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Coinfection accelerates transmission to new hosts despite no effects on virulence and parasite growth

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1 **Title:** Coinfection acelbrates transmission to new hosts despite no effects on virulence and parasite
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10

11 **Abstract**

12 One of the fundamental aims of ecological, epidemiological and evolutionary studies of host-parasite
13 interactions is to unravel which factors affect parasite virulence. Theory predicts that virulence and
14 transmission are correlated by a trade-off, as too much virulence is expected to hamper transmission
15 due to excessive host damage. Coinfections may affect each of these traits and/or their correlation.
16 Here, we used inbred lines of the spider-mite *Tetranychus urticae* to test how coinfection with *T.*
17 *evansi*, impacted virulence-transmission relationships, at different conspecific densities. The presence
18 of *T. evansi* on a shared host did not change the relationship between virulence (leaf damage) and the
19 number of transmitting stages (i.e., adult daughters). The relationship between these traits was
20 hump-shaped across densities, both in single and coinfections, which corresponds to a trade-off.
21 Moreover, transmission to adjacent hosts increased in coinfection, but only at low *T. urticae* densities.
22 Finally, we tested whether virulence and the number of daughters were correlated with measures of
23 transmission to adjacent hosts, in single and coinfections at different conspecific densities. Traits were
24 mostly independent, meaning interspecific competitors may increase transmission without affecting
25 virulence. Thus, coinfections may impact epidemiology and parasite trait evolution, but not
26 necessarily the virulence-transmission trade-off.

27

28 **Keywords:** trade-off hypothesis, coinfection, host-parasite interactions, multiple infections,
29 herbivorous arthropods, interspecific competitors .

30 **Introduction:**

31 Studies on host-parasite interactions, be it from an evolutionary, ecological or disease perspective,
32 generally evaluate the causes and consequences of parasite-induced fitness costs to hosts (i.e.
33 virulence) and the spread of parasites among hosts (transmission). Coinfections, i.e. the presence of
34 other parasites (strains or species) within the same host, are ubiquitous and a key factor affecting
35 parasite life-history traits (1-3). Experimental work shows that coinfections can both increase or
36 decrease within-host parasite growth (e.g. (4-7), often with consequences for virulence (4, 8, 9) and
37 transmission (10), which are often related. Indeed, the virulence-transmission trade-off hypothesis
38 posits that, despite virulence being a by-product of parasite growth, too high virulence leads to
39 excessive host damage, curtailing transmission (11, 12). Evidence for the existence of a trade-off
40 between these traits is limited (13), possibly due to environmental factors, such as host and/or
41 parasite demography, interactions with the host immune system or coinfection, changing the
42 selection environment and relationships between traits (1, 3, 12, 14, 15). Therefore, coinfection may
43 be a factor affecting traits involved in the trade-off and/or modulating the interaction among them (1,
44 16). However, most studies on coinfections focus on its effect on individual traits, not on their
45 relationship.

46 One important environmental factor that may affect the outcome of coinfections is the relative
47 density of each competitor in the within-host environment (5, 7, 17-19). Increasing densities of a
48 competitor may increasingly reduce parasite growth (7, 18). Alternatively, the impact of a competitor
49 may depend on parasite densities (5, 17, 19). For example, interspecific competition may only affect
50 traits at lower intraspecific densities (17). Interactions among parasites may also impact transmission-
51 related traits, independently of growth and virulence, such as triggering dispersal from hosts infected
52 with competitors or impacting whether a new host becomes infected. Indeed, certain parasites avoid
53 or choose a host, or a host tissue, depending on its infection status (20, 21). This means that multiple
54 parasites in the environment have the potential to impact parasite life-history ecology and evolution
55 at different scales, not restricted to the within-host environment.

56 A key aspect that may affect how parasite interactions in coinfections modify parasite traits is whether
57 they are genetically correlated. Indeed, if that is the case, then any genetic change in one trait driven
58 by the presence of competitors will affect the genetic value of other traits, which has major
59 consequences for the evolutionary trajectories of populations. In contrast, if the correlation is purely
60 environmental, then no direct evolutionary consequences are expected, but the ecological impact of
61 the parasite, such as parasite severity or epidemic onset, may be modified.

62 This study used inbred lines of the spider mite *Tetranychus urticae* to investigate how an interspecific
63 competitor, the closely related species *T. evansi*, impacts virulence, the number of adult daughters
64 produced and transmission to a new host patch as well as the potential correlations, genetic or
65 environmental, among these traits. In *T. urticae*, there is genetic variation for dispersal distance (22,
66 23), which here we refer to, and is the same as transmission, and for host use (24), which may be
67 correlated with virulence. Moreover, dispersal is a plastic trait, with individuals having higher dispersal
68 at elevated intraspecific densities (22) and in the presence of kin (25). Moreover, selection for higher
69 dispersal has been shown to be associated with higher diapause incidence and lower fecundity (23)
70 and dispersing individuals had smaller eggs (26) and fewer offspring surviving to adulthood when
71 laying eggs at higher densities (27). Further, in a recent companion study to this one conducted with
72 the same inbred lines, we found a positive genetic correlation between virulence and the number of
73 adult daughters produced when transmission was possible during the infectious period (28). Hence,
74 there are both genetic and environmental relationships between transmission and other life-history
75 traits. Finally, the outcome of competition between *T. evansi* and *T. urticae* can change due to
76 variation across populations (29) but also depending on the sequence of arrival, as *T. evansi* excludes
77 *T. urticae* except when the latter arrives first and occupies *T. evansi*'s preferred niche (30). In sum,
78 competitive interactions between these mite species are strong and strongly impacted by both genetic
79 and environmental factors. Therefore, the system composed of these two spider mite species is ideal
80 to address how the presence of a competitor affects virulence, parasite growth (number of adult
81 daughters) and transmission, as well as the potential (genetic) interactions among these traits.

82

83 **Materials and Methods**

84 Biological system

85 Spider mites are macroparasites of plants, including many economically important crops, with their
86 complete life cycle occurring on their host plant (31). Both *T. urticae* and *T. evansi* females lay eggs on
87 leaves, which take ~4 days to hatch. The juvenile stage comprises 1 nymph stage and 2 deuteronymph
88 stages, with adults emerging after approximately 14 days in our laboratory (25°C, 16:8 L: D cycle). All
89 stages feed by injecting their stylet into parenchyma cells and sucking out the cytoplasm, which leaves
90 chlorotic damage on the leaf surface, our measure of virulence (32). *T. urticae* is a generalist species,
91 feeding on more than 1000 different plant species (33), whereas *T. evansi* is a specialist species, mostly
92 feeding on *Solanaceae* plants (34). In natural systems, co-occurrence of different spider mite species
93 in the same geographical area is common, leading to co-infection of the same host plant (35).

94

95 Spider-mite populations

96 Inbred lines of *T. urticae* were created from an outbred population through 14 generations of sib
97 mating at the University of Lisbon (35). A subset of 15 inbred lines was transferred to the University
98 of Montpellier in January 2018 and maintained on bean leaves (*Phaseolus vulgaris*; variety Pongo) as
99 described in Godinho et al. 2023. The *T. evansi* population was originally collected in October 2010 in
100 the Alpes Maritimes (43.75313 N, 7.41977 E) on *Solanum nigrum*.

101

102 Prior to each experiment, cohorts of 40 mated female spider mites from each inbred line were
103 isolated on a bean patch (2-3 leaves placed together). These females were allowed to lay eggs for 48h.
104 Fourteen days later, the mated daughters of these females, of approximately the same age, were used
105 in the experiments. The same procedure was used to create cohorts of *T. evansi*. All spider-mite
106 populations, inbred lines and cohorts used in these experiments were maintained on bean leaves
107 (*Phaseolus vulgaris*; variety Pongo) placed on water saturated cotton wool, in small plastic boxes (255
108 mm length x 183 mm width x 77 mm height), at 25°C with a 16:8 L: D cycle, at 60% relative humidity.
109 Not all inbred lines are represented in each experiment due to too few individuals available at the
110 start of the experiment (N = between 14 to 16 lines).

111

112 *Experiment 1. Impact of interspecific competitors on within-host traits*

113 Females of each *T. urticae* inbred line were randomly assigned to one 'intraspecific density' treatment
114 (5, 10 and 20 females), with or without 'interspecific competition' (10 *T. evansi* females) (Figure 1a).
115 In all treatments, females were placed on a 2 x 2 cm bean leaf patch placed on wet cotton wool in
116 plastic boxes. There were 3 to 13 replicates for each inbred line per treatment combination
117 (intraspecific density x interspecific competition) distributed across 3 blocks. Variation in the number
118 of replicates per line arose due to differences in the number of adult females produced in the
119 synchronised cohorts. All females were allowed to feed and lay eggs on their leaf patches for 4 days.
120 After this period, females were killed, the number of eggs was counted and a photograph of each
121 patch was taken using a Canon EOS 70D camera. The damage inflicted by these adult female spider
122 mites on each host patch, used as a measure of virulence, was determined using ImageJ and Ilastik
123 1.3, as described in (28). Succinctly, the background from each photo was removed in ImageJ, then we
124 used Ilastik to distinguish damaged area from healthy leaf and finally the damaged area was calculated
125 via the colour contrast between damaged and undamaged leaf tissue in ImageJ. Because some leaf
126 veins were incorrectly assigned as damage by Ilastik, uninfested bean leaf patches were left in the
127 experimental boxes for the same period of time and photographed; these control patches were used

128 to establish an average baseline level of falsely assigned damage, which was subtracted from each
129 measurement to estimate the actual damage (hereafter: 'damage'). After a period of 14 days, the
130 female offspring surviving on each patch were counted. Only females were counted because the males
131 of both species are not easily distinguishable, females are the main dispersers in these species and the
132 number of females produced correlates with transmission (28). The data on damage inflicted and
133 production of adult females, for the "intraspecific density" treatments in the absence of *T. evansi* are
134 published elsewhere (28).

135

136 *Experiment 2. Impact of interspecific competition on transmission*

137 We measured differences in dispersal traits (transmission) for the different *T. urticae* inbred lines
138 assigned to the same 'intraspecific density' and 'interspecific competition' treatments as in
139 Experiment 1. Adult *T. urticae* females were placed in groups of 5, 10 or 20 on a 2cm² bean leaf patch
140 on wet cotton wool alone or with 10 *T. evansi* females. This first host patch was connected, in a row,
141 to 2 other bean patches via 3 x 1 cm Parafilm bridges from day 1 of the experiment (Figure 1b). This
142 experimental setup was replicated across several boxes. Females were allowed to feed and disperse
143 across patches, and the number of mites on each patch was counted on days 1, 2, 3, 6, and 9 after the
144 beginning of the experiment. There were 3 to 9 replicates for each inbred line per treatment
145 combination (intraspecific density x interspecific competition) distributed across 2 blocks. Variation in
146 the number of replicates is due to the number of offspring emerging as adult females among lines.

147

148 Statistical analysis

149 Analyses were performed using the software JMP SAS version 17 and SAS OnDemand for Academics
150 (36).

151 *Impact of interspecific competitors on within-host traits*

152 In Experiment 1 General Linear Mixed Models (GLMM) were used to investigate how intraspecific
153 density, interspecific competition and their interaction affected virulence and the number of female
154 offspring becoming adult. These analyses included intraspecific density as a covariate and interspecific
155 competition as a fixed factor. Next, we used a GLMM to test whether the relationship between
156 virulence and the production of adult daughters (transmitting stages) changed with interspecific
157 competition and if this effect varied across intraspecific densities. In this model, the number of adult
158 daughters remained the response variable, with the linear, quadratic and saturating terms for

159 virulence, intraspecific density, interspecific competition and their interactions, up to and including 3-
160 way interactions, included as explanatory variables. Full models were simplified by removing non-
161 significant terms in a stepwise fashion. Inbred line and block were included in these models as random
162 factors.

163

164 *Impact of interspecific competitors on transmission*

165 In Experiment 2, different measures were taken to assess dispersal across host patches. We used a
166 dispersal score to evaluate the spread of mites across the 3 host patch system. This was calculated
167 each day as the (number of mites on host patch 2 + the number of mites on host patch 3*2)/total
168 number of mites (22). This score weights greater distances more, as they represent higher
169 dispersal propensity, and corrects for the differences in the initial density of mites (22). The dispersal
170 score was analysed in a GLMM with interspecific competition as a fixed factor, intraspecific density
171 and time as covariates, and their interactions. The linear and quadratic terms for time were included
172 in the model to account for saturation in transmission through time. As there was a significant
173 interaction between competition and density, we separately tested the effect of interspecific
174 competition on the dispersal score at each of the different densities. We also investigated, in separate
175 GLMMs, including intraspecific density as a covariate and interspecific competition as a fixed factor,
176 how interspecific competition affected the time for mites to reach, and the maximum number of *T.*
177 *urticae* on host patches 2 and 3. Full models included interactions between explanatory variables that
178 were simplified by removing non-significant terms in a stepwise fashion. All the above models included
179 inbred line and block as random factors.

180

181 *Genetic variance for within-host traits and transmission*

182 Broad-sense heritability, $H^2 = \frac{Var(G)}{Var(G)+Var(E)}$ (37), for each trait in each experiment was determined
183 by extracting the proportion of total variance in models explained by inbred line (among inbred line
184 variance) by re-running models for within-host and transmission related life-history traits including all
185 terms as random (competition, line, density, block and patch nested within block for dispersal score)
186 to obtain all variance components. Note that these models did not include interaction terms. Traits
187 were divided by the total number of adult females placed on a patch (e.g. traits per capita) since they
188 are passed from parents to offspring at the level of an individual, not in groups of individuals. The
189 significance of each model was assessed by comparing the Akaike's Information Criterion (AIC) of a
190 model including inbred line with a model excluding it. A significant H^2 indicates that trait variance is

191 significantly explained by differences in the additive or dominance genetic variance across
192 individuals and/or by differences in maternal effects.

193

194 *Correlations between within-host traits and transmission*

195 We assessed genetic correlations between traits measured in the 2 experimental set-ups separately
196 for each combination of density and competition treatments. If correlations between traits are
197 genetic, this can provide predictions for how they might evolve, given that selection on one trait will
198 also affect the expression of the other. We only included traits for which there was significant genetic
199 variance among inbred lines (Table S1).

200 First, we reported the Pearson's correlation coefficient across mean trait values for each of the inbred
201 lines. Next, we extracted the standard errors for each correlation coefficient and associated p-values
202 from a PROC MIXED COVTEST model as described in (38) using SAS Studio. As measures were taken in
203 different experimental set-ups we bootstrapped (with replacement) the mean value for each inbred line
204 at each density and interspecific competition treatment 20 times and randomly paired the different
205 values. Trait values were standardised across all lines for each density and competition treatment,
206 such that each variable had a mean of zero and standard deviation of one. This was so that values for
207 pairs of traits were of a similar scale as required for the PROC MIXED COVTEST in SAS Studio.

208 All p-values < 0.05 were corrected for multiple testing (within each pair of traits) using the
209 Bonferroni correction method.

210

211 **Results**

212 *Impact of interspecific competitors on within-host traits*

213 Interspecific competitors had no effect on the virulence of *T. urticae* at any intraspecific density
214 (interspecific competition; $F_{1, 637} = 0.34$, $p = 0.5609$, interspecific competition*intraspecific density; $F_{1, 636} = 0.36$, $p = 0.5567$) nor on the number of adult daughters (interspecific competition; $F_{1, 637} = 1.09$, $p = 0.2959$, interspecific competition*intraspecific density; $F_{1, 636} = 0.14$, $p = 0.7107$; Figures 2
217 and S1; Table S2). When virulence was included as a covariate, the presence of interspecific
218 competitors did not change the relationship between virulence and the production of adult
219 daughters, i.e. transmitting stages (interspecific competition*virulence; $F_{1, 635} = 2.17$, $p = 0.1416$,
220 interspecific competition*virulence²; $F_{1, 637} = 0.03$, $p = 0.8532$, Figure 2; Table S1).

221

222 A significant interaction between intraspecific density and virulence ($F_{1, 643} = 16.07$, $p < 0.0001$)
223 showed that the shape of the relationship between virulence and the production of adult daughters,
224 i.e. transmitting stages, changed at different densities (positive at low densities, no relationship at
225 intermediate densities, and negative at high densities). This was corroborated with a significant
226 quadratic term for virulence in a second model investigating factors affecting the number of adult
227 daughters (virulence; $F_{1, 648} = 12.45$, $p = 0.0004$, and virulence²; $F_{1, 643} = 9.36$, $p = 0.0023$; Figure 2;
228 Table S2). However, as these results were not influenced by interspecific competition they are not
229 discussed further here, as they are presented elsewhere (28).

230

231 *Impact of interspecific competitors on transmission*

232 The dispersal score was affected by both interspecific competition ($F_{1, 447} = 20.68$, $p < 0.0001$) and
233 intraspecific density ($F_{1, 1177} = 16.69$, $p < 0.0001$), with a significant interaction between these two
234 factors ($F_{1, 445} = 7.68$, $p = 0.0058$; Figure 3, Table S3). Models investigating the effect of interspecific
235 competition separately at each density showed that there was only a significant effect of competition
236 in the low density treatment (Table S4). This meant that *T. urticae* females were more likely to leave
237 the first host patch in the presence of interaspecific competitors only at low densities. The interaction
238 between *T. urticae* density and the quadratic term for time was also significant ($F_{1, 1844} = 23.57$, $p <$
239 0.0001), with values of the dispersal score saturating through time for patches in the intermediate
240 and high density treatments (Figure 3, Table S3). Note, the dispersal score captured time to arrive on,
241 and maximum numbers on patches 2 and 3 (Figure S2 and Table S5). We observed an effect of
242 intraspecific density on all these underlying traits and of interspecific competition on the time to arrive
243 to patch 2 and 3, the latter depending on the density of intraspecific competitors, as there was a
244 significant interaction between the two factors (Table S5).

245

246 *Genetic variance for within-host traits and transmission*

247 For the within-host traits, we found low but significant broad-sense heritability for the number of adult
248 daughters ($H^2 = 0.057$) and virulence ($H^2 = 0.060$). For measures of transmission, inbred line explained
249 a significant portion of the variance (H^2) for time to reach host patches 2 ($H^2 = 0.062$) and 3 ($H^2 = 0.036$)
250 and the dispersal score ($H^2 = 0.039$), but not the maximum number of individuals on host patch 2 or 3
251 (Table S1).

252

253 *Correlations between within host traits and transmission*

254 We explored the genetic relationships between traits related with transmission between hosts (i.e.,
255 day arriving on and maximum number on each patch) and traits measured in the within host-
256 environment (i.e., virulence and number of adult daughters) for traits that showed significant among
257 line variation (virulence, number of adult daughters, day arriving on patches 2 and 3; Table S1), giving
258 a total of 24 correlations. Of these, 6 models did not converge. Of the remaining 18 models, only 1
259 was significant (Table 1), showing a negative correlation between virulence and the time to arrive on
260 host patch 2 at high *T. urticae* densities in the presence of interspecific competition (Table 1; Figure
261 S3). Collectively these results show that virulence and the number of adult daughters measured in the
262 within-host environment are mostly independent of traits measuring transmission between hosts.

263

264 **Discussion**

265 In this study, we found that interspecific competition did not modify virulence, the production of adult
266 daughters (i.e., transmitting stages) or the relationship between these traits. However, the presence
267 of interspecific competitors increased transmission of *T. urticae* to new host patches at low
268 intraspecific densities, which may be a mechanism to escape interspecific competition (at higher
269 intraspecific densities this effect may be masked by more intense intraspecific competition).
270 Differences in transmission between hosts were mostly genetically unrelated to measures of virulence
271 or the number of adult daughters produced in the within-host environment. Therefore, selection is
272 expected to act on each trait independently, that is, selection for virulence or the number of adult
273 daughters is mostly unlinked to that on traits that foster early transmission. This means that selection
274 for faster spread across host patches is not necessarily associated with higher virulence.

275

276 *Impact of interspecific competitors on within-host traits*

277 We found no significant impact of *T. evansi* on virulence or the production of adult daughters in *T.*
278 *urticae*. This may stem from the fact that *T. evansi* is a poor competitor on bean plants. These results
279 may have been very different had this experiment been done on a host plant to which *T. evansi* is
280 better adapted. For instance, *T. evansi* is generally found to be the superior competitor on tomato
281 plants, often excluding *T. urticae* ((19, 39) but see (29, 30)). As interspecific competition did not modify
282 these traits, it also did not lead to changes in their interaction. Our results contrast with other studies
283 that show that the impact of interspecific competition on parasite growth and virulence can change
284 in response to the relative densities of each parasite in coinfection (5, 7, 18). Instead, as previously

285 found in the absence of competitors (28), we here find a positive relationship between virulence and
286 transmission at low densities, no relationship at intermediate densities and a negative relationship at
287 high densities. This result is because of intense within-host intraspecific competition among juvenile
288 *T. urticae* developing on the host patch: at higher densities, despite many more eggs being laid, fewer
289 offspring become adults (28).

290

291 *Impact of interspecific competitors on transmission*

292 Interspecific competition changed how *T. urticae* moved among host patches, but this depended on
293 the intensity of intraspecific competition. At low intraspecific densities, the presence of *T. evansi*
294 increased transmission of *T. urticae* females to the second and third host patch sooner and increased
295 the density of mites on these host patches. At intermediate and high *T. urticae* densities, however,
296 interspecific competitors did not affect transmission. This is probably because the density of *T. urticae*
297 was so high that there was no additional effect of interspecific competition. It could be that *T. urticae*
298 females just respond to the total number of spider mites on the patch. However, this is unlikely since
299 the dispersal score in the absence of interspecific competition did not change across intraspecific
300 densities (Figure 3).

301 The finding that interspecific competition causes *T. urticae* to move to a new host faster means that
302 coinfection may be an important driver of epidemic spread. Coinfection can cause individuals to
303 become superspreaders, when an infected host is responsible for a disproportionate number of
304 transmission events (40). Here, we only measured the number of spider mites moving from one host
305 patch to another, which is not the same as the number of new hosts infected. Nevertheless, it gives
306 an idea of the number of transmission stages leaving an infected host, which is a measure of infection
307 potential, similar to parasite shedding (10, 41-43). These different effects of parasite intraspecific
308 densities and coinfection could be used to predict parasite spread in natural populations and to
309 manage or control epidemics, for instance by identifying (and isolating or treating) the most infectious
310 individuals (44).

311 Whereas some studies have shown that the intensity of interspecific parasite competition modulates
312 the effect of intraspecific competition within the host (5, 7, 17, 19, 30), the effect on transmission is
313 less clear. From the dispersal literature, it is clear that intra- and interspecific competition can interact
314 to shape the movement of organisms at different scales (45). However, parasite studies are rare (e.g.
315 (10) for an example with different parasite strains) and do not measure other traits (e.g., virulence),
316 which are key to evaluate the impact of interspecific competition on disease epidemics. Thus, it is as

317 yet unclear how the relative impact of inter and intraspecific competition among parasites affects
318 transmission. This is especially true because we here found that interspecific competition affects
319 transmission differently depending on intraspecific density. New infections are often seeded by low
320 densities of mites (a few adult females), but during the course of infection densities become very high
321 until the host is completely overexploited (46). Whether coinfections with *T. evansi* will foster the
322 spread of *T. urticae* through host populations may thus depend on how often coinfections involve
323 early or late stages of infection. One possible scenario is that *T. evansi* promoting *T. urticae*
324 transmission will result in the latter arriving first to a host plant, which will give a headstart to *T. urticae*
325 and, as a consequence facilitate coexistence due to priority effects (30). In turn, this headstart may
326 result in *T. urticae* reaching higher densities before the arrival of *T. evansi*, which may reduce the
327 impact of *T. evansi* on transmission. This would then diminish the effect of interspecific competitors
328 on transmission, thus creating a negative feedback loop, such that this effect would only be detected
329 transiently.

330

331 *Correlations between within-host traits and transmission*

332 We showed that within-host traits are mostly genetically independent of measures of transmission
333 between hosts. Indeed, there was no genetic relationship among traits in 17/18 possible tests across
334 treatment combinations, despite these traits being genetically determined. These results contrast
335 with previous a study in which we found a genetic correlation between adult daughters (i.e
336 transmitting stages) and transmission (28). However, in that case, transmission was measured from
337 hosts where virulence was inflicted, and, thus, highly dependent on within-host processes that lead
338 to the production of transmitting stages. Here, by obtaining independent measures for virulence and
339 transmission, we did not find that these traits are genetically linked and, by removing the connection
340 mediated by within-host processes, we also do not observe an effect of intra and interspecific
341 competition on this relationship. Only one genetic correlation was found to be significant, that
342 between virulence and the time to arrive on host patch 2. Moreover, the sign of this correlation
343 hinged on interspecific competition: it was negative in the presence of *T. evansi*, with no correlation
344 between these traits in the absence of competition. This means that more virulent lines are
345 responding to the presence of *T. evansi* as a trigger to leave the first host patch sooner.

346

347 How such direct measures of transmission, independent of within-host processes (e. g. virulence),
348 actually scale up and affect the spread of *T. urticae* across a population of potential hosts is not
349 straightforward and may well depend on the presence and relative densities of interspecific
350 competitors (20). Moreover, different life-history strategies could co-exist in a parasite population,

351 some maximising fitness within hosts and others maximising the spread across hosts. If genetic
352 variation for within-host traits and transmission are uncoupled, then contrasting selection pressures
353 in each environment may maintain variation for both across scales.

354

355 *Conclusion*

356 Our results show that interspecific competition may increase the rate of parasite spread across hosts
357 and that this trait is genetically independent of traits measured in the within-host environment, i.e.,
358 virulence and the production of adult daughters (transmitting stages). Therefore, parasites selected
359 for higher virulence locally are not those necessarily favoured in travelling wave epidemics, or those
360 that spread far to seed infections in new host populations. In the future it would be interesting to
361 explore how the traits measured in this study relate to those affecting the infection of a greater
362 quantity of hosts or parasite spread over longer distances.

363

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375 SM.

376

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482

483 Figure legends

484 Figure 1: Experimental set up in a) Experiment 1, in which adult female *T. urticae* (black and white
485 spider mites) were placed in groups of 5, 10 or 20 on a 2cm² bean leaf patch with or without 10 *T.*
486 *evansi* (red spider mites), b) Experiment 2, in which adult female *T. urticae* were placed in groups of
487 5, 10 or 20 on a 3 x 2cm² bean leaf patches with or without 10 *T. evansi*. The figure only depicts the
488 low density treatment.

489

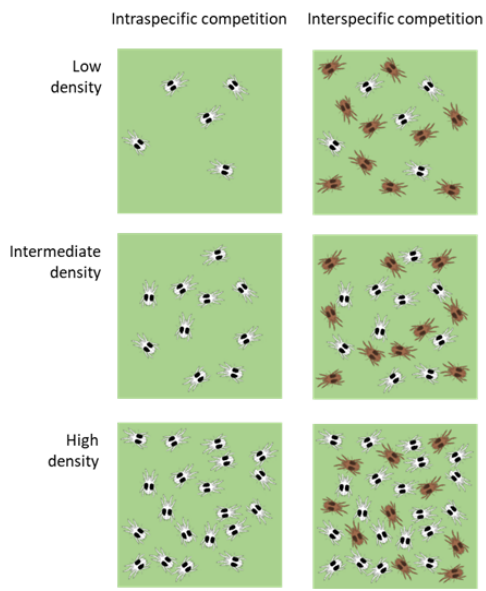
490 Figure 2: Relationship between virulence and the production of adult daughters (i.e., transmitting
491 stages) in experiment 1 at low, intermediate and high intraspecific density in a) the absence (blue)
492 and b) presence (red) of 10 *T. evansi* interspecific competitors. Values are given at low (5 females;
493 lighter colour solid line and circles), intermediate (10 females; medium colour, dotted line, triangles)
494 and high (20 females; darker colour, dashed line, squares) densities. Each dot is the mean value for an
495 inbred line at each density (+ SE). The effect of intraspecific density creating a humped-shape
496 relationship between virulence and adult daughters is not affected by coinfection.

497

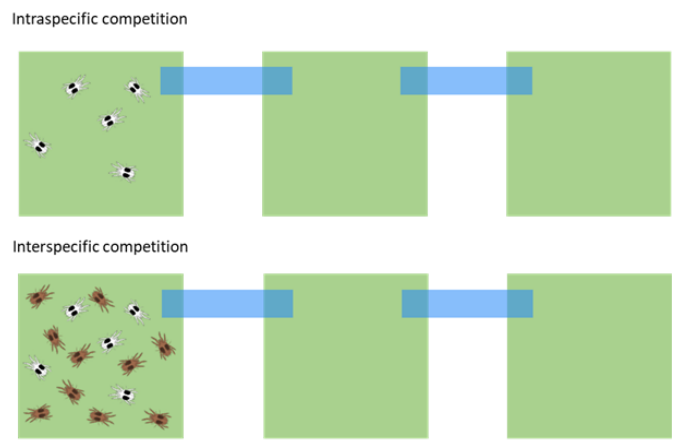
498 Figure 3: Mean dispersal score through time (\pm standard error) measured in Experiment 2 at each of
499 the different *T. urticae* densities in the presence (red) or absence (blue) of *T. evansi*. The presence of
500 the interspecific competitor leads to increased transmission to patches 2 and 3 at lower intraspecific
501 densities.

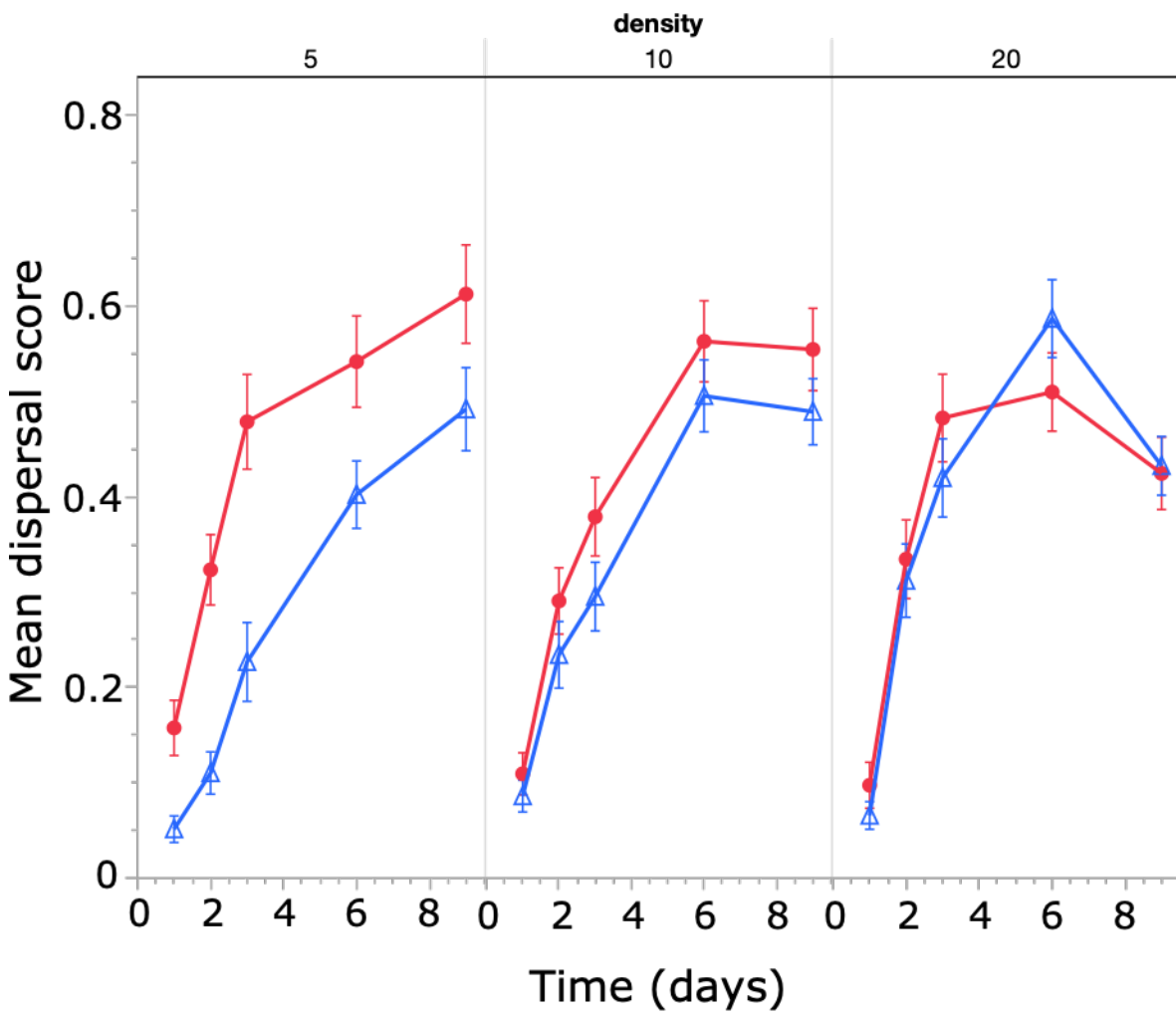
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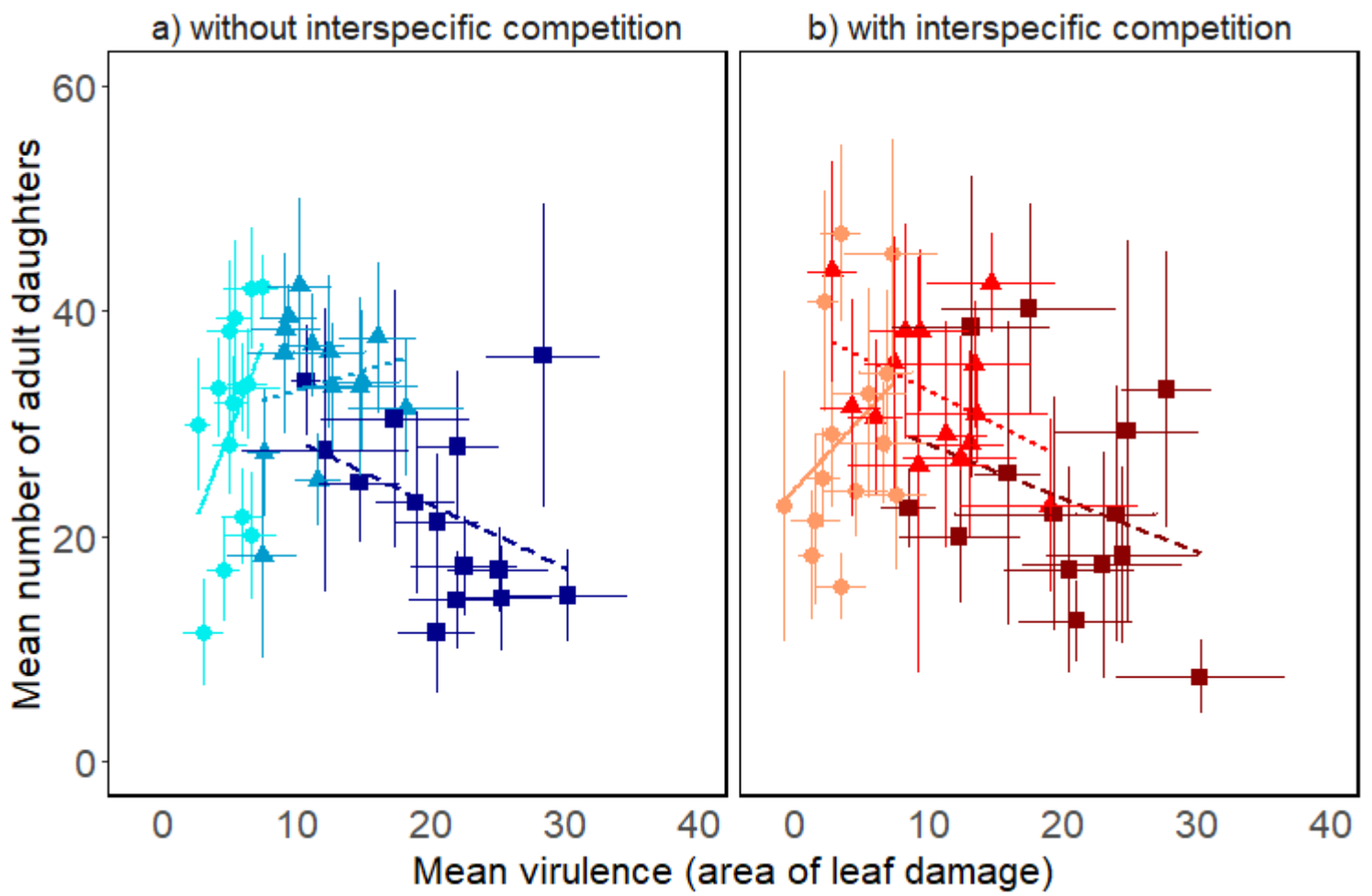
a)



b)







503 **Table 1:** Summary of genetic correlations between transmission-related traits at each of the different intraspecific densities, in the presence or absence of
504 interspecific competition. The Pearson's correlation across mean trait values for the different inbred lines is presented for each pair of traits \pm the standard
505 error calculated from the PROC MIXED COVTEST on the bootstrapped data. The χ^2 and log likelihood test comparing models with and without the genetic
506 correlation are also shown. All values of $p < 0.05$ were corrected using Bonferroni corrections (counting 6 tests per pair of traits). Significant correlations
507 are shown in bold.

508

Trait measuring transmission between hosts	Trait in within-host environment	Density 5		Density 10		Density 20	
		No competition	Competition	No competition	Competition	No competition	Competition
Day arriving on host patch 2	Virulence	$r_g = -0.10 \pm 0.28$ SE $\chi^2 = 0.6, p = 0.4386$	$r_g = 0.16 \pm 0.29$ SE $\chi^2 = 1.1, p = 0.2943$	$r_g = 0.11 \pm 0.28$ SE $\chi^2 = 0.3, p = 0.5839$	$r_g = -0.19 \pm 0.27$ SE $\chi^2 = 0.4, p = 0.5271$	$r_g = 0.49 \pm 0.24$ SE $\chi^2 = 2.7, p = 0.1003$	$r_g = -0.67 \pm 0.14$ SE $\chi^2 = 8.1, p = 0.0264$
	No. adult daughters	$r_g = -0.22 \pm 0.27$ SE $\chi^2 = 6, p = 0.0858$	$r_g = -0.05 \pm 0.29$ SE $\chi^2 = 0.1, p = 0.7518$	$r_g = 0.22 \pm 0.03$ SE $\chi^2 = 1.3, p = 0.2542$	$r_g = -0.07 \pm 0.29$ SE $\chi^2 = 0, p = 1.0$	$r_g = -0.14 \pm 0.31$ SE $\chi^2 = 0, p = 1.0$	$r_g = 0.40 \pm 0.27$, $\chi^2 = 1.7, p = 0.1923$
Day arriving on host patch 3	Virulence	$r_g = -0.01 \pm 0.28$ SE $\chi^2 = 0.5, p = 0.4795$	$r_g = 0.19 \pm 0.28$ SE $\chi^2 = 0.3, p = 0.5839$	$r_g = 0.65 \pm 0.17$ SE $\chi^2 = 6.00, p = 0.0858$	Model does not converge	Model does not converge	Model does not converge
	No. adult daughters	$r_g = -0.07 \pm 0.03$ SE $\chi^2 = 0.3, p = 0.5839$	$r_g = 0.26 \pm 0.23$ SE $\chi^2 = 0.5, p = 0.4795$	$r_g = 0.003 \pm 0.18$ SE $\chi^2 = 6, p = 0.0858$	Model does not converge	Model does not converge	Model does not converge

509