fig. 1A-C, Fig. 2B). This is also true if the costs of the SA traits

265 occur through reproduction (Fig. S1) rather than viability (Fig. 1). In contrast to a viability-

266 reducing STI, evolution of either host sex never drives a reproduction-reducing STI extinct for the
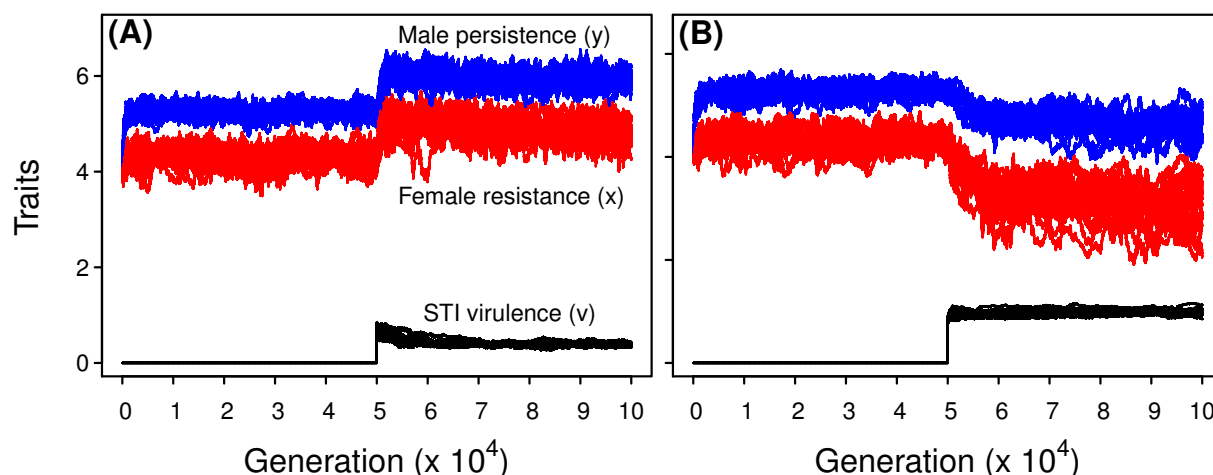
267 parameters examined here.

Figure 2: Sample runs from individual-based simulations (20 replicates) where hosts evolve to their disease-absent equilibrium values before a viability-reducing STI (A) or a reproduction-reducing STI (B) is introduced. Male persistence is shown in blue, female resistance in red, and STI virulence in black. Parameter values: $\alpha = 10$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, $d = 0.02$, $c = 0.0005$, $\delta = 0.0005$.

Establishment of the Sexually Transmitted Infection

269 In the section above, we focused on cases where both hosts and parasites can evolve and the disease

270 establishes itself within the host population. (We use the term disease "establishment" rather than

271 the more typical term "persistence" to avoid confusion with the host male's "persistence" trait.)

12

272 We now focus on understanding how a lack of genetic variation that prevents evolution of either

273 host or parasite traits affects both the establishment and virulence of the STI. We discussed one

274 important case above in which a lack of genetic variation in the male trait but not the female

275 trait prevents the establishment of an evolving viability-reducing STI. Here we focus on contrasts

276 between hosts and parasites in their evolvability (rather than on differences in evolvability between

277 male and female host traits).

278   When neither the host nor parasite can evolve, a viability-reducing STI near its equilibrium

279 virulence always establishes when introduced into host populations at equilibrium except for rare

280 stochastic extinctions (for the parameter values considered here). In comparison, a relatively avir-

281 ulent viability-reducing STI almost always goes extinct and a highly virulent one (shown in Fig. 3,

282 open symbols) establishes unless mating rates are very low (i.e., when mating costs are high and

283 there are high persistence costs to males or low resistance costs to females). In contrast, in all cases

284 examined, a reproduction-reducing STI establishes regardless of whether it was introduced at low,

285 high, or equilibrium virulence. By virtue of not killing its host, a fertility STI has a long dura-

286 tion of infection and can establish and spread at very low mating rates through rare transmission

287 events. Because of the ease of establishment of a reproduction-reducing STI (for all combinations

288 of evolvability), further discussion of extinction rates and the accompanying figure (Fig. 3) pertain

289 only to a viability-reducing STI.

290   If the host but not the parasite can evolve, then females are selected to increase their SA

291 resistance trait in the presence of a viability-reducing STI, driving an increase in average host trait

292 values and a decrease in the difference between the sexes (not shown). The accompanying decrease

293 in mating rate can drive a highly virulent, non-evolving STI extinct (i.e., at high persistence costs

294 to males, low resistance costs to females, and high mating costs to females). In Fig. 3 we show

295 the fraction of runs in which the parasite went extinct when hosts can evolve but parasites cannot

296 (black symbols). Extinction rates are higher over a wider range of parameter values when female

297 resistance costs are low ($\delta = 0.0001$, data not shown). As noted above, STI extinction is even more

298 likely if the female trait can evolve but the male trait cannot. For any of the parameter values

299 shown in Fig. 3 a viability-reducing STI is unable to invade if only the female host trait evolves

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300 (not shown). If the STI can evolve but hosts cannot evolve, an initially virulent viability-reducing

301 STI only goes extinct for very low mating rates (not shown).

302   Finally, we consider evolution in both the host and the STI (gray symbols, Fig. 3). STI

303 extinction occurs most often when the cost of resistance to females is low (Fig. 3C). At low

304 resistance costs, elevated female trait values quickly evolve in the presence of the STI, which lowers

305 the mating rate enough (albeit, transiently) that a viability-reducing STI goes extinct due to lack

306 of transmission opportunity. When the STI can coevolve with an evolving host, it is likely to

307 successfully establish across a wide range of conditions under which it would otherwise go extinct

308 (Fig. 3). Contrasting the case where the costs of sexual antagonism are paid through viability (Fig.

309 3) rather than reproduction (Fig. S2), the STI is more likely to go extinct in the latter scenario for

310 most parameter values. Differences between these scenarios in STI establishment probability are

311 likely driven by the pronounced decrease in mating rate accompanying the introduction of the STI

312 in host populations paying costs for SA traits through reproduction (compare Fig. 1D-F to Fig.

313 S1D-F).

Virulence of a Viability-Reducing STI

315 Conditional on establishment of a viability-reducing STI, three-way evolution of the STI with male

316 and female host traits can give rise to quantitatively different results than if the STI was introduced

317 into a non-evolving host population (Fig. 4A-C). A viability-reducing STI becomes more virulent if

318 hosts coevolve than if they do not, but only if the cost of SA traits occur through viability. This is

319 because the addition of the STI to the system causes evolutionary increases in female resistance and,

320 consequently, male persistence, thereby increasing the host mortality rate; optimal STI virulence

321 is expected to increase with host mortality rate (Knell, 1999). The equilibrium virulence of a

322 viability-reducing STI increases as the average host trait values increase and does not depend on

323 the difference in trait values. The difference in trait values only affects the establishment and

324 prevalence of the STI and not its evolutionary equilibrium virulence. In contrast, when the cost of

325 SA traits occur through reproduction, the virulence of a viability-reducing STI does not increase

326 with an evolving host (Fig. 4A-C) even where there is host conflict escalation in the presence of
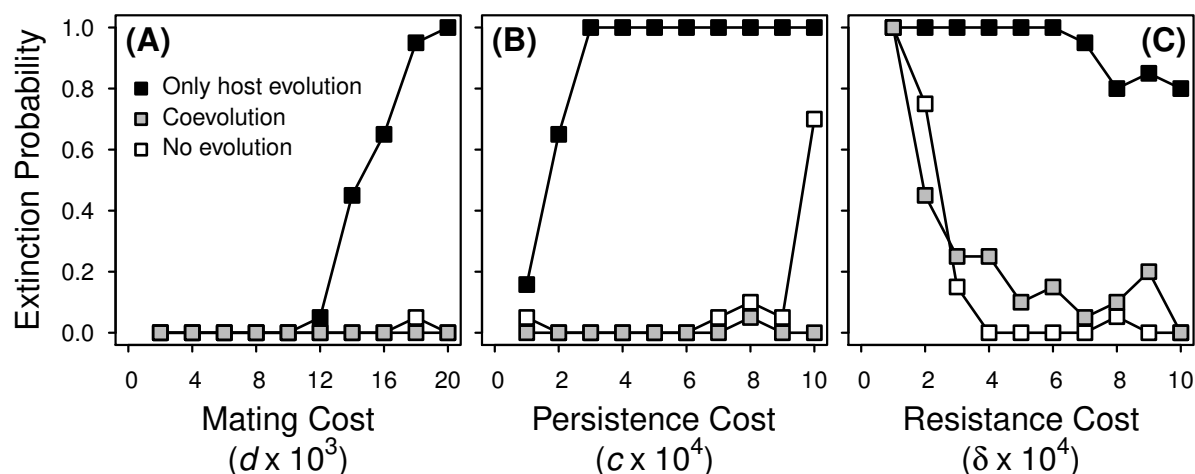
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Figure 3: Fraction of simulation runs where a highly virulent viability-reducing STI was driven extinct in hosts paying viability costs. The parasite was introduced with $v = 0.8$ at the STI-absent male persistence and female resistance host trait values and neither host nor parasite evolved (open symbols), host traits evolved in the presence of a non-evolving parasite (black symbols), or hosts and STI both evolved (gray symbols). Parameter values (unless shown otherwise): $\alpha = 10$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, with $c = 0.0005$, $\delta = 0.0005$, and $d = 0.02$. The extinction probability was determined from 20 independent simulations. STI extinction probability was slightly higher, on average, in hosts paying reproduction costs for sexually antagonistic traits, but overall patterns of extinction probability were qualitatively similar (Supplementary Material Fig. S1).

the STI (Fig. S1A-C). Escalated male and female trait values do not increase host mortality and invoke subsequent coevolution of the STI.

Virulence of a Reproduction-Reducing STI

Virulence evolution of a reproduction-reducing STI is shaped by its effects on males. In infected females, a reproduction-reducing STI does not face a transmission-virulence trade-off because the consequences of infection (decreased fecundity) do not reduce transmission opportunities. However, infected males in our model suffer reduced mating success with females. Thus, males infected with a highly virulent reproduction-reducing STI do not mate as often as uninfected males with similar persistence trait values. This reduction in transmission opportunities creates a transmission-

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336 virulence trade-off in one sex (males) that limits virulence evolution. The transmission-virulence

337 trade-off is not affected by the host trait values themselves, regardless of whether costs of SA traits

338 are paid through viability or reproduction. Consequently, the evolution of sexually antagonistic host

339 traits induced by the presence of a reproduction-reproducing STI does not invoke a corresponding

340 response in STI virulence evolution. In the absence of a coevolutionary response, virulence of a

341 reproduction-reducing STI is the same in host populations that can and cannot evolve (Fig. 4D-F).

Comparison with a Horizontally Transmitted Disease

343 In our focal case, a viability-reducing STI coevolves with a host population paying SA costs through

344 viability. As noted, STI virulence evolution depends on host mortality much like a horizontally

345 transmitted disease or "ordinary infectious disease" (OID). Furthermore, STI virulence in our model

346 causes disease-induced mortality. Given that both STI and OID virulence affect viability and are

347 shaped by the same parameters, we might expect similar co-evolutionary outcomes if we model an

348 OID infecting hosts with sexual conflict over mating rate. An OID, however, is not transmitted

349 sexually and therefore will not exert direct selection on females to reduce the mating rate. We find

350 that an OID has the same effect on the outcome of the conflict as increasing the baseline mortality

351 rate μ (not shown). In contrast to a viability-reducing STI, an OID has little effect on the extent

352 of conflict escalation. However, an OID drives an increase in the mating rate metric because male

353 persistence increases and female resistance decreases.

Discussion

355 Although the evolution of STI virulence and sexual conflict have each been studied in isolation

356 (e.g., STIs: Knell 1999; Lipsitch and Nowak 1995, sexual conflict: Gavrilets et al. 2001; Gavrilets

357 and Hayashi 2006), the link between them has received little attention. The models presented here

358 are aimed at understanding this connection. In some cases, each species reciprocally affects the

359 evolution of the other (i.e., true coevolution) whereas in other cases only one species evolves in

360 response to the other. Whether, and in which direction, each species evolves in response to the

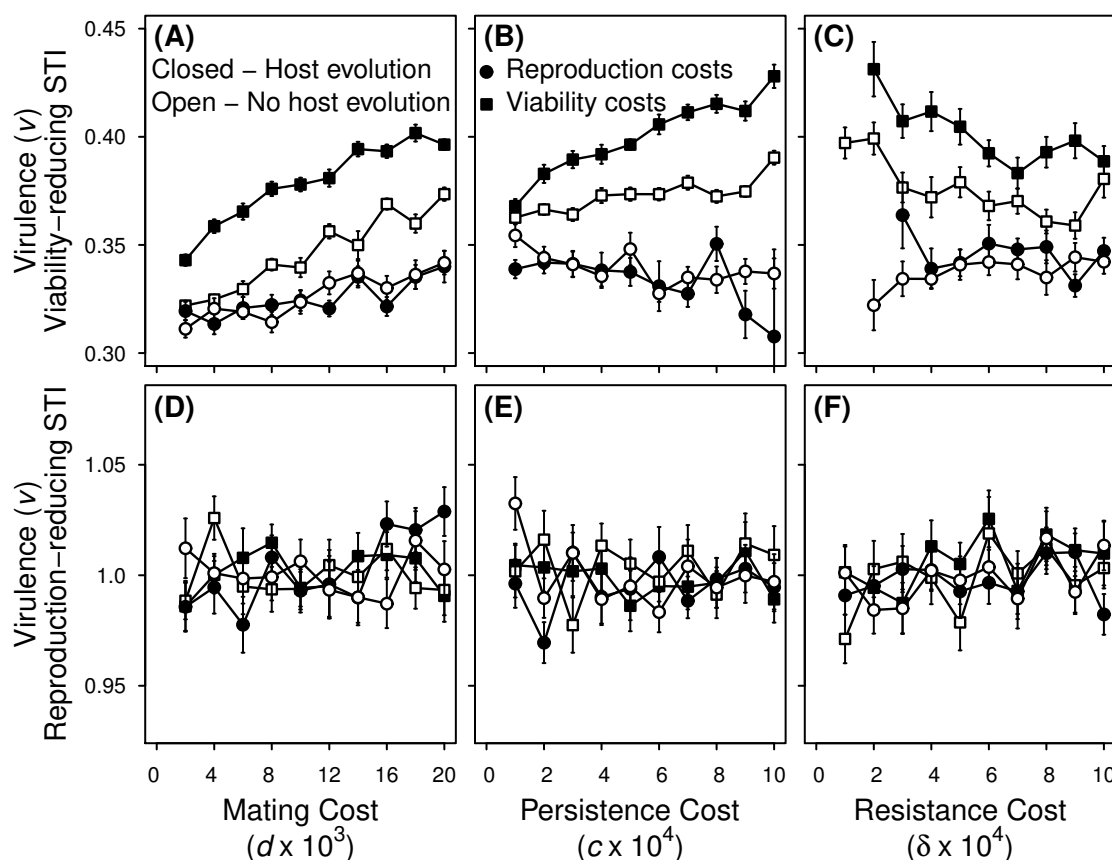361 other depends on how trade-offs are structured.

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Figure 4: Evolutionary equilibrium virulence of two types of evolving STIs in a population where hosts do not evolve (open symbols) or do evolve (closed symbols). Virulence of a viability-reducing STI (A-C) and a reproduction-reducing STI (D-F). In both cases, the parasite was introduced with $v = 0.8$ at the STI-absent male persistence and female resistance host trait values. Parameter values (unless shown otherwise): $\alpha = 10$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, with $c = 0.0005$, $\delta = 0.0005$, $d = 0.02$. Each point represents the average of 20 independent simulations +/- standard error. Simulations in which the disease went extinct are excluded from calculating the average and can lead to larger error bars at high extinction probabilities. At very low resistance costs a viability-reducing STI infecting hosts paying viability costs went extinct in 100% of simulations (see Fig. 3) and there is no data for the three-way evolutionary equilibrium.

362   We found that a sexually transmitted infection affects the level of escalation of traits mediating

363   sexual conflict within the host, but that viability-reducing STIs and reproduction-reducing STIs

364   do so in opposite ways. The introduction of a viability-reducing STI escalates the conflict. Female

17

365 resistance increases to reduce the additional cost of mating imposed by the risk of disease infection.

366 Male persistence increases to stay above increasing female resistance levels. Thus, average host

367 trait values increase, i.e., there is elevated conflict escalation. If the cost of SA traits occurs

368 through viability, then a co-evolving viability-reducing STI increases its virulence level in response

369 to these changes in the host, not because of a change in host mating rate but rather because of

370 the increased host death rate resulting from the escalated sexual conflict. In contrast, if the cost

371 of SA traits occurs through reproduction, then increased conflict escalation evolves in response to

372 a viability-reducing STI but in this case there is no feedback affecting virulence evolution.

373   In both cases described above, the three traits reach a stable equilibrium. We find no evi-

374 dence of cycling as has been observed under some conditions in a coevolution STI model involving

375 conventional sexual selection Ashby and Boots (2015). Based on our understanding of why the

376 observed changes occur, cycling is not expected. Stabilizing selection maintains the evolutionary

377 equilibrium. Male persistence increases male reproductive success at the expense of other costs

378 associated with persistence such as increased predation risk (Rowe, 1994) or reduced foraging time

379 (Robinson and Doyle, 1985). Females suffer costs of mating but must balance these with the direct

380 cost of expressing the resistance trait and the risk of remaining unfertilized if resistance is too high.

381 In the presence of a viability-reducing STI, males experience stronger selection to obtain mates

382 quickly in the face of higher total mortality and females experience stronger selection to reduce

383 the additional cost of mating associated with a prevalent STI. The escalation of sexual conflict

384 increases total host mortality, selecting for higher STI virulence, which in turn should drive further

385 escalation of the sexual conflict traits. However, the "faster than linear" increasing costs to hosts

386 of the escalating sexual conflict traits and the "slower than linear" increasing transmission benefits

387 of increased STI virulence ensures that the system reaches an equilibrium rather than evolving

388 to ever higher levels of all three traits. (Such non-linear costs are a common assumption in both

389 sexual conflict and virulence models and are necessary to have sensible equilibria (e.g., Gavrilets

390 et al. 2001 and Otto and Day 2007).) Evolutionary equilibrium is even easier to understand when

391 hosts pay costs of SA traits through reproduction because then conflict escalation does not select

392 for higher STI virulence and create the potential for a host-STI arms race.

393 Compared to viability-reducing STIs, reproduction-reducing STIs have the opposite effect on
394 the outcome of sexual conflict: a reproduction-reducing STI de-escalates the conflict. Females
395 evolve lower resistance in response to the alleviation in mating pressure they experience because
396 infected males have reduced mating success. The accompanying (but smaller) decline in the male
397 trait contributes to conflict de-escalation. Regardless of whether hosts pay the cost of SA traits
398 through viability or reproduction, virulence of a reproduction-reducing STI does not seem to be
399 affected by the decrease in average host trait values. All three traits reach evolutionary equilibrium
400 due to stabilizing selection. Continual de-escalation of the conflict is prevented by selection on
401 females to resist costly matings and selection on males to stay competitive given they will enter
402 fewer matings if they are infected.

403 When the cost of SA traits occur through viability, conflict escalation occurs in response to
404 either a viability-reducing STI or a reproduction-reducing STI. However, a coevolutionary response
405 from the STI occurs only with a viability-reducing STI. As known from past virulence theory
406 (Anderson and May, 1982; Ewald, 1983), the transmission-virulence trade-off can be thought of as
407 a trade-off between current and future transmission for the STI, the latter of which requires the
408 current host's survival. In cases where host mortality is higher for reasons not directly due to the
409 disease (e.g., higher extrinsic mortality or higher investment into SA traits carrying viability costs),
410 then future transmission is down weighted and the disease evolves higher virulence (i.e., increased
411 investment in current transmission). This logic does not apply to a reproduction-reducing STI. As
412 modeled here, the transmission-virulence trade-off in a reproduction-reducing STI occurs because
413 increased transmission *given a mating* comes at the expense of reducing a male's probability of
414 successfully obtaining a mate. That is, the two components of this trade-off both affect current
415 transmission. Thus, their value relative to one another is unaffected by extrinsic factors that alter
416 the value of future transmission.

417 One of the costs we did not explore in depth is the risk of females remaining unfertilized. In
418 the simulation model, a female achieved her full fecundity provided that she mated with at least
419 one male. However, if fertilization was not guaranteed by mating, the increased risk of being left
420 unfertilized would be expected to affect the evolution of female resistance. Thrall et al. (1997)

19

421 constructed a model investigating how male and female mating behaviour affected reproductive

422 success in the presence of an STI. At high disease prevalence, females could achieve the same fitness

423 by being monogamous and minimizing infection risk or by being promiscuous and minimizing the

424 risk of being left unfertilized but increasing the risk of infection. It is possible that if there were

425 higher probabilities of females being unfertilized in our model, divergent female strategies (low and

426 high resistance) would be maintained.

427 We investigated the full three-way evolutionary interaction over a range of mating costs to

428 females. However, many empirical investigations of sexual conflict in natural systems have reported

429 females suffer a cost of harassment instead of, or in addition to, the cost of mating (Alcock et al.,

430 1977; Rowe, 1994; Stone, 1995; Jormalainen, 1998; Watson et al., 1998). At high male densities

431 or male-biased sex ratios, the cost of rejecting harassing males can become so great that females

432 are selected to decrease resistance, increasing overall mating activity in these systems (Rowe, 1992;

433 Rowe et al., 1994; Lauer et al., 1996). However, the introduction of a STI would effectively increase

434 the cost of mating, possibly tipping the balance in favour of high resistance for females.

435 The majority of models that have investigated the evolution of host mating strategies in the

436 presence of an STI have assumed there is sexual selection, but no sexual conflict. The distinction

437 between male attractiveness and female preference versus male persistence and female resistance

438 has important consequences for the evolution of an STI. Choosiness may help females gain indirect

439 benefits from mating with a preferred male. Resistance on the other hand helps females avoid the

440 direct costs of mating (Gavrilets et al., 2001). Unless indirect benefits are strong (Thrall et al., 1997;

441 Boots and Knell, 2002), the presence of an STI will cause selection for non-choosy females because

442 the most popular males have high infection prevalence (Thrall et al., 2000). In a system with sexual

443 conflict, those males with the highest persistence traits would be more likely than average males to

444 be infected, adding to the cost of mating for females. Females do not reduce their risk of mating

445 with such males by having lower resistance (i.e., lower resistance is not equivalent to being less

446 choosy because the latter can mean less discriminant mating without changing mating frequency).

447 A female with lower resistance may have a lower fraction of her matings with high persistence

448 (probable STI-carrying) males but she will not have a fewer number of matings with such males.

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449   Moreover, she will have more total matings, increasing her infection risk. Consistent with this, we

450   found that the presence of a viability-reducing STI resulted in increased female resistance, which

451   might be construed as increased choosiness, opposite to what is observed in sexual selection models.

452   On the other hand, a reproduction-reducing STI results in decreased female resistance, which might

453   be construed as decreased choosiness. Though this would appear to match the outcome of sexual

454   selection models, the reason for this result is very different. In the sexual conflict model, reduced

455   female resistance evolves not to avoid infection but rather because the STI reduces the mating

456   effort exhibited by infected males so females do not need to invest as heavily in their resistance

457   trait.

458   For the most part, we have assumed that infection does not directly alter a male's persistence or

459   a female's resistance. However, in some cases STIs manipulate host behaviour in ways that directly

460   affect sexual conflict. An STI that influences male competitive ability could change the relative

461   costs of mating. For example, parasitized males can be less competitive (Siva Jothy and Plaistow,

462   1999; Thomas et al., 1999), decreasing the risks of mating for females in a population with a highly

463   prevalent STI. This is the scenario we modeled by allowing a reproduction-reducing STI to decrease

464   mating probability for infected males. Conversely, as in the milkweed leaf beetle (Abbot and Dill,

465   2001), STI-infected males can be more aggressive than their uninfected counterparts, which could

466   increase the number of matings and therefore the costs to females. Alternatively, the STI may

467   manipulate host behaviour in such a way that its interests are aligned with one sex. It has been

468   suggested that an STI that reduced female remating rate would be beneficial for males (Knell

469   and Webberley, 2004) because a male that infects his mate would reduce her remating rate and

470   ensure his own paternity. If the benefits of reducing sperm competition outweigh the costs of the

471   STI, males may be selected to increase persistence and consequently their likelihood of acquiring

472   an STI. Similarly, there is some evidence that infection by an STI increases oviposition rates in

473   the fall army worm moth, meaning that males could benefit from acquiring and transmitting an

474   STI (Simmons and Rogers, 1994). There are numerous ways males and females could evolve in

475   response to these changes in host mating behaviour. Overall, these examples suggest that a change

476   in the cost structure in the presence of an STI could affect the outcome of sexual conflict in natural

21

477   systems.

478   There are many well-known examples of STIs and sexual conflict but we are aware of no systems

479   where both are well studied. Evidence of sexual conflict and sexually transmitted diseases has

480   been reported in ungulates (conflict: Bro-Jørgensen 2010; STI: Lockhart et al. 1996), Drosophila

481   (conflict: e.g., Rice et al. 2006; STI: Knell and Webberley 2004), and the two-spot ladybird *Adalia*

482   *bipunctata* (conflict: Haddrill et al. 2013; STI: Webberley et al. 2002; Ryder et al. 2005). Studies

483   of sexually antagonistic traits in populations infected with a viability- or reproduction-reducing

484   STI should look for escalated or de-escalated trait values, respectively, compared to populations

485   where the STI is absent. Research on viability-reducing STIs should compare different populations

486   or closely related host species that experience different persistence, resistance, or mating costs.

487   Variation in the costs of sexually antagonistic host traits could arise between populations if, for

488   example, the persistence or resistance trait made the bearer more vulnerable to predation in an

489   open versus closed habitat (see Fricke et al., 2009, for a discussion of the dependence of sexually

490   antagonistic selection on environmental conditions). Higher mortality rates in one habitat should

491   select for higher virulence in an endemic viability-reducing STI. Additionally, we expect lower

492   disease prevalence in populations where male persistence is strongly selected against and there are

493   high costs of mating or low costs of resistance for females, i.e., low mating rate will limit STI

494   transmission.

495   We have shown that an STI has the potential to influence the outcome of sexually antagonistic

496   coevolution. Because STIs are ubiquitous in nature (Lockhart et al., 1996), they should co-occur

497   with sexual conflict often enough that it is worth considering how STIs change sexually antagonistic

498   selection. Furthermore, considering the full coevolutionary interaction has important implications

499   for both the susceptibility of a host population to invasion of a new STI and the level of virulence

500   expected to evolve.

Acknowledgements

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504 We would like to thank Samuel Alizon and Philip Greenspoon for comments on an earlier version

505 of the manuscript.

506 Supplemental Information

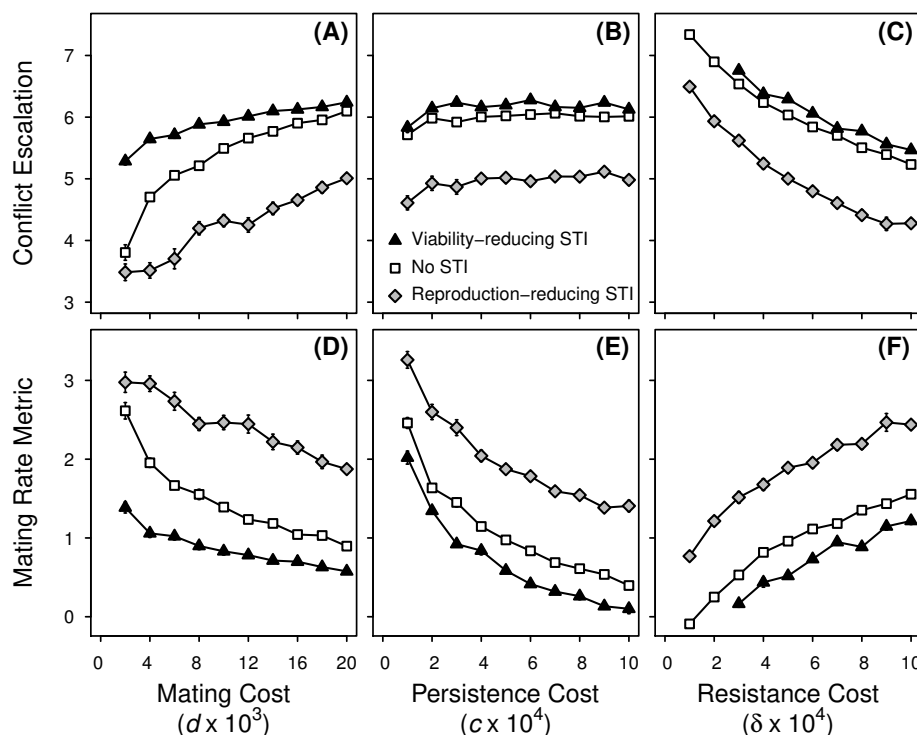## 507 Reproduction Costs for Sexually Antagonistic Traits

Figure S1: The outcome of coevolution between males and females in the absence of an STI and in the presence of two types of evolving STIs. Both sexes pay reproduction costs for expressing sexually antagonistic traits. Shown here are the degree of escalation in sexual conflict, measured as the average of y and x (A - C), and the difference between persistence, y, and resistance, x (D - F); values are at evolutionary equilibrium. The mating rate is an increasing function of the difference between male persistence and female resistance $(y - x)$; thus, we label this difference the 'mating rate metric'. Simulations with an evolving STI began at the STI-absent host trait values and the results for a reproduction-reducing STI (diamonds) and a viability-reducing STI (triangles) are a three-way evolutionary equilibrium with the STI at evolutionary equilibrium virulence v (see Fig. 4). Parameter values (unless shown otherwise): $\alpha = 10$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, with $c = 0.0005$, $\delta = 0.0005$, $d = 0.02$. Each point represents the average of 20 independent simulations +/- standard error. Simulations in which the disease went extinct are excluded from calculating the average. At very low resistance costs, a viability-reducing STI went extinct in 100% of simulations (see Fig. S2) and there is no data for the three-way evolutionary equilibrium.
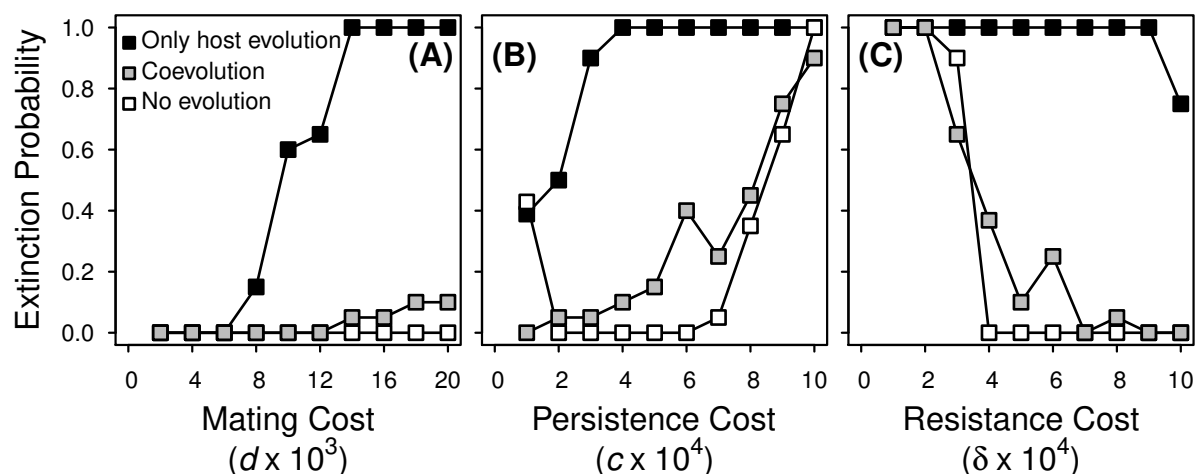
Figure S2: Fraction of simulation runs where a highly virulent viability-reducing STI was driven extinct in hosts paying reproduction costs. The parasite was introduced with $v = 0.8$ at the STI-absent male persistence and female resistance host trait values and neither host nor parasite evolved (open symbols), host traits evolved in the presence of a non-evolving parasite (black symbols), or hosts and STI both evolved (gray symbols). Parameter values (unless shown otherwise): $\alpha = 10$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, with $c = 0.0005$, $\delta = 0.0005$, and $d = 0.02$. The extinction probability was determined from 20 independent simulations.

Analytical Model

509 We use a system of differential equations to describe sexually antagonistic coevolution in a popula-

510 tion of haploid hosts (parameter definitions summarized in Table 1). Most parameters are the same

511 as the individual-based simulations with the exception of α, which captures the sexual encounter

512 rate as a fraction of the density-dependent interaction of males and females (instead of α being a

513 fixed number of males that a female encounters in the computer simulation model). The probability

514 of a female not mating is described by $\gamma[M] = (1 - \phi[u])^{\alpha}M$, where $\phi[u] = y - x$ (as above) and

515 M is the number of males in the host population.

516 The ecological dynamics are captured by the following system of equations:

$$\frac{dM}{dt} = \frac{1}{2}Fb(1 - \frac{M+F}{K})(1 - \gamma[M]) - (ce^y + \mu_m)M \tag{1a}$$

$$\frac{dF}{dt} = \frac{1}{2}Fb(1 - \frac{M+F}{K})(1 - \gamma[M]) - (d\alpha M\phi[u] + \delta e^x + \mu_f)F \tag{1b}$$

517 We find evolutionary stable (ESS) persistence and resistance, y^* and x^*, respectively, using se-

518 quential evolutionary invasion analyses for multiple traits. We plot the degree of conflict escalation,

519 $(y^* + x^*)/2$, and the mating rate metric, $u^* = y^* - x^*$, for varying costs in Fig. S3.

520 We introduce a sexually transmitted infection into the model by dividing the host popula-

521 tion into susceptible and infected classes. Susceptible individuals may contract the disease by

522 mating with infected individuals of the opposite sex. We model transmission of the STI as density-

523 dependent because it is reasonable to assume that the number of matings per capita will increase

524 with density in systems governed by sexual conflict (i.e., at high density, females encounter more

525 males and thus are likely to end up mating more total times holding x and y constant). To deter-

526 mine a female's full cost of mating, we must track the expected number of males a female mates

527 with and not just the fraction of her mates that were infected (as would be done under frequency-

528 dependent transmission). Density-dependent sexual disease transmission has been documented in

529 nature (for example in two-spot ladybird beetles, *Adalia bipunctata*, Ryder et al. 2005).

530 STI virulence, v, results in higher mortality of disease carriers. We assume a trade-off between
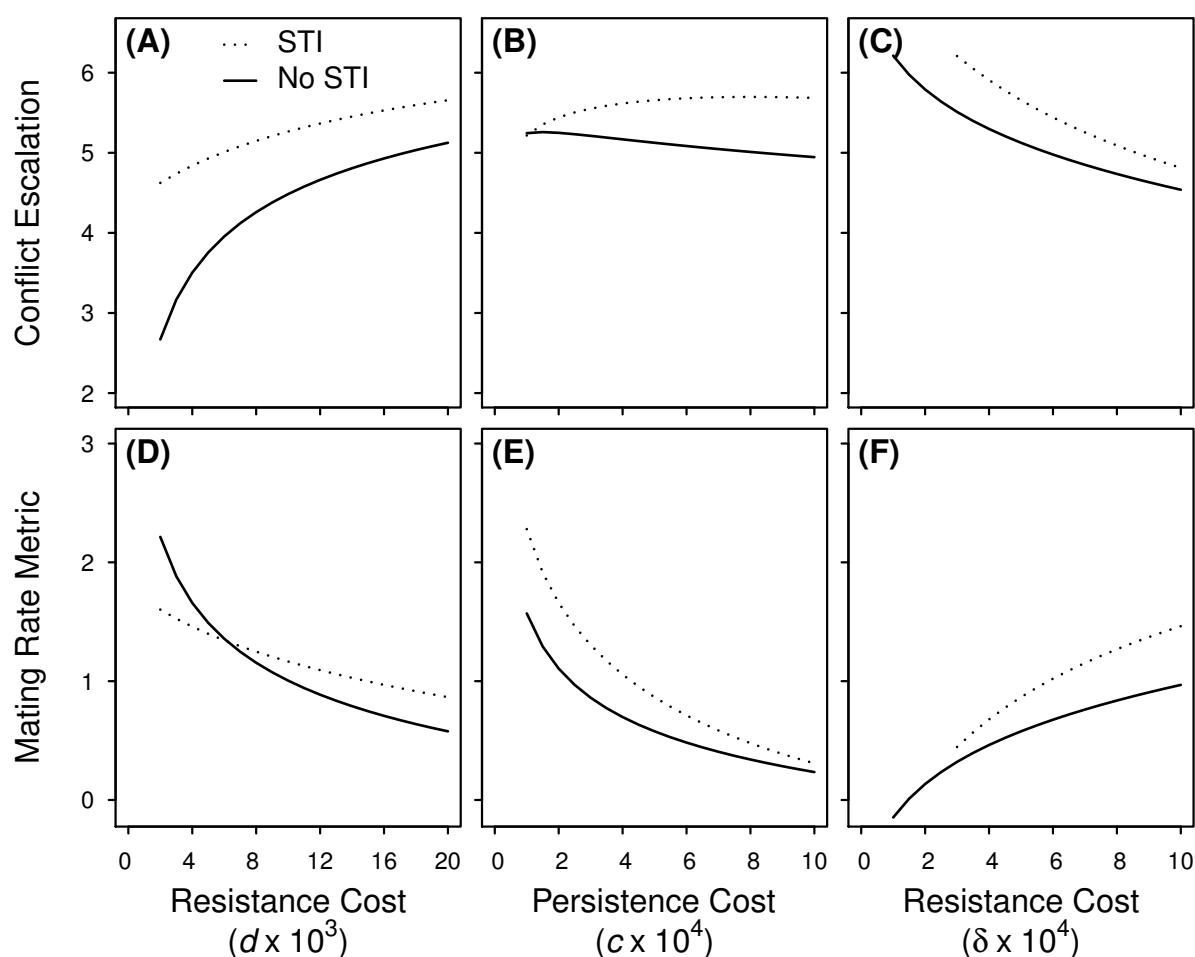
Figure S3: The outcome of coevolution between males and females in the absence of an STI and in the presence of a coevolving viability-reducing STI based on numerical solutions to the analytical model. Both sexes pay viability costs for expressing sexually antagonistic traits. Shown here are the degree of escalation in sexual conflict, measured as the average of y^* and x^* (A - C), and the difference between persistence, y^*, and resistance, x^* (D - F); values are evolutionarily stable strategies (ESS). The mating rate is an increasing function of the difference between male persistence and female resistance ($y^* - x^*$); thus, we label this difference the 'mating rate metric'. The results shown are a three-way co-ESS with an STI with ESS virulence v^* (see Fig. S4). Parameter values (unless shown otherwise): $\alpha = 0.03$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, with $c = 0.0005$, $\delta = 0.0005$, $d = 0.02$. At very low resistance costs, there was no co-ESS with the STI present.

531 transmission and virulence such that the transmission rate during mating is a saturating function of

532 virulence $\beta[v] = v/(w+v)$, where w determines the shape of the function (i.e., low values of w cause

533 the transmission benefits of increased virulence to saturate more quickly). The epidemiological

534 dynamics are described by the following set of differential equations:

$$\frac{dS_m}{dt} = \frac{1}{2}\psi[S_m, S_f, I_m, I_f] - \alpha I_f \phi[u]\beta[v]S_m - (ce^y + \mu_m)S_m \tag{2a}$$

$$\frac{dS_f}{dt} = \frac{1}{2}\psi[S_m, S_f, I_m, I_f] - \alpha I_m \phi[u] \, \beta[v]S_f - (d\alpha(S_m + I_m)\phi[u] + \delta e^x + \mu_f)S_f \tag{2b}$$

$$\frac{dI_m}{dt} = \alpha I_f \phi[u]\beta[v]S_m - (ce^y + \mu_m + v)I_m \tag{2c}$$

$$\frac{dI_f}{dt} = \alpha I_m \phi[u] \, \beta[v]S_f - (d\alpha(S_m + I_m)\phi[u] + \delta e^x + \mu_f + v)I_f \tag{2d}$$

535 where

$$\psi[S_m, S_f, I_m, I_f] = b(S_f + I_f)\left(1 - \frac{S_m + S_f + I_m + I_f}{K}\right)(1 - \gamma[S_m, I_m]) \tag{3}$$

536 Similar to the STI-absent model, we find ESS persistence (y^*), resistance (x^*), and virulence (v^*)

537 using sequential evolutionary invasion analyses for multiple traits. We compare the degree of host

538 conflict escalation and the mating rate metric to their respective values in the absence of an STI

539 (Fig. S3). Finally, we compare STI virulence in a non-evolving host population (at its STI-absent

540 ESS) to virulence in a coevolving host population (Fig. S4). Results are qualitatively similar to

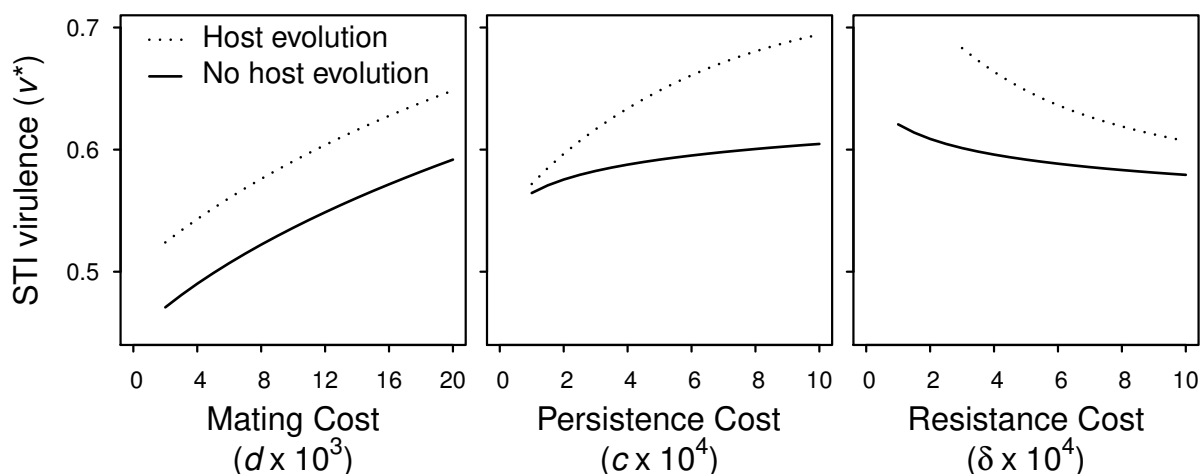541 those of the individual-based computer simulations.

28

Figure S4: Evolutionary stable virulence (v^*, ESS virulence) of a viability-reducing STI in a population where hosts do not evolve (open symbols) or evolve (closed symbols). In the absence of host evolution, male persistence and female resistance were treated as constant parameters and set at their STI-absent ESS values. Parameter values (unless shown otherwise): $\alpha = 0.03$, $\mu = 0.2$, $b = 4$, $K = 1000$, $w = 1$, with $c = 0.0005$, $\delta = 0.0005$, $d = 0.02$. At very low resistance costs, there was no co-ESS with the STI present.

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