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Parasitism and host dispersal plasticity in an aquatic model system

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1 **Parasitism and host dispersal plasticity in an aquatic model system**

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3

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17

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22 **Author Contributions section**

23 OK, GZ, LN, NZ and EAF conceived the study. OK, GZ, LN and NZ designed the experiments. GZ, LN, NZ, CGB
24 and OK performed the experimental work. GZ, EAF, OK, GP and NZ performed the statistical analysis. All
25 authors interpreted the results and contributed to the writing of the manuscript.

26 **Conflict of Interest statement**

27 The authors have no conflict of interest to declare.

28

29 **Abstract**

30 Dispersal is a central determinant of spatial dynamics in communities and ecosystems, and various ecological
31 factors can shape the evolution of constitutive and plastic dispersal behaviours. One important driver of
32 dispersal plasticity is the biotic environment. Parasites, for example, influence the internal condition of
33 infected hosts and define external patch quality. Thus state-dependent dispersal may be determined by
34 infection status and context-dependent dispersal by the abundance of infected hosts in the population. A
35 prerequisite for such dispersal plasticity to evolve is a genetic basis on which natural selection can act. Using
36 interconnected microcosms, we investigated dispersal in experimental populations of the freshwater protist
37 *Paramecium caudatum* in response to the bacterial parasite *Holospora undulata*. For a collection of 20
38 natural host strains, we found substantial variation in constitutive dispersal, and to a lesser degree in
39 dispersal plasticity. First, infection tended to increase or decrease dispersal relative to uninfected controls,
40 depending on strain identity, potentially indicative of state-dependent dispersal plasticity. Infection
41 additionally decreased host swimming speed compared to the uninfected counterparts. Second, for certain
42 strains, there was a weak negative association between dispersal and infection prevalence, such that
43 uninfected hosts tended to disperse less when infection was more frequent in the population, indicating
44 context-dependent dispersal plasticity. Future experiments may test whether the observed differences in
45 dispersal plasticity are sufficiently strong to react to natural selection. The evolution of dispersal plasticity as
46 a strategy to mitigate parasite effects spatially may have important implications for epidemiological
47 dynamics.

48

49 **Keywords:**

50 Condition-dependent dispersal, dispersal plasticity, eco-evolution, epidemiology, *Holospora undulata*, host-
51 parasite interactions, *Paramecium caudatum*, reaction norms, spatial dynamics

52

53

54 Introduction

55 Dispersal, broadly defined as the movement of individuals with consequences for gene flow, is a key life-
56 history trait (Bonte & Doherty, 2017) driving metapopulation and metacommunity dynamics as well as the
57 geographic distribution of species (Hanski, 1999). In recent years, the study of dispersal and dispersal
58 syndromes have received increasing interest (Clobert *et al.*, 2012; Stevens *et al.*, 2014), as landscapes are
59 seeing large-scale environmental alterations and fragmentation, rendering dispersal crucial to potentially
60 mitigate these changes (Parmesan & Yohe, 2003; Cote *et al.*, 2017). Although dispersal is often considered a
61 constitutive trait, plastic dispersal behaviour represents a flexible alternative, responding to changes in the
62 internal condition of an individual (state-dependent dispersal) and to external environmental factors
63 (context-dependent dispersal) (Clobert *et al.*, 2009). State-dependent dispersal has been associated with
64 variation in factors such as body size, the developmental stage or sex of individuals (Bowler & Benton, 2005).
65 In contrast, context-dependent dispersal decisions may be based on cues that provide information on biotic
66 and abiotic patch properties, such as food availability, population density, or kin competition (see Ronce,
67 2007 and references therein).

68

69 In communities, dispersal plasticity may be advantageous in mitigating adverse interactions with other
70 species (Fronhofer *et al.*, 2015a). Parasites are particularly interesting in this respect: they are ubiquitous and
71 impose strong selection pressures, and potentially drive the evolution of both state-dependent and context-
72 dependent dispersal of their hosts (Iritani & Iwasa, 2014; Iritani, 2015; Narayanan *et al.*, 2020; Deshpande *et al.*,
73 2021). Empirical studies have investigated aspects of parasite-related dispersal (see below), but still little
74 is known about the genetic basis of this kind of dispersal plasticity and its adaptive significance.

75

76 State-dependent dispersal may relate to morphological or physiological changes induced by parasites. The
77 exploitation of host resources might decrease general activity levels, and thereby reduce movement and
78 dispersal. Such negative effects have been documented for various organisms (Binning *et al.*, 2017; Nørgaard
79 *et al.*, 2019; Baines *et al.*, 2020), even though it is not necessarily a general rule (Nelson *et al.*, 2015; Csata *et al.*,
80 2017). While in many examples the observed effects may represent side effects, theory has identified
81 conditions under which increased (but also decreased) dispersal when infected is adaptive, namely under kin
82 selection (Iritani & Iwasa, 2014; Iritani, 2015) or when infection can be lost during dispersal (Shaw & Binning,
83 2016; Daversa *et al.*, 2017). Indeed, increased dispersal of infected hosts is not uncommon (Suhonen *et al.*,
84 2010; Brown *et al.*, 2016), although it may also be the result of parasite manipulation (Lion *et al.*, 2006;
85 Martini *et al.*, 2015).

86

87 Natural enemies may also produce context-dependent dispersal, as a means to reduce immediate predation
88 or infection risk. For example, herbivores or predators can induce the production of specific dispersal morphs

89 (Weisser *et al.*, 1999; de la Pena *et al.*, 2011). A recent multi-species study further showed that chemical
90 predator-related cues increase dispersal probability (Fronhofer *et al.*, 2018). Such cues may also exist in host-
91 parasite systems, where infection-avoidance behaviour is well known (Behringer *et al.*, 2006; Curtis, 2014).
92 Recent theory shows that hosts may indeed evolve reaction norms, with dispersal being a function of the
93 parasite infection prevalence (Deshpande *et al.*, 2021). To date, few if any empirical studies have tested for
94 the existence of such plastic population-level responses (French & Travis, 2001).

95

96 Adaptive phenotypic plasticity is a powerful solution in many situations (Chevin *et al.*, 2013; Stamp &
97 Hadfield, 2020), and just like constitutive traits, it has a genetic basis on which selection can act (Pigliucci,
98 2005; Garland & Kelly, 2006; Laitinen & Nikoloski, 2019). Dispersal-related traits have such a genetic basis
99 (Saastamoinen *et al.*, 2018) and constitutive dispersal can evolve rapidly in a parasite context (Koskella *et al.*,
100 2011; Zilio *et al.*, 2020). However, the genetics and evolution of dispersal plasticity is less well studied. In fact,
101 how plastic dispersal varies between different genotypes under parasite challenge is rarely evaluated in
102 empirical studies (Suhonen *et al.*, 2010; Fellous *et al.*, 2011), or the genetic diversity is treated as a random
103 effect (Csata *et al.*, 2017). Moreover, the number of genotypes evaluated is usually small, making it difficult
104 to draw general conclusions (Leggett *et al.*, 2013).

105

106 Here, using interconnected microcosms, we tested a collection of 20 natural strains of *Paramecium caudatum*
107 for dispersal in the presence and absence of the bacterial parasite *Holospira undulata*. Previous work in this
108 system had shown that infection reduces dispersal for a small number of strains (Fellous *et al.*, 2011;
109 Nørgaard *et al.*, 2021). The first objective of the present study was to test whether this negative effect was
110 general, or whether strains varied in infection-state dependent dispersal. Second, we tested for genetic
111 variation in context-dependent dispersal by comparing the dispersal of uninfected hosts over a range of
112 infection prevalences that had naturally established in the experimental populations. We found that parasite
113 reduced or increased dispersal levels depending on strain identity, indicating a state-dependent plastic
114 response of the infected hosts, but no general negative effect of infection. Furthermore, increasing infection
115 prevalence tended to reduce host dispersal for certain strains, suggesting context-dependent dispersal
116 plasticity of uninfected hosts. Such genetic variation in dispersal plasticity may provide the raw material for
117 parasite-mediated selection, in natural settings or for the purpose of experimental evolution.

118

119 **Materials and methods**

120 **Study system**

121 *Paramecium caudatum* is a freshwater filter-feeding protist from stagnant waters of the Northern
122 hemisphere (Wichterman, 2012). Like all ciliates, paramecia have a macronucleus for somatic gene
123 expression and a germ-line micronucleus, used for sexual reproduction. The micronucleus can be infected by

124 *Holospira undulata*, a gram-negative alpha-proteobacterium (Fokin, 2004). Infectious spores are released
125 for horizontal transmission after host cell division or upon host death. Infectious spores are immobile and
126 therefore rely on host movement or water current for their own dispersal. Vertical transmission occurs when
127 hosts divide mitotically. Infection reduces *P. caudatum* division and survival (Restif & Kaltz, 2006) and also
128 host dispersal (Fellous *et al.*, 2011; Nørgaard *et al.*, 2021).

129

130 **Experimental setup**

131 Preparation of replicates. We established mass cultures for a collection of 20 genetically distinct strains of *P.*
132 *caudatum* from different geographical regions (provided by S. Krenek, TU Dresden, Germany; Table S1,
133 Supplementary Information). Distributed over two experimental blocks, 6 infected replicate cultures were
134 established for each strain (20 strains x 2 blocks x 3 replicates = 120 replicates). Inocula were prepared from
135 a mix of infected stock cultures in the lab, all originating from a single isolate of *H. undulata* established in
136 2001 (Dohra *et al.*, 2013). Following standard protocols for the extraction of infectious spores (e.g., Nørgaard
137 *et al.*, 2021) we used c. 10^4 spores to inoculate samples of c. $3\text{-}5 \times 10^3$ host cells in 1.5 mL per assay replicate.
138 Four days after inoculation, when infections have established, we expanded the cultures by regular addition
139 of lettuce medium (supplemented with the food bacterium *Serratia marcescens*), until a volume of 50 mL
140 was reached. In the same way, we set up three uninfected control populations per strain, giving a total of
141 180 experimental cultures. After three weeks, prior to the dispersal assay, population size (mean: $190 \text{ mL}^{-1} \pm$
142 9 SE ; 95% range [172; 208]) and infection prevalence (mean: $26.8 \% \pm 2.1$; 95% range [3.1; 90.7]) had settled
143 naturally in each experimental replicate.

144

145 Dispersal assay. We assayed the dispersal of infected and uninfected replicates in dispersal arenas, as
146 described in Nørgaard *et al.* (2021). A dispersal arena consisted of three 50-mL Falcon tubes, linearly
147 connected by 5-cm long silicon tubing (inner diameter: 0.8 cm). The 3-patch system was filled with 75 mL of
148 medium to establish connections. Then the connections were blocked with clamps and 20 mL of a given
149 replicate culture added into the middle tube. The lateral tubes received 20 mL of *Paramecium*-free medium.
150 Connections were then opened, and the *Paramecium* allowed to disperse to the lateral tubes for 3h. After
151 blocking the connections, we counted the individuals in samples from the middle tube (500- μL) and from the
152 combined lateral tubes (3 mL) to estimate the number of non-dispersing and dispersing individuals
153 (dissecting microscope, 40x). From the same samples, we also made lacto-aceto-orcein fixations (Görtz &
154 Wiemann, 1989) and determined the infection status (infected / uninfected) of up to 30 dispersing and non-
155 dispersing individuals, respectively (light microscope, phase contrast, 1000x). From the cell counts and the
156 infection status data, we estimated the population density and infection prevalence in the middle tube at
157 the beginning of the assay. From the same data, we also estimated the proportion of infected and uninfected
158 dispersers for each replicate, referred to as per-3h “dispersal rate” or dispersal, hereafter.

159 In addition, to investigate a potential link between dispersal and movement (Banerji *et al.*, 2015; Pennekamp
160 *et al.*, 2019), we assayed swimming behaviour. For each strain, 1 infected and 1 uninfected individual were
161 isolated from arbitrarily selected assay replicates, and allowed to replicate in a 2-mL plastic tubes for 8 days.
162 For the resulting 40 monoclonal cultures (20 strains x 2 infection status) we placed 200- μ L samples (10-20
163 individuals) on a microscope slide and recorded individual movement trajectories under a Perflex Pro 10
164 stereomicroscope, using a Perflex SC38800 camera (15 frames per second; duration: 10 s; total magnification:
165 10x). For each sample, average swimming speed (μ m/s) and swimming tortuosity (standard deviation of the
166 turning angle distribution, describing the extent of swimming trajectory change) were determined using
167 video analysis (“BEMOVI” package; Pennekamp *et al.*, 2015).

168

169 **Statistical analysis**

170 Statistical analyses were conducted in R, v. 3.6.3 (R Core Team, 2020) using Bayesian models with the ‘rstan’
171 (version 2.19.3) and ‘rethinking’ (version 2.0.1) packages (McElreath 2020).

172 For state-dependent dispersal, we compared the dispersal of the infected individuals (in infected replicates)
173 with the dispersal in the uninfected control replicates. We fitted four models, from the intercept to the full
174 interaction model, using a binomial regression with logit link function (chain length: warmup = 20,000
175 iterations, chain = 40,000 iterations). In the full model, the explanatory factors were infection status (infected
176 or uninfected control), *Paramecium* strain identity, and the strain x status interaction. Experimental block
177 only explained a negligible fraction of the dispersal variation (preliminary analysis, not shown) and was
178 omitted from all further analyses. We fitted the models choosing vaguely informative priors; the intercepts
179 and slope parameters followed a normal distribution with mean -2 and standard deviation 3 for the first, and
180 mean 0 and standard deviation 1.75 for the latter. To account for overdispersion we included an observation-
181 level random effect. The mean and standard deviation of the observation-level hyperprior followed a normal
182 and half-normal distribution respectively, with mean 0 and standard deviation 1. The four state-dependent
183 models were compared and ranked using the Watanabe-Akaike information criterion, WAIC (Watanabe,
184 2010), a generalized version of the Akaike information criterion (Gelman *et al.*, 2014). The posterior
185 predictions of the models were then averaged based on WAIC weights, and the relative importance (RI) of
186 the explanatory variables was calculated as the sum of the respective WAIC model weights in which that
187 variable was included. Due to loss of replicates, low population density, and/or very low levels of infection,
188 159 replicates (from 20 strains) of the 180 initial replicates were available for this analysis.

189

190 For context-dependent dispersal, we analysed the dispersal of uninfected *Paramecium* in infected assay
191 replicates. We fitted 6 models, from the intercept to the full interaction model, using the same binomial
192 regression with logit link function, chain lengths and prior specifications as above. The explanatory factors of
193 the full model (varying intercept and slope) were infection prevalence, strain identity and the strain x

194 infection prevalence interaction. The posterior predictions were averaged and ranked, and the RI calculated
195 based on WAIC model weights as described above. For this analysis, 99 assay replicates (from 19 strains) of
196 the initially 120 inoculated replicates were available.

197

198 We used similar analyses to test whether swimming speed and tortuosity varied as a function of strain
199 identity and infection status. We standardized the response variable and fitted four models (from the
200 intercept to the additive model, see Table S2 and S3, Supplementary Information) using a linear regression
201 (chain length: warmup = 20,000 iterations, chain = 40,000 iterations) with an exponentially distributed prior
202 (rate = 1) for standard deviation. As for the dispersal analysis, the parameter priors were vaguely informative;
203 the intercept and slope parameters followed a normal distribution with mean 0 and standard deviation 2.
204 We averaged and ranked the posterior predictions, and we obtained RI based on WAIC model weights. We
205 further tested for correlations between these two swimming traits and mean strain dispersal, for infected
206 and uninfected *Paramecium* (chain length: warmup = 2,000 iterations, chain = 10,000 iterations). Due to
207 missing data, only 17 of the 20 strains were used for these analyses.

208

209 **Results**

210 **State-dependent dispersal**

211 Our analysis revealed substantial variation in constitutive dispersal among the 20 *P. caudatum* strains
212 (relative importance, RI, of strain identity = 0.85; Table 1), ranging from 1% (95% compatibility Interval [0.001;
213 0.135]) to 41% ([0.02; 0.80]) of the individuals moving from the central to the lateral tubes (Fig. 1A).

214 Our models provided limited evidence for state-dependent dispersal plasticity. Infection status (RI = 0.57)
215 was retained in the best model fit (lowest WAIC; Model 3 in Table 1), indicating a general trend of infection
216 to increase host dispersal. Even though the signal of the strain x infection status interaction (RI = 0.22) was
217 only weak, patterns in Fig. 1B indicate that effects of infection varied with strain identity: several strains
218 indeed dispersed more when infected (Fig. 1B right side of panel), but in at least half of the strains, infection
219 had little effect or decreased host dispersal.

220

221 **Context-dependent dispersal**

222 As in the above analysis, we found substantial genotypic variation in overall constitutive levels of dispersal
223 for uninfected *Paramecium* (RI of strain identity = 0.80; Table 2). The best model (model 4 in Table 2) included
224 an effect of infection prevalence (RI = 0.67), and thus context-dependent dispersal. Namely, uninfected
225 individuals tended to disperse less at higher parasite infection prevalence in the population (Fig. 2): such
226 negative dispersal-prevalence relationships were predicted for all but one strain (negative median slope
227 values; Fig. 2B). To some degree, however, the strength of this relationship varied between strains (RI of
228 infection prevalence x strain interaction = 0.21). As shown in Fig. 2B, distributions of predicted slopes show

229 considerable variation and for the majority of strains there is considerable overlap with 0. Only a small
230 number of strains (e.g., C139, C116, C083) show clearly negative slopes (Fig. 2B).

231

232 **Swimming behaviour**

233 The analysis of standardized swimming speed revealed strong effects of strain identity (RI = 0.9; Table S2)
234 and infection status (RI = 1; Table S2). Namely, standardized swimming speed of uninfected *Paramecium*
235 (median = 0.57, 95% CI [-0.64; 2.34]) was generally higher than that of infected ones (median = -1.20, 95% CI
236 [-1.63; -0.77]), corresponding to a difference of almost 40% (median = 0.39, 95% CI [0.10; 0.68]; Fig. S1A-B).
237 Swimming tortuosity was not affected by strain and weakly affected by infection status (RI strain = 0; RI status
238 = 0.28; Table S3). Neither swimming speed (uninfected: $r = 0.08$, 95% CI [-0.39; 0.52]; infected: $r = 0.07$, 95%
239 CI [-0.41; 0.54]) nor swimming tortuosity (uninfected: $r = 0.15$, 95% CI [-0.29; 0.56]; infected: $r = -0.10$, 95%
240 CI [-0.55; 0.39]) were strongly correlated with dispersal.

241

242 **Discussion**

243 Dispersal affects epidemiology and host-parasite (co)evolution in metapopulations (Lion & Gandon, 2015;
244 Parratt *et al.*, 2016), but how dispersal itself evolves due to antagonistic species interactions is less well
245 known (Poethke *et al.*, 2010; Drown *et al.*, 2013; Deshpande *et al.*, 2021). Here we focused on dispersal
246 plasticity in response to parasitism, which may evolve as a means to reduce infection risk of the dispersing
247 individuals and/or their relatives (Iritani & Iwasa, 2014; Iritani, 2015; Deshpande *et al.*, 2021). Our study takes
248 a first step towards an understanding of population-level processes, by measuring dispersal of infected and
249 uninfected hosts in experimental microcosms and by exploring the genetic variation in plasticity for a
250 collection of host strains. Overall, signals of dispersal plasticity were weak. Both infection status and infection
251 prevalence modified dispersal to some degree, with at least some strains showing indications of state-
252 dependent dispersal (i.e., when infected) and/or context-dependent dispersal (i.e., in response to infection
253 prevalence).

254

255 **State-dependent plasticity: the dispersal of infected hosts**

256 In previous studies, infection by *H. undulata* reduced dispersal in *P. caudatum* for a small set of strains
257 (Fellous *et al.*, 2011; Nørgaard *et al.*, 2021). Here we used strains from a worldwide collection (Table S1) and
258 find the entire range of trends, from negative or no impact of infection to even positive effects on host
259 dispersal (Fig. 1). Reduced host dispersal may be explained by general negative effects of infection, through
260 the energetic demand of an immune response, the diversion of host resources by the parasite or direct
261 physical damage (Mideo, 2009). Indeed, *H. undulata* consumes nuclear proteins and nucleotides
262 (Garushyants *et al.*, 2018) and also causes massive interior swelling of the infected micronucleus, which
263 would explain the clear and pervasive reduction in swimming speed observed in the complementary

264 experiment (Fig. S1A-B). However, dispersal reductions were far from being universal, suggesting that the
265 amount of host damage differs between genotypes. Differential fitness effects (virulence) and variation in
266 resistance are known for this system (Restif & Kaltz, 2006), indicating the strong potential for genotypic-
267 specificity in the responses to this parasite.

268

269 Moreover, it should be noted that the absence of a difference between infected and uninfected dispersal
270 does not necessarily mean the absence of plasticity. Infected hosts may compensate parasite damage by re-
271 allocating resources to maintain vital functions, such as foraging and feeding activity, and this may lead to a
272 net-zero effect of infection on dispersal. Interestingly, some of our strains even seemed to 'overcompensate'
273 and dispersed more when infected. Such a positive state-dependent dispersal may be selectively favoured in
274 a metapopulation because it can reduce kin competition and kin infection (Iritani & Iwasa, 2014; Iritani, 2015;
275 Deshpande *et al.*, 2021). However, increased host dispersal may equally well reflect parasite manipulation,
276 enhancing its dispersal to novel infection sites (Kamo & Boots, 2006; Lion *et al.*, 2006; Martini *et al.*, 2015).

277

278 The main purpose here was to quantify the (variation in) population-level effects of infection on dispersal.
279 More work is needed to better understand the links between parasite action, host movement and dispersal.
280 This concern, for example the relationship between parasite load, virulence and dispersal. Furthermore,
281 unlike in other protists (Pennekamp *et al.*, 2019), swimming speed was not a good predictor of dispersal.
282 Other aspects of swimming behaviour (Ricci, 1989) may be more relevant in our system. Namely,
283 *Paramecium* show a characteristic vertical distribution (Fels *et al.*, 2008) relating to food and oxygen
284 availability (Wichterman, 2012). Parasites are known to affect the position of hosts in the water column
285 (Cezilly *et al.*, 2000; Fels *et al.*, 2004), and this may directly influence the probability of infected individuals
286 finding the dispersal corridors in our microcosms.

287

288 **Context-dependent plasticity: the dispersal of uninfected hosts**

289 Predator chemical signals induce dispersal in various organisms, including *P. caudatum* (Fronhofer *et al.*
290 2018). We tested for a similar parasite effect in our microcosm populations, by measuring the dispersal of
291 uninfected hosts at different infection prevalences, with the assumption that higher prevalence equals a
292 stronger signal of 'parasite presence'. Unlike in the predator-cues study, we found little evidence for a
293 positive dispersal-inducing effect. Dispersal decreased at higher infection prevalence, at least for certain
294 strains. Interestingly, Deshpande *et al.*'s model (2021) predicts the evolution of such negative prevalence-
295 dependent dispersal, as the result of complex spatio-temporal variations in eco-evolutionary processes. We
296 do not know the evolutionary history of the strains, but our results suggest a possible genetic basis of context-
297 dependent dispersal in this system and hence genetic variation that might be seen by natural selection.

298

299 Our experimental approach of using naturally established infection prevalence may not have produced
300 strong enough signal variation for all strains. This could be remedied via more artificial designs, by mixing of
301 infected and uninfected individuals to establish well-defined gradients. Infected cultures or inocula may also
302 be filtered to specifically test for chemical cues (see Fronhofer *et al.*, 2018). Finally, we made the simplifying
303 assumption of linear dispersal reaction norms. However, dispersal responses may well follow non-linear
304 rules, e.g., if there are signal thresholds (Fronhofer *et al.*, 2015b), as observed for other traits (Morel-Journel
305 *et al.*, 2020) and predicted by Deshpande *et al.* (2021). Tests for non-linear relationships would require a
306 much finer resolution (i.e., more replication) on the signal axis.

307

308 **Conditions for plasticity selection: outlook**

309 The heritability of phenotypic plasticity of morphological or behavioural traits is generally lower than their
310 constitutive heritability (Scheiner, 1993; Stirling *et al.*, 2002). In line with this, we find much less among-strain
311 differentiation for parasite-related dispersal plasticity than for constitutive dispersal, suggesting a weaker
312 potential for responding to selection. However, the available genetic variation alone does not determine the
313 relative importance of phenotypic plasticity in shaping evolutionary trajectories (Stamp & Hadfield, 2020).
314 Phenotypic plasticity is generally favoured in variable, but nonetheless predictable environments (Leung *et al.*,
315 2020). In a parasite context, dispersal plasticity evolution may thus depend on the spatio-temporal
316 predictability of parasite encounter rates across a metapopulation (Deshpande *et al.*, 2021). Additional
317 factors are parasite virulence, the cost of dispersal (or its advantage if parasite release is possible during
318 dispersal), or correlations with other traits (Iritani & Iwasa, 2014). For example, a recent experiment with the
319 protist *Tetrahymena* revealed few genetic constraints on the concurrent evolution of plasticity across various
320 traits (Morel-Journel *et al.*, 2020). Indeed, state- and context-dependent dispersal might also evolve
321 simultaneously in the presence of parasites, even though not necessarily in a correlated fashion (Deshpande
322 *et al.*, 2021). Our data indicate no genetic correlation between state- and context-dependent plasticity ($r = -$
323 0.11 , 95% CI $[-0.55; 0.36]$; based on strain averages), suggesting that independent responses to selection are
324 possible, as shown in the model.

325 Our study represents one of the first accounts of the naturally existing genetic variation for state-dependent
326 and context-dependent dispersal plasticity in relation to parasites. The signals of plasticity are weak and there
327 are many open questions regarding the mechanistic and physiological basis of trait expression or information
328 use. Nonetheless, in microbial systems such as ours, the observed variation opens promising avenues for
329 future experiments. In microcosm landscapes, allowing the free interplay between dispersal and
330 epidemiological processes, we can assess how dispersal plasticity affects parasite spread at the
331 metapopulation level. Over longer time spans, we can also explore dispersal evolution and test evolutionary
332 predictions on dispersal plasticity and its adaptive role in host-parasite interactions.

333 **Data availability statement**

334 The experimental data will be made available upon potential acceptance (via Dryad/Figshare repository).

335

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495 **Tables**

496

497 **Table 1.** Different statistical models and parameters included for the analysis and model averaging of the state-
 498 dependent dispersal. The rows represent the different models (the best model is highlighted in bold) and the columns
 499 the factors included in each model with the corresponding WAIC, standard error of the WAIC and WAIC weights. The RI
 500 row shows the relative importance of the explanatory variables.

501

| | Strain | Status | Strain * Status | WAIC | SE | WAIC weight |
|----------------|----------|----------|-----------------|---------------|--------------|-------------|
| Model 1 | | | | 1165 | 7.36 | 0.15 |
| Model 2 | X | | | 1163.8 | 27.76 | 0.27 |
| Model 3 | X | X | | 1163.3 | 27.67 | 0.35 |
| Model 4 | X | X | X | 1164.3 | 7.68 | 0.22 |
| RI | 0.85 | 0.57 | 0.22 | | | |

502

503 **Table 2.** Statistical models and parameters for the analysis and model averaging of the context-dependent dispersal.
 504 Each row represents a different model, the best model is highlighted in bold and the last row indicates the relative
 505 importance (RI) of the explanatory variables. The columns are the variables included in the six models with the
 506 corresponding WAIC, standard error of the WAIC and WAIC weights.

507

| | Strain | Prevalence | Strain * Prevalence | WAIC | SE | WAIC weight |
|----------------|----------|------------|---------------------|--------------|--------------|-------------|
| Model 1 | | | | 746.4 | 17.97 | 0.10 |
| Model 2 | X | | | 744.6 | 18.62 | 0.23 |
| Model 3 | | X | | 746.4 | 17.93 | 0.10 |
| Model 4 | X | X | | 744.2 | 18.77 | 0.28 |
| Model 5 | X | X | | 746.7 | 18.16 | 0.08 |
| Model 6 | X | X | X | 744.8 | 18.61 | 0.21 |
| RI | 0.80 | 0.67 | 0.21 | | | |

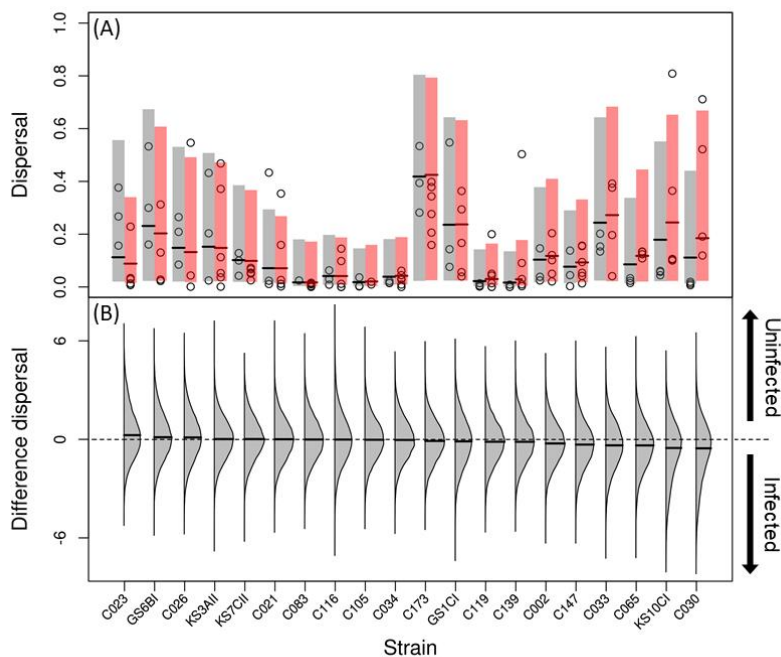
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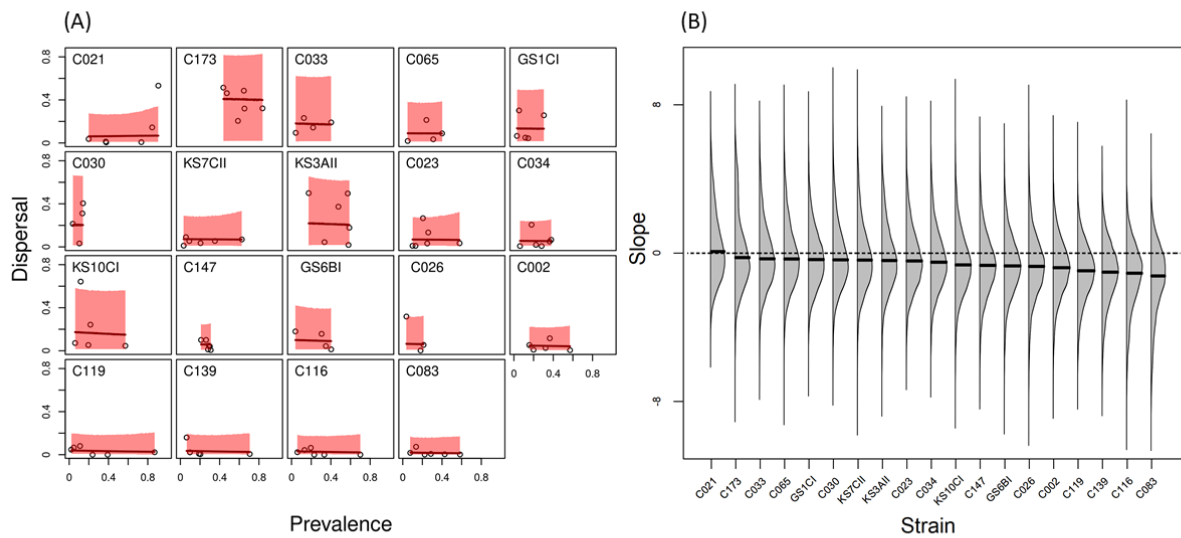
512 **Figures**



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514 **Figure 1.** State-dependent dispersal of 20 *Paramecium caudatum* strains, as a function of infection status (uninfected /
515 uninfected with *Holospira undulata*). (A) Shaded bars and thick lines represent the 95% compatibility interval and the
516 median of the averaged model predictions of the posterior distributions. Strains are ordered according to the difference
517 between uninfected (grey) and infected (red) dispersal. Each circle represents an experimental replicate. (B) Difference
518 between uninfected and infected averaged model posterior predictions for each strain (expressed in logits), the thick
519 black line represents the median of the difference distribution. Distributions shifted below zero (dashed grey line)
520 indicates higher dispersal in the infected (pointing-down arrow) compared to the uninfected (pointing-up arrow)
521 treatment.

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524 **Figure 2.** Context-dependent dispersal of 19 uninfected *Paramecium caudatum* strains, as a function of parasite
525 (*Holospora undulata*) infection prevalence in the microcosm population. (A) Each panel represents a strain, and each
526 circle an experimental replicate; the red shaded area and thick red lines are the 95% compatibility interval and median
527 of the averaged model of the posterior distributions. (B) Averaging of the posterior distributions of the slope parameter
528 calculated in logit (model 3-6, Table 2) with the thick black lines showing the median. Positive or negative slopes
529 distributions (above or below zero, dashed grey line), indicate a higher or lower dispersal in response to increasing
530 frequency of infected hosts.

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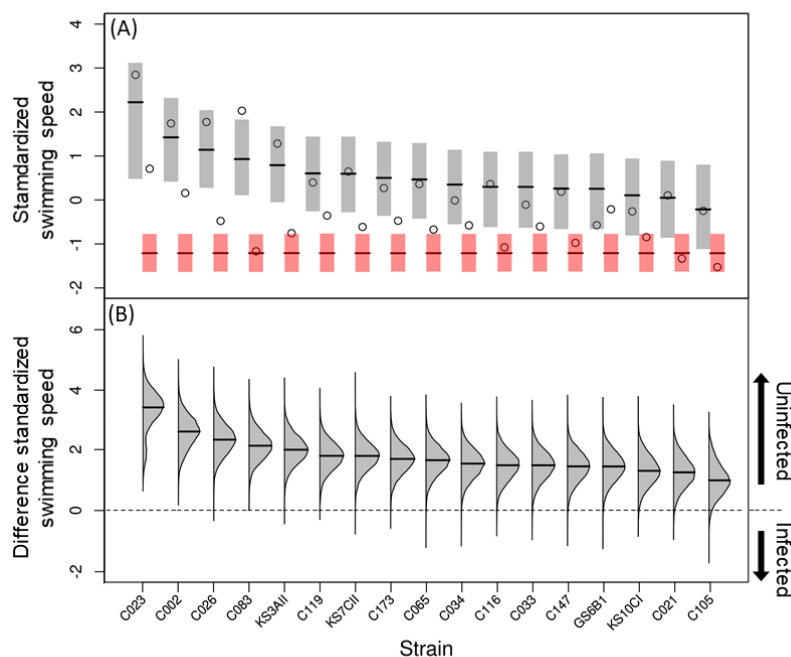
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547 Supplementary Information



548

549 **Figure S1.** Standardized swimming speed of 17 *Paramecium caudatum*, as a function of infection status (uninfected /
550 infected with *Holospira undulata*). (A) Strains are ordered according to the difference between uninfected (grey) and
551 infected (red) dispersal. Each circle represents an experimental replicate. Shaded bars and thick lines are the 95%
552 compatibility interval and median of the averaged model predictions of the posterior distributions, and each circle
553 represents the measured data of swimming speed per strain. (B) The difference in swimming speed between uninfected
554 and infected averaged model posterior predictions for each strain. The thick black lines are the median of the difference
555 distribution. Distributions shifted above zero (dashed grey line) indicates higher swimming speed in the uninfected
556 treatment (pointing-up arrow) compared to the infected treatment (pointing-down arrow).

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571 **Table S1.** Host strain identity, location of origin and GPS coordinates, (*) indicates approximate location.

| Strain | Location code | Origin | GPS coordinates |
|---------------|----------------------|----------------|----------------------------|
| C002 | Hainberger See | Germany | 51.035688, 12.285028 |
| C021 | Erzgebirge E1 | Germany | 50.647032,13.256963 |
| C023 | Plön K 2 | Germany | 54.15021, 10.440307 |
| C026 | Fokin 1 UBR 42 | USA, Louisiana | 53.131996, 13.107747 |
| C030 | Österreich_Lahnalp | Austria | 47.695948, 12.218064 (*) |
| C033 | Peking 1_C3 | China | 39.128165, 117.185083 (*) |
| C034 | Plön K 2 | Germany | 54.15021, 10.440307 |
| C065 | SWE 17.1 | Sweden | 60.105747, 15.966911 |
| C083 | USBL-511 | USA, Indiana | 39.069861, -86.414361 |
| C105 | Sp 10C | Spain | 39.548852, -1.502887 |
| C116 | Frankreich 10-2.1 | France | 43.430297, 6.126616 |
| C119 | Peru | Peru | -12.046184, -77.040842 (*) |
| C139 | My43c3d | Japan | 38.480973, 141.372414 (*) |
| C147 | KNZ5414 | Japan | 36.519469, 136.709415 |
| C173 | Greece 10.1 | Greece | 40.805947, 21.983306 |
| GS1CI | Globsowsee | Germany | 53.128590, 13.118740 |
| GS6BI | Globsowsee | Germany | 53.128590, 13.118740 |
| KS10CI | Kochsee | Germany | 53.131996, 13.107747 |
| KS3AII | Kochsee | Germany | 53.131996, 13.107747 |
| KS7CII | Kochsee | Germany | 53.131996, 13.107747 |

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585 **Table S2.** Statistical models and parameters for the analysis and model averaging of the swimming speed. Each row
 586 shows a different model, from the intercept to the additive model. The best model is highlighted in bold and the last
 587 row indicates the relative importance (RI) of the strain and status effect. The columns are the explanatory variables
 588 included in the four models with the corresponding WAIC, standard error of the WAIC and WAIC weights.
 589

| | Strain | Status | WAIC | SE | WAIC weight |
|----------------|------------|------------|-------------|-------------|-------------|
| Model 1 | | | 100.0 | 10.45 | 0.0 |
| Model 2 | X | | 114.1 | 5.84 | 0.0 |
| Model 3 | | X | 83.7 | 10.29 | 0.1 |
| Model 4 | X | X | 79.4 | 7.56 | 0.9 |
| RI | 0.9 | 1.0 | | | |

590

591 **Table S3.** Models and parameters for the analysis and model averaging of the swimming tortuosity. Each row
 592 corresponds to a different model used for the analysis, with the best model is highlighted in bold. The last row shows
 593 the relative importance (RI) of the explanatory variables. The columns are the variables of the models with the
 594 corresponding WAIC, standard error of the WAIC and WAIC weights.
 595

| | Strain | Status | WAIC | SE | WAIC weight |
|----------------|-------------|-------------|-------------|-------------|-------------|
| Model 1 | | | 99.1 | 6.52 | 0.72 |
| Model 2 | X | | 119.4 | 5.89 | 0.00 |
| Model 3 | | X | 101.0 | 6.39 | 0.28 |
| Model 4 | X | X | 121.4 | 5.86 | 0.00 |
| RI | 0.00 | 0.28 | | | |

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