



<b>Title</b>	Parasite infection induces size-dependent host dispersal: consequences for parasite persistence
<b>Author(s)</b>	Terui, Akira; Ooue, Keita; Urabe, Hirokazu; Nakamura, Futoshi
<b>Citation</b>	Proceedings of the royal society b-biological sciences, 284(1866), 20171491 <a href="https://doi.org/10.1098/rspb.2017.1491">https://doi.org/10.1098/rspb.2017.1491</a>
<b>Issue Date</b>	2017-11-15
<b>Doc URL</b>	<a href="http://hdl.handle.net/2115/67851">http://hdl.handle.net/2115/67851</a>
<b>Type</b>	article (author version)
<b>Additional Information</b>	There are other files related to this item in HUSCAP. Check the above URL.
<b>File Information</b>	HUSCAP-manuscript_Proc_R2_final.pdf



[Instructions for use](#)

1 **Parasite infection induces size-dependent host dispersal:**  
2 **consequences for parasite persistence**

3 Akira Terui<sup>1, 2\*</sup>, Keita Ooue<sup>2</sup>, Hirokazu Urabe<sup>3</sup>, Futoshi Nakamura<sup>2</sup>

4

5 **Affiliation:**

6 <sup>1</sup>Department of Ecology, Evolution, and Behavior, University of Minnesota, 1479

7 Gortner Avenue, St. Paul, MN 55108, USA

8 <sup>2</sup>Department of Forest Science, Graduate School of Agriculture, Hokkaido University,

9 Kita 9, Nishi 9, Kita-ku, Sapporo 060-8589, Japan

10 <sup>3</sup>Salmon and Freshwater Fisheries Research Institute, Hokkaido Research Organization,

11 3-373 Kitakashiwagi, Eniwa, 061-1433, Japan,

12

13 \* Corresponding author

14 Tel., Fax: +81 11 706 3842

15 E-mail address: hanabi0111@gmail.com (A. Terui)

16

17 **Abstract**

18 Host dispersal is now recognized as a key predictor of the landscape-level persistence  
19 and expansion of parasites. However, current theories treat post-infection dispersal  
20 propensities as a fixed trait, and the plastic nature of host's responses to parasite  
21 infection has long been underappreciated. Here, we present a mark-recapture  
22 experiment in a single-host parasite system (larval parasites of the freshwater mussel  
23 *Margaritifera laevis* and its salmonid fish host *Oncorhynchus masou masou*) and  
24 provide the first empirical evidence that parasite infection induces size-dependent host  
25 dispersal in the field. In response to parasite infection, large fish become more  
26 dispersive, whereas small fish tend to stay at the home patch. The observed plasticity in  
27 dispersal is interpretable from the viewpoint of host fitness: expected benefits (release  
28 from further infection) may exceed dispersal-associated costs for individuals with high  
29 dispersal ability (i.e., large fish) but are marginal for individuals with limited dispersal  
30 ability (i.e., small fish). Indeed, our growth analysis revealed that only small fish hosts  
31 incurred dispersal costs (reduced growth). Strikingly, our simulation study revealed that  
32 this plastic dispersal response of infected hosts substantially enhanced parasite  
33 persistence and occupancy in a spatially structured system. These results suggest that  
34 dispersal plasticity in host species is critical for understanding how parasites emerge,

35 spatially spread, and persist in nature. Our findings provide a novel starting point for

36 building a reliable, predictive model for parasite/disease management.

37

38 Key words: dispersal, plasticity, Bayesian statistics, freshwater mussel, salmon

39

40 **Introduction**

41 The rising tide of infectious parasites has motivated parasite/disease ecologists to  
42 establish the factors that influence parasite persistence and expansion in nature [1-3].  
43 Classic studies have explored basic rules for parasite persistence in a locally well-mixed  
44 host population and advance the concept of “critical community size” (i.e., the threshold  
45 host density below which a parasite species cannot persist) [3]. More recently, however,  
46 researchers have begun to recognize the importance of large-scale spatial processes, in  
47 which host dispersal plays a pivotal role in mediating spatial expansion of locally  
48 infected host groups/parasite-contaminated habitats [4-6]. In general, parasites *per se*  
49 have a very limited dispersal capability [7]. Hence, host dispersal is a primary  
50 determinant for the landscape-level dynamics of spatially structured parasite  
51 populations [5-7].

52 Host dispersal is thought to be an effective behavior that enables host  
53 individuals to escape from parasite-contaminated habitats [8-10]. However, the  
54 evolutionarily stable strategy [11] of infected host’s dispersal depends on the cost-  
55 benefit balance of dispersal: if dispersal ensures the avoidance of further infection risks  
56 with little or no mortality, theory predicts that natural selection favors increased  
57 dispersal tendencies of infected hosts (and *vice versa*) [10]. Predicting such parasite-

58 induced changes in host dispersal behavior is a crucial issue of parasite/disease ecology,  
59 since host dispersal propensity drives the spatial spread of parasites in the landscape [5,  
60 6]. However, current theories build on the implicit assumption that dispersal changes in  
61 response to parasite infection are constant within a single host species (i.e., a fixed trait)  
62 [10]. Host populations are heterogeneous entities of individuals with varying phenotype  
63 (e.g., body size), and the net benefits of dispersal may depend on pre-infection  
64 individual status. For example, the inherent costs of dispersal, such as reduced energy  
65 reserves or survival [12], may outweigh its potential benefits if the host's performance  
66 is insufficient to survive consecutive dispersal processes (i.e., departure, transition, and  
67 settlement [13]). Hence, individual-level variation likely exists among post-infection  
68 dispersal propensities (i.e., a plastic response). Nevertheless, neither theoretical nor  
69 empirical studies have explored the possibility of plastic post-infection dispersal  
70 (conditional on individual phenotype) to date, and its potential consequences for  
71 spatially structured parasite populations are virtually unknown.

72         Here, we investigate whether a host species shows plastic post-infection  
73 dispersal propensities by using a tractable host-parasite system: larval parasites of the  
74 Japanese freshwater mussel *Margaritifera laevis* and its salmonid host *Oncorhynchus*  
75 *masou masou*. As with many metazoan parasites, the life cycle of *M. laevis* can be

76 divided into free-living and obligate parasitic stages [cf. 7]. The free-living animals of  
77 *M. laevis* are sedentary, stream-dwelling benthic organisms (mussels) broadcasting  
78 millions of larval parasites (glochidia): their larvae are obligate, external parasites on  
79 the gills of masu salmon *O. m. masou* (or subspecies *O. m. ishikawae*) [14]. After a 40–  
80 50-day parasitic period, glochidia transform into juvenile mussels [14], which recruit  
81 into existing mussel aggregations or invade into unoccupied, parasite-free habitats via  
82 host dispersal (Fig. 1; [15, 16]). Thus, host dispersal is a key factor determining the  
83 landscape-level expansion of the parasite species [15, 16].

84         This single-host parasite interaction (no intermediate host or direct host-to-host  
85 transmission) serves as an excellent model system to test how host individuals respond  
86 to parasite infection, for the following reasons. First, salmonid populations have a clear  
87 size structure with a competitive dominance hierarchy [17, 18], and their body size is  
88 known to vary positively with total energy reserves (e.g., time until fatigue) and  
89 swimming ability [19]. Therefore, salmonid body size may be a simple, but powerful  
90 predictor of post-infection dispersal propensities. Second, Margaritiferidae employ a  
91 simple infection strategy by which drifting glochidia released from female mussels  
92 parasitize the gills of salmonid hosts [14, 20]. The infection status of masu salmon  
93 (infected or uninfected) can be readily manipulated, allowing us to experimentally

94 compare the dispersal propensity between infected and uninfected fish hosts in the field.  
95 Finally, these host and parasite species inhabit relatively small streams, where direct  
96 observation of host dispersal is highly feasible [21].

97         In this study, we tested the hypothesis that glochidial infection induces size-  
98 dependent dispersal in salmonid fish hosts. Specifically, we predicted that glochidial  
99 infection enhances the dispersal tendency of large fish hosts, while suppressing that of  
100 small fish. We directly compared dispersal kernels between uninfected and infected fish  
101 using data from 215 marked individuals, half of which were artificially infected with *M.*  
102 *laevis* glochidia. We also examined how host fitness (growth rate) varied with dispersal  
103 distance and tested the hypothesis that small fish host have higher costs of dispersal  
104 than large fish. Finally, to predict the consequences of the observed host dispersal on  
105 landscape-level persistence and expansion of the parasite, we carried out a simulation  
106 study in a one-dimensional landscape of 100 habitat patches.

107

108

## 109 **Methods**

### 110 *Study site and study species*

111 We conducted this study in the Chitose river system, Hokkaido, Japan. In Hokkaido,



112 glochidia of *M. laevis* (~50 µm in shell length [14]) are released in the summer, from  
113 mid- to late-July [15, 22], and infect the gills of masu salmon (Fig. 1). Female mussels  
114 release synchronously millions of glochidial parasites into the water column (~4 million  
115 glochidia per female; [14, 15]), causing extremely high prevalence of glochidial  
116 infection near dense mussel aggregations (~100% for hundreds of fish hosts) [15, 16].  
117 The proportion of infected fish declines sharply with distance from the infection source  
118 [15]. The maximum life span of *M. laevis* is ~79 years [14]. *Margaritifera laevis* is the  
119 only species of freshwater mussel within the river system.

120           Adult masu salmon spawn in the autumn, and eggs hatch and develop into  
121 juvenile salmon (parr) by early summer. The population of available hosts during the  
122 brooding period of *M. laevis* (beginning in July) is composed mainly of parr (fish at age  
123 0+), which are suitable hosts for Margaritiferidae [23, 24].

124           We conducted a mark-recapture experiment in the Osatsu stream (42°50'N  
125 141°36'E), a small tributary flowing into the Chitose river. This spring-fed stream serves  
126 as a suitable experimental venue because 1) this system is characterized by little  
127 temporal variation in water temperature (range: 4–12 °C) and discharge [25], and  
128 because 2) *M. laevis* does not occur in this stream (confounding *M. laevis* infection can  
129 be avoided). Field surveys were approved by the Hokkaido prefecture, and all research

130 was performed in line with the Animal Care and Use guideline of Hokkaido University.

131

132 *Mark-recapture experiment*

133 We selected a 1,200-m stretch of the Osatsu stream with a 3.0–6.0-m wetted width,  
134 where local habitat conditions varied little along the stream stretch, and no apparent  
135 dispersal barriers existed. The stretch was divided into 60 capture subsections (each 20  
136 m in length). The first capture session was carried out from July 21 to 24, 2015, and the  
137 recapture session occurred ca. 50 days later (Sep 8–11, 2015). This capture-recapture  
138 interval was intended to mimic the duration of glochidial infection under the summer  
139 water temperature of the Osatsu stream (~12 °C) [26].

140         During the first capture session, we collected host fish using consecutive three-  
141 pass electrofishing in each subsection [27]. We anesthetized captured fish in a 2-  
142 phenoxyethanol solution and measured their fork length (millimeters) and wet mass (0.1  
143 g). We batch-marked them with fluorescent visible implant elastomer tags (Northwest  
144 Marine Technologies, Shaw Island, Washington) applied to three adipose locations  
145 (behind the eye, behind the nose, and the lower jaw). We used six tag colors, and the  
146 combination of tag color and position allowed the identification of individual fish ( $6^3 =$   
147 216 patterns). We allowed marked fish to recover in a container for 10–15 min. We did

148 not mark 1+ fish hosts as they have been identified as unsuitable hosts for  
149 Margaritiferids [23, 24].

150           Half the marked fish collected from each individual subsection were infected  
151 with *M. laevis* glochidia by placing them into an infection bath (5-L bucket,  $4 \times 10^4$   
152 glochidia  $L^{-1}$ ) for 30 minutes. The other half was kept in a “sham” infection bath with  
153 no glochidia (control). This experimental design isolates infection-treatment effects  
154 from any environmental variation among subsections. We obtained a preliminary  
155 confirmation that this glochidial density provides no infection failure and natural levels  
156 of glochidial load ( $48 \pm 29$  glochidia per fish), based on the level commonly observed in  
157 the Chitose river [22, 28]. We created the infection bath using fresh viable glochidia that  
158 were naturally released from a total of four gravid *M. laevis* females (collected every  
159 morning from a single population of the Chitose river). The averages and variance of  
160 fish body size (fork length) were almost identical between infected and uninfected fish  
161 groups (t-test,  $P = 0.18$ ; mean for uninfected fish =  $92.5 \pm 11.2$  mm, mean for infected  
162 fish =  $90.5 \pm 11.2$  mm).

163           Marked fish were then released near the center of the subsection where they  
164 were caught. We completed all procedures from 7:00 to 15:00 in light of the short  
165 longevity of glochidia [29]. We marked a total of 215 individuals.

166 At the recapture session, we recaptured marked fish with three-pass  
167 electrofishing in each of the subsections. The longitudinal position (recorded as  
168 subsection ID, 1–60), fork length, and wet mass of all recaptured fish were recorded.

169

170 *Dispersal model coupled with observation process*

171 We employed the Laplace (double exponential) kernel, which has been proven to  
172 provide adequate fits to dispersal data in various salmonid fish [30, 31]. The Laplace  
173 density function,  $f_L$ , has a symmetrical exponential decay to either side of the origin,  
174 with the inverse of parameter  $\tau$  equal to the mean dispersal distance (i.e., smaller values  
175 of  $\tau$  indicate greater dispersal tendency):

176

177 
$$f_L(x_{j(i)}, \mu_i, \tau_i) = \frac{1}{2} \tau_i \exp(-\tau_i |x_{j(i)} - \mu_i|) \quad \text{Eq.1}$$

178

179 where  $x_{j(i)}$  is the distance class (i.e., distance to the downstream end of the whole study  
180 section) at the center of recapture subsection  $j$  (subscript  $j(i)$  denotes  $j$ th subsection in  
181 which fish individual  $i$  was recaptured), and  $\mu_i$  is the distance class at the center of the  
182 capture subsection for fish individual  $i$ . It is important to note that the variance ( $2/\tau_i^2$ )  
183 nonlinearly increases with increasing mean dispersal distance ( $1/\tau_i$ ). Thus, the model

184 can express outliers adequately (i.e., robust to outlier data), which is typical for  
185 dispersal data [30, 32]. We related the mean dispersal distance to individual-level  
186 predictors with a log-link function:

187

$$188 \quad \log(1/\tau_i) = \beta_0 + \beta_1 \cdot \text{Infection}_i + \beta_2 \cdot \text{Size}_i + \beta_3 \cdot \text{Infection}_i \cdot \text{Size}_i \quad \text{Eq. 2}$$

189

190 where  $\beta_0$  is an intercept and  $\beta_1$ – $\beta_3$  are standardized regression coefficients of infection  
191 status  $\text{Infection}_i$  (binary variable; infected = 1, uninfected = 0), initial body size  $\text{Size}_i$   
192 (continuous variable; standardized with a mean 0 and SD 1) and their interaction. As  
193 initial body size was standardized, parameter  $\beta_0$  indicates the average  $1/\tau$  of the  
194 population in a logarithmic scale. This formulation allows us to evaluate the effects of  
195 individual-level predictors on the form of dispersal kernels.

196 To incorporate sampling designs into the parameter inference of dispersal  
197 kernels, we modified the inference framework proposed by Pepino *et al.* [31]. The  
198 binary variable of capture history  $Y_i$  ( $Y_i = 1$  if recaptured, otherwise 0) was modeled  
199 based on a Bernoulli distribution (see Electronic Supplementary Material for  
200 derivation),

201

202  $Y_i \sim \text{Bernoulli}(\varphi_{j(i)}s_iD_i)$  Eq. 3

203

204 where  $\varphi_{j(i)}$  is the section-specific probability of capture and  $s_i$  is the survival probability  
 205 during the study period. For recaptured individuals,  $D_i$  is the probability that individual  $i$   
 206 moves from release point  $\mu_i$  to subsection of recapture  $j$ . For unrecaptured individuals,  
 207  $D_i$  represents the probability of staying in the 1,200-m study stretch:

208

209 
$$D_i = \begin{cases} \int_{x_{j(i),low}}^{x_{j(i),up}} f_L(x_{j(i)}, \mu_i, \tau_i) dx_{j(i)}, & \text{if recaptured} \\ \int_{Low}^{Up} f_L(x_{j(i)}, \mu_i, \tau_i) dx_{j(i)}, & \text{if unrecaptured} \end{cases}$$
 Eq. 4

210

211 where  $x_{j(i),up}$  and  $x_{j(i),low}$  are the distance classes at the upper and lower boundaries of the  
 212 recapture subsection  $j$  for individual  $i$ , and  $Up$  and  $Low$  are the distance classes at the  
 213 upper (1,200 m) and lower ends (0 m) of the whole study section. Individual-specific  
 214 survival probability  $s_i$  is an identifiable parameter, as we obtained an independent  
 215 estimate of section-specific capture probability  $\varphi_{j(i)}$  using the three-pass depletion  
 216 surveys with a Bayesian modification (see Electronic Supplementary Material) [27].  
 217 Survival probability was normally distributed in a logit scale:  $\text{logit}(s_i) \sim$   
 218  $\text{Normal}(\text{logit}(s_{global}), \sigma_s^2)$ , where  $s_{global}$  represents the mean survival probability.

219 However, it was impossible to determine  $\varphi_{j(i)}$  for unrecaptured individuals,

220 since we have no information on subsection ID of recapture. Instead, for unrecaptured  
 221 individuals, we estimated the weighted mean of  $\varphi_{j(i)}$  across subsections ( $\varphi_{w,i}$ ) given the  
 222 dispersal parameter  $\tau_i$ :

223

$$224 \quad \varphi_{w,i} = \sum_{j=1}^{60} w_{j(i)} \varphi_j \quad \text{Eq. 5}$$

$$225 \quad w_{j(i)} = \frac{\int_{x_{j(i),low}}^{x_{j(i),up}} f_L(x_{j(i)}, \mu_i, \tau_i) dx_{j(i)}}{\int_{Low}^{Up} f_L(x_{j(i)}, \mu_i, \tau_i) dx_{j(i)}} \quad \text{Eq. 6}$$

226

227 In equation 6, the numerator indicates the probability of movement from release point  $\mu_i$   
 228 to  $j$ th subsection (i.e., the probability of unrecaptured individual  $i$  present at  $j$ th  
 229 subsection during the recapture session). The denominator (the probability of staying in  
 230 the 1,200-m study stretch) scales the numerator so that  $\sum_{j=1}^{60} w_{j(i)}$  equals 1.0.

231 Vague priors were assigned to the parameters: normal distributions for  $\beta$  (mean  
 232 = 0, variance =  $10^4$ ), a beta distribution for  $s_{global}$  (shape = 1, scale = 1), and a truncated  
 233 normal distribution for  $\sigma_s^2$  (mean = 0, variance =  $10^4$ , range = 0–100) The model was  
 234 fitted to the data with JAGS ver. 4.1.0 and the package “*runjags*” [33] in R 3.3.1 [34].  
 235 Three Markov chain Monte Carlo (MCMC) chains were run with 9,000 iterations  
 236 (3,000 burn-in), and 500 samples per chain were used to calculate posterior  
 237 probabilities. Convergence was assessed by examining whether the R-hat indicator of

238 each parameter approached 1 [35].

239

240 *Host growth analysis*

241 We analyzed factors that influence host growth using data from recaptured fish ( $n =$

242 116). We estimated individual host growth  $G$  as:

243

$$244 \quad G = \log(FL_{50}/FL_0) \quad \text{Eq. 7}$$

245

246 where  $FL_0$  and  $FL_{50}$  denote fork length at capture and recapture, respectively.

247 We then constructed a linear mixed effect model with a random effect of initial

248 capture subsection ID to investigate factors influencing host growth  $G$ . Host growth  $G$

249 was assumed to follow a normal distribution and was modeled as a function of distance

250 moved (continuous), logarithm of fork length at initial capture ( $\log(FL_0)$ ; continuous),

251 infection (binary), and their two-way interactions. Continuous explanatory variables

252 were standardized prior to the analysis (mean = 0, SD = 1). However, an analytical issue

253 can arise when using  $G$  as a response variable:  $\log(FL_0)$  will appear in both sides of the

254 equation, causing spurious correlations [36]. To avoid this analytical issue, we put

255  $\log(FL_0)$  in the response variable into the right side (i.e., offset term):  $\log(FL_{50}) = \mathbf{X}\boldsymbol{\beta} +$



256  $\varepsilon + \log(FL_0)$ , where  $\mathbf{X}$  is a matrix of predictors,  $\boldsymbol{\beta}$  is a vector of regression coefficients,  
257 and  $\varepsilon$  is the random effect of initial capture subsection ID.

258 Vague priors were assigned to regression coefficients (normal distributions:  
259 mean = 0, variance = 1,000) and standard deviations of residuals and the random effect  
260 (uniform distributions: range, 0–100). Three MCMC chains were run with 15,000  
261 iterations (5,000 burn-in) using JAGS, and 500 samples per chain were used to calculate  
262 posterior probabilities. Convergence was assessed as indicated above.

263

#### 264 *Simulation*

265 To investigate the consequences of the observed dispersal probabilities on the  
266 landscape-level expansion and persistence of the parasite, we modified a simulation  
267 model described by Grant *et al.* [37]. In our simulation, we assumed a linear landscape  
268 of 100 habitat patches of equal quality and length (20 m). Simulation space boundaries  
269 were wrapped. We initially introduced five parasite-occupied patches into the landscape  
270 (5% occupancy), and a random, independent patch-extinction of parasite-occupied patch  
271 occurred with the probability  $E$  in each time step (i.e., transition from a parasite-  
272 occupied to parasite-free patch). After random extinction events of parasite-occupied  
273 patches, we allowed immediate (re)colonization of mussel aggregation (i.e., transition

274 from a parasite-free to parasite-occupied patch) from other parasite-occupied patches  
275 through host dispersal (Fig. 1): in each time step  $t$ , all host fish at parasite-occupied  
276 patch  $i$  ( $N_{i,t}$ ) were infected with glochidia (100% prevalence of local glochidial  
277 infection; [15, 16]) and dispersed randomly based on the predefined Laplace dispersal  
278 kernels (see below). Every host dispersers had information on its own position (i.e.,  
279 distance [m] to the downstream end). During dispersal, infected host fish survived with  
280 the probability  $s$  ( $s = 0.87$ ; see Results), and each surviving individual that reached a  
281 parasite-free patch  $j$  (i.e., between  $x_{j(i),low} - x_{j(i),up}$ ) had the potential to create a new  
282 infection source (parasite-occupied patch) with the probability  $C$ . Thus, the realized per-  
283 fish colonization probability is the product of  $s$  and  $C$ . Host fish present at parasite-free  
284 patches (i.e., no mussel aggregation) were not considered as dispersal agents.

285           The number of host fish at patch  $i$  at time step  $t$  was drawn from a Poisson  
286 distribution as  $N_{i,t} \sim \text{Poisson}(\lambda_t)$ . This reflects real situations: that is, in each time step,  
287 newly emerged susceptible hosts (i.e., 0+ fish of the year) were randomly (re)distributed  
288 across the simulation space. The mean host density (fish/patch) at time step  $t$ ,  $\lambda_t$ , was  
289 determined by the Ricker model [38-40]:

290

$$291 \quad \log(\lambda_{t+1}) = r - b \cdot \lambda_t + \log(\lambda_t) + \varepsilon_t \quad \text{Eq. 8}$$

292

293 where  $r$  is the intrinsic population growth rate,  $b$  is the parameter that determines  
294 negative density dependence, and  $\varepsilon_t$  is the environmental stochasticity. In this  
295 formulation, the host carrying capacity  $K$  can be written as  $r/b$ . The parameter  $\varepsilon_t$  is  
296 governed by a normal distribution with a mean of 0 and variance of  $\sigma_\varepsilon^2$ , and the  
297 variance parameter  $\sigma_\varepsilon^2$  determines the degree of environmental stochasticity. Host  
298 population dynamics were assumed to be independent of parasite infection, as we did  
299 not find negative effects of glochidial infection on fish growth (see Table 2).

300 The dispersal parameter  $\tau_i$  for the Laplace density function was described as  
301 follows:

302

$$303 \quad \log(1/\tau_i) = \gamma + \delta \cdot z_i \quad \text{Eq. 9}$$

304

305 where  $\gamma$  is the intercept,  $\delta$  is the slope that determines the strength of size-dependence in  
306 host dispersal, and  $z_i$  is the standardized random variable of body size,  $z_i \sim \text{Normal}(0, 1)$ .

307 Note that the parameters ( $\gamma$  and  $\delta$ ) can be substituted by empirical dispersal estimates in  
308 equation 2:  $\log(1/\tau_i) = [\beta_0 + \beta_1 \cdot \text{Infection}_i] + [\beta_2 + \beta_3 \cdot \text{Infection}_i] \cdot \text{Size}_i$ . This  
309 representation allowed us to predict the consequences of observed dispersal patterns on

310 parasite persistence (control [*Infection* = 0]:  $\gamma = \beta_0$ ,  $\delta = \beta_2$ ; treatment [*Infection* = 1]:  $\gamma =$   
311  $\beta_0 + \beta_1$ ,  $\delta = \beta_2 + \beta_3$ ).

312 We examined 16 dispersal parameter combinations ( $\gamma = 1.5, 2.0, 2.5, 3.0$ ;  $\delta =$   
313  $0.0, 0.5, 1.0, 1.5$ ) that cover the empirical estimates (see Results). We also set the  
314 following parameters:  $C = 0.005$ ,  $E = 0.01$ ,  $K = 4$  or  $8$ ,  $r = 1.43$ , and  $\sigma_\varepsilon = 0.18$ . Per-fish  
315 colonization rate  $C$  was parameterized according to the reported mussel mortality during  
316 their early benthic life stage (~99.5%) [41]. Specific estimates of annual per-patch  
317 extinction probability were not available, but descriptive evidence suggests that the  
318 parameter should be  $\leq 0.01$  [42]. The range of parameter  $K$  was determined based on the  
319 masu salmon density in the Osatsu stream (0–12 fish per 20-m subsection). We used  
320 fixed values of  $r$  and  $\sigma_\varepsilon$  based on a meta-analysis by Myers *et al.* [39], and the density-  
321 dependence parameter  $b$  was calculated as  $r/K$ . For a connection with traditional  
322 epidemiological parameters (e.g., the basic reproductive number  $R_0$ ), see Electronic  
323 Supplementary Material.

324 We ran the simulation model for a maximum of 10,000 time steps. We obtained  
325 persistence time of the spatially structured parasite population (time to the entire  
326 extinction of parasite-occupied patch) and median proportion of parasite-occupied patch  
327 (hereafter, “occupancy”) during the persistent period (i.e., a period during which non-

328 zero occupancy of parasite-occupied patch was observed). Each parameter combination  
329 was replicated 25 times. The initial mean host density  $\lambda_0$  was set to be five fish/patch for  
330 all cases. All simulations were conducted in the C++ environment using the R package  
331 “*Rcpp*” [43].

332

333

## 334 **Results**

### 335 *Mark-recapture study*

336 Among 215 marked fish hosts (fork length: 62–121 mm), 116 individuals (infected fish,  
337 54 individuals; uninfected fish, 62 individuals) were successfully recaptured and  
338 identified (no complete tag loss was observed). The average capture probability of our  
339 three-pass electrofishing  $\phi$  during the recapture session was reasonably high (median:  
340 0.93, 95% CI: 0.83–0.98).

341       Using the dispersal data as well as the capture history of the marked fish, we  
342 developed a Bayesian dispersal model coupled with observation processes (capture  
343 probability  $\phi$ , survival probability  $s$ , and sampling designs). Estimated survival  
344 probability  $s_{global}$  during the mark-recapture period was 0.87 (95%CI: 0.62–0.99) with  
345 among-individuals variation ( $\sigma_s^2$ ) of 6.03 (95%CI: 0.84–17.18). The model also revealed

346 that parasite infection interacted with host body size to modify host dispersal kernels  
347 (Table 1). Glochidial infection influenced large fish to be more dispersive, but had  
348 opposite effects on small individuals (Fig. 2b). The probability of leaving behind the  
349 home subsection was high for large fish hosts (0.76; 80<sup>th</sup> percentile of body size);  
350 specifically, it was 4.3 times greater than that of small fish hosts (0.18; 20<sup>th</sup> percentile of  
351 body size). However, such clear size-dependence in dispersal was not observed for  
352 uninfected fish hosts (Fig. 2a). The interactive effect of host body size and parasite  
353 infection remained significant even after removing two “super dispersers” (i.e., outliers;  
354 only two individuals dispersed  $\geq 180$  m; see Table S1 and Fig. S1). For raw data, see  
355 Fig.S1 in Electronic Supplementary Material.

356 Growth analysis revealed that dispersal costs were size-specific. The main  
357 effect of body size and the interaction term with dispersal distance were detected with a  
358 probability of  $\geq 0.95$  (Table 2). The growth of small fish hosts decreased with increasing  
359 dispersal distance (Fig. 3). In contrast, this pattern was less apparent for large fish hosts  
360 (Fig. 3). The lack of small “super” dispersers likely reflects the size-specific costs of  
361 dispersal. Glochidial infection had little effect on fish growth.

362

363 *Simulation*

364 Dispersal plasticity had a strong positive impact on the landscape-level persistence and  
365 expansion of the parasite, especially when the host population size was large (see Fig.  
366 4b and d; larger values of y-axis represent stronger size-dependence in dispersal). The  
367 observed plasticity in dispersal led to approximately four times longer persistence of  
368 spatially structured parasite population with greater occupancy (~8,000 time steps with  
369 ~30% occupancy; filled dot in Fig.4b and d) compared with the weak plastic dispersal  
370 scenario (~2,000 time steps with ~6% occupancy; open dot in Fig.4b and d). However,  
371 this contrast was not so clear when the host population size was small (Fig. 4a and c).

372

373

## 374 **Discussion**

375 Host dispersal is now recognized as a key mediator of the landscape-level persistence  
376 and expansion of parasites [4-6]. However, the plastic nature of host dispersal responses  
377 to parasites has long been underappreciated, despite the fact that intrapopulation  
378 variation in phenotype (e.g., body size) is ubiquitous. Inherent difficulties exist in  
379 manipulating infection status and quantifying dispersal in natural settings, and these  
380 problems have hindered the progress of this research field. Here, using a tractable host-  
381 parasite system embedded in a one-dimensional landscape (i.e., a stream), our field

382 experiment overcame these difficulties and provided the first quantitative evidence that  
383 parasite infection induces size-dependent host dispersal. Strikingly, our simulation  
384 suggested that the observed individual-level variation in host dispersal may greatly  
385 enhance parasite persistence and occupancy in a spatially structured system. This is an  
386 emergent phenomenon that cannot be understood without the inclusion of among-  
387 individuals differences in dispersal propensities. These findings provide an important  
388 insight into how parasites emerge, spatially spread, and persist in stochastic natural  
389 environments.

390         As *M. laevis* glochidia parasitism occurs mainly in the vicinity of adult mussel  
391 aggregations (infection rate drops approximately 0.20 with every hundred meters of  
392 distance from aggregation) [15], dispersal seems to be an effective measure to avoid  
393 further infection for the salmonid host. Concordant with our hypothesis, there was  
394 substantial variation in host dispersal: large fish hosts became more dispersive, whereas  
395 small individuals tended to stay in the home patch. This size-specific dispersal is  
396 interpretable from the viewpoint of host fitness. In brief, host dispersers cannot benefit  
397 from parasite-avoidance unless they survive the dispersal process and reach a new,  
398 parasite-free habitat. Our results are consistent with this intuitive prediction. Even under  
399 the influence of parasite virulence, the plentiful energy reserves of large salmonid fish



400 may enable them to survive the risky transition process. In contrast, for small fish hosts,  
401 glochidia-induced changes (e.g., respiratory burden) [44] may lead to a failure to  
402 transition, given their presumed susceptibility to energetic and/or risk costs during  
403 dispersal (see [19] for body size effects on swimming performance). Further costs can  
404 be levied at the settlement stage, as small salmonid fish are competitively inferior in the  
405 dominance hierarchy [17, 18]. Indeed, our growth analysis produced some support for  
406 this interpretation, as only small fish hosts incurred dispersal costs (reduced growth; see  
407 Fig. 2). Therefore, the net benefits of dispersal are expected to be higher for large fish  
408 hosts, but marginal for small fish.

409           Alternatively, it is possible that *M. laevis* glochidia actively manipulated host  
410 dispersal behavior. However, *M. laevis* does not seem to have strong incentive for  
411 manipulating host dispersal, as the parasite possesses neither a complex life cycle (i.e.,  
412 no secondary host) nor specific spawning habitats, both of which are often associated  
413 with active host manipulation [7]. Considering the evidence, we suggest that the  
414 observed dispersal changes may be a plastic response of salmonid fish hosts, and a  
415 “fixed dispersal response” to parasites (e.g., all individuals disperse more; increase in  $x$ -  
416 axis values in Fig. 3) may not be the best option for the host species owing to the size-  
417 specific costs of dispersal.

418 Intriguingly, our simulation revealed that the host's plastic response to parasite  
419 infection has the potential to increase landscape-level parasite persistence and  
420 occupancy, provided that host carrying capacity  $K$  is high. The rationale behind this  
421 finding is the following: large host population sizes ensure a certain fraction of a host  
422 population consists of movers (large fish hosts) that allow parasites to colonize distant  
423 patches and spread spatially. Meanwhile, stayers (small fish hosts) effectively reinforce  
424 (or recolonize) adjacent patches (i.e., the mixture of individual dispersal kernels makes  
425 up a "fat-tailed dispersal kernel"). This result deserves further attention. If the observed  
426 host response is truly adaptive, then a host's adaptive behavior, either avoiding infection  
427 risks or dispersal costs, may lead to undesired consequences: the parasite invades a  
428 larger fraction of the patches and persists in the spatially structured system. However,  
429 we do not have the valid evidence that dispersal plasticity maintains host fitness (i.e.,  
430 adaptive), and further experimentation is needed to confirm this possibility. Future  
431 investigations addressing this issue would shed light on how host-parasite interactions  
432 are stably sustained in spatially structured systems.

433 Mark-recapture studies are recurrently criticized for the limited coverage of  
434 potential dispersal ranges [e.g., 45]. However, our statistical approach is robust against  
435 this problem, as we incorporated sampling designs into the dispersal parameter

436 inference (see Eq. 3; individuals that had left the study section were taken into account).  
437 This has been shown to provide reliable dispersal parameters, especially when the  
438 length of the study stretch is  $> 4$  times greater than the average dispersal distance [46].  
439 This likely holds true in our study, as the length of the whole study section (1,200 m)  
440 was  $\sim 92$  times longer than the average dispersal distance of masu salmon ( $\exp(2.57) =$   
441 13.1 m; see Table 1). Therefore, we are confident in our dispersal parameter inference.

442           Another potential issue would be whether our findings are applicable to  
443 horizontally transmissible systems (direct host-to-host transmission). Although our  
444 system has several comparative advantages owing to the lack of horizontal transmission  
445 (e.g., infection status is readily controllable throughout the experiment), there are  
446 certain differences in local transmission dynamics. Nevertheless, we expect that our  
447 findings may be equally important in those systems because the landscape-level  
448 expansion of horizontally transmissible parasites should also occur mainly through host  
449 dispersal. The integration of local horizontal transmission is beyond the scope of our  
450 study, but this issue may be a fruitful avenue for future theoretical research.

451           Although the importance of host dispersal has been increasingly appreciated in  
452 the field of infectious parasite research, researchers have failed to fully account for the  
453 heterogeneous nature of wild organisms. By combining empirical and simulated

454 approaches, the present study provides a novel parameter (i.e., individual-level variation  
455 in host phenotype) for predicting the long-term persistence of parasites and the  
456 landscape-level expansion of parasite-contaminated habitats. As intrapopulation  
457 heterogeneity of phenotype can be found in almost all animals, our findings may be  
458 widely applicable to other host-parasite systems. Future generalization across systems  
459 should provide a novel and critical perspective on parasite/disease management issues.

460

461

462 **Acknowledgements** We thank a member of forest ecosystem management laboratory  
463 for their help in the field. We are also grateful to Drs. H. Katahira, N. Ishiyama, Y. Morii  
464 and S. Yamanaka for their constructive comments on the early draft of this manuscript.

465

466 **Author contributions** AT and KO designed and conducted the experiment. AT  
467 performed statistical analysis and simulation. All authors participated in conception,  
468 discussion of the results and manuscript preparation.

469

470 **Data accessibility**

471 Data and scripts (JAGS and Rcpp) are available at Dryad [47]

472 (<http://dx.doi.org/10.5061/dryad.14mt6>).

473

474 **References**

- 475 1. Altizer S., Bartel R., Han B.A. 2011 Animal migration and infectious disease risk. *Science* **331**,  
476 296-302.
- 477 2. Harvell D., Aronson R., Baron N., Connell J., Dobson A., Ellner S., Gerber L., Kim K., Kuris  
478 A., McCallum H. 2004 The rising tide of ocean diseases: unsolved problems and research priorities.  
479 *Frontiers in Ecology and the Environment* **2**, 375-382.
- 480 3. McCallum H., Barlow N., Hone J. 2001 How should pathogen transmission be modelled?  
481 *Trends in Ecology & Evolution* **16**, 295-300.
- 482 4. Cross P.C., Lloyd-Smith J.O., Johnson P.L.F., Getz W.M. 2005 Duelling timescales of host  
483 movement and disease recovery determine invasion of disease in structured populations. *Ecology Letters*  
484 **8**, 587-595.
- 485 5. Hess G. 1996 Disease in metapopulation models: implications for conservation. *Ecology* **77**,  
486 1617-1632.
- 487 6. Lopez J., Gallinot L., Wade M. 2005 Spread of parasites in metapopulations: an experimental  
488 study of the effects of host migration rate and local host population size. *Parasitology* **130**, 323-332.
- 489 7. Poulin R. 2007 *Evolutionary ecology of parasites*. Oxfordshire, UK, Princeton University  
490 Press.
- 491 8. Suhonen J., Honkavaara J., Rantala M.J. 2010 Activation of the immune system promotes  
492 insect dispersal in the wild. *Oecologia* **162**, 541-547.
- 493 9. Brown G.P., Kelehear C., Pizzatto L., Shine R. 2016 The impact of lungworm parasites on rates  
494 of dispersal of their anuran host, the invasive cane toad. *Biological Invasions* **18**, 103-114.
- 495 10. Iritani R., Iwasa Y. 2014 Parasite infection drives the evolution of state-dependent dispersal of  
496 the host. *Theoretical Population Biology* **92**, 1-13.
- 497 11. Smith J.M., Price G.R. 1973 The logic of animal conflict. *Nature* **246**, 15-18.
- 498 12. Bonte D., Van Dyck H., Bullock J.M., Coulon A., Delgado M., Gibbs M., Lehouck V.,  
499 Matthysen E., Mustin K., Saastamoinen M. 2012 Costs of dispersal. *Biological Reviews* **87**, 290-312.
- 500 13. Bowler D.E., Benton T.G. 2005 Causes and consequences of animal dispersal strategies:  
501 relating individual behaviour to spatial dynamics. *Biological Reviews* **80**, 205-225.
- 502 14. Kondo T. 2008 *Monograph of Unionoida in Japan (Mollusca: Bivalvia)*. Tokyo, Malacological  
503 Society of Japan.
- 504 15. Terui A., Miyazaki Y. 2015 A "parasite-tag" approach reveals long-distance dispersal of the  
505 riverine mussel *Margaritifera laevis* by its host fish. *Hydrobiologia* **760**, 189-196.
- 506 16. Terui A., Miyazaki Y., Yoshioka A., Kadoya T., Jopp F., Washitani I. 2014 Dispersal of larvae  
507 of *Margaritifera laevis* by its host fish. *Freshwater Science* **33**, 112-123.
- 508 17. Nakano S. 1995 Individual-differences in resource use, growth and emigration under the

- 509 influence of a dominance hierarchy in fluvial red-spotted masu salmon in a natural habitat. *Journal of*  
510 *Animal Ecology* **64**, 75-84.
- 511 18. Sato T., Watanabe K. 2014 Do stage-specific functional responses of consumers dampen the  
512 effects of subsidies on trophic cascades in streams? *Journal of Animal Ecology* **83**, 907-915.
- 513 19. Ojanguren A.F., Braña F. 2003 Effects of size and morphology on swimming performance in  
514 juvenile brown trout (*Salmo trutta* L.). *Ecology of freshwater fish* **12**, 241-246.
- 515 20. Haag W.R. 2012 *North American Freshwater Mussels: Natural History, Ecology, and*  
516 *Conservation*. New York, USA, Cambridge University Press.
- 517 21. Lowe W.H. 2009 What drives long-distance dispersal? A test of theoretical predictions.  
518 *Ecology* **90**, 1456-1462.
- 519 22. Awakura T. 1969 On the correlation between the age composition of the freshwater pearl  
520 mussel, *Margaritifera laevis* (Haas) and the population size of salmonid fishes. *Scientific Report of the*  
521 *Hokkaido Salmon Hatchery* **24**, 55-88.
- 522 23. Awakura T. 1968 The ecology of parasitic glochidia of the fresh-water pearl mussel,  
523 *Margaritifera laevis* (Haas). *Scientific Report of the Hokkaido Salmon Hatchery* **23**, 1-17.
- 524 24. Hastie L.C., Young M.R. 2001 Freshwater pearl mussel (*Margaritifera margaritifera*)  
525 glochidiosis in wild and farmed salmonid stocks in Scotland. *Hydrobiologia* **445**, 109-119.
- 526 25. Kawai H., Ishiyama N., Hasegawa K., Nakamura F. 2013 The relationship between the  
527 snowmelt flood and the establishment of non-native brown trout (*Salmo trutta*) in streams of the Chitose  
528 River, Hokkaido, northern Japan. *Ecology of freshwater fish* **22**, 645-653.
- 529 26. Kobayashi O., Kondo T. 2005 Difference in host preference between two populations of the  
530 freshwater pearl mussel *Margaritifera laevis* (Bivalvia: Margaritiferidae) in the Shinano River system,  
531 Japan. *Venus* **64**, 63-70.
- 532 27. Dorazio R.M., Jelks H.L., Jordan F. 2005 Improving removal - based estimates of abundance  
533 by sampling a population of spatially distinct subpopulations. *Biometrics* **61**, 1093-1101.
- 534 28. Akiyama Y. 2007 Factors causing extinction of a freshwater pearl mussel, *Margaritifera laevis*  
535 in Japan (Bivalvia: Unionoida) [thesis]. Sapporo, Hokkaido University.
- 536 29. Akiyama Y., Iwakuma T. 2007 Survival of glochidial larvae of the freshwater pearl mussel,  
537 *Margaritifera laevis* (Bivalvia : Unionoida), at different temperatures: A comparison between two  
538 populations with and without recruitment. *Zoological Science* **24**, 890-893.
- 539 30. Rodriguez M.A. 2002 Restricted movement in stream fish: The paradigm is incomplete, not  
540 lost. *Ecology* **83**, 1-13.
- 541 31. Pepino M., Rodriguez M.A., Magnan P. 2012 Fish dispersal in fragmented landscapes: a  
542 modeling framework for quantifying the permeability of structural barriers. *Ecological Applications* **22**,  
543 1435-1445.
- 544 32. Nathan R., Klein E., Robledo-Arnuncio J.J., Revilla E. 2012 Dispersal kernels: review. In

- 545 *Dispersal Ecology and Evolution* (eds. Clobert J., Baguette M., Benton T.G., Bullock J.M.), pp. 187-210.  
546 Oxford, UK, Oxford University Press.
- 547 33. Denwood M. 2013 runjags: An R package providing interface utilities, parallel computing  
548 methods and additional distributions for MCMC models in JAGS. *Journal of Statistical Software* **1**, 0-7.
- 549 34. R Core Team (2016). R: A language and environment for statistical computing. R Foundation  
550 for Statistical Computing, Vienna, Austria. <https://www.R-project.org/>. Available at: [https://www.R-](https://www.R-project.org/)  
551 [project.org/](https://www.R-project.org/) Last accessed 13 July 2016.
- 552 35. Gelman A., Hill J. 2007 *Data Analysis Using Regression and Multilevel/Hierarchical Models*.  
553 New York, USA, Cambridge University Press.
- 554 36. Jackson D., Somers K. 1991 The spectre of 'spurious' correlations. *Oecologia* **86**, 147-151.
- 555 37. Grant E.H.C., Nichols J.D., Lowe W.H., Fagan W.F. 2010 Use of multiple dispersal pathways  
556 facilitates amphibian persistence in stream networks. *Proceedings of the National Academy of Sciences of*  
557 *the United States of America* **107**, 6936-6940.
- 558 38. Krkošek M., Ford J.S., Morton A., Lele S., Myers R.A., Lewis M.A. 2007 Declining wild  
559 salmon populations in relation to parasites from farm salmon. *Science* **318**, 1772-1775.
- 560 39. Myers R.A., Bowen K.G., Barrowman N.J. 1999 Maximum reproductive rate of fish at low  
561 population sizes. *Canadian Journal of Fisheries and Aquatic Sciences* **56**, 2404-2419.
- 562 40. Ricker W.E. 1954 Stock and recruitment. *Journal of the Fisheries Board of Canada* **11**, 559-  
563 623.
- 564 41. Schmidt C., Vandre R. 2010 Ten years of experience in the rearing of young freshwater pearl  
565 mussels (*Margaritifera margaritifera*). *Aquatic Conservation-Marine and Freshwater Ecosystems* **20**,  
566 735-747.
- 567 42. Hastie L.C., Boon P.J., Young M.R., Way S. 2001 The effects of a major flood on an  
568 endangered freshwater mussel population. *Biological Conservation* **98**, 107-115.
- 569 43. Eddelbuettel D., François R., Allaire J., Chambers J., Bates D., Ushey K. 2011 Rcpp: Seamless  
570 R and C++ integration. *Journal of Statistical Software* **40**, 1-18.
- 571 44. Thomas G.R., Taylor J., de Leaniz C.G. 2014 Does the parasitic freshwater pearl mussel *M.*  
572 *margaritifera* harm its host? *Hydrobiologia* **735**, 191-201.
- 573 45. Gowan C., Fausch K.D. 1996 Mobile brook trout in two high-elevation Colorado streams: re-  
574 evaluating the concept of restricted movement. *Canadian Journal of Fisheries and Aquatic Sciences* **53**,  
575 1370-1381.
- 576 46. Pépino M., Rodríguez M.A., Magnan P. 2016 Assessing the detectability of road crossing  
577 effects in streams: mark-recapture sampling designs under complex fish movement behaviours. *Journal*  
578 *of Applied Ecology* **53**, 1831-1841.
- 579 47. Terui A., Ooue K., Urabe H., Nakamura F. 2017 Data from: Parasite infection induces size-  
580 dependent host dispersal: consequences for parasite persistence. Dryad.



581 <http://dx.doi.org/10.5061/dryad.14mt6>.

582

583

584 **Table 1** Results of the Bayesian model that explains individual-level variation in  
 585 dispersal kernel. Posterior probability represents the proportion of parameter estimates  
 586 (MCMC samples) assigned to be either negative or positive. Parameters with a posterior  
 587 probability of  $> 0.95$  are shown in bold. SE: standard error.

Effect	Estimate	SE	Posterior probability	
			Negative	Positive
Intercept ( $\beta_0$ )	2.571	0.092	-	-
Infection ( $\beta_1$ )	0.151	0.135	0.12	0.88
Body size ( $\beta_2$ )	<b>0.319</b>	0.089	0.00	<b>1.00</b>
Infection · Size ( $\beta_3$ )	<b>0.829</b>	0.185	0.00	<b>1.00</b>

588

589 **Table 2** Results of the Bayesian regression model that explains host growth rate.  
 590 Posterior probability represents the proportion of parameter estimates (MCMC samples)  
 591 assigned to be either negative or positive. Parameters with a posterior probability of >  
 592 0.95 are shown in bold. SE: standard error.

Effect	Estimate	SE	Posterior probability	
			Negative	Positive
Intercept	0.098	0.006	-	-
Body size	<b>-0.016</b>	0.006	<b>1.00</b>	0.00
Dispersal distance	-0.009	0.010	0.82	0.18
Infection	0.003	0.009	0.36	0.64
Size · Distance	<b>0.013</b>	0.007	0.05	<b>0.95</b>
Size · Infection	0.008	0.009	0.19	0.81
Infection · Distance	-0.008	0.010	0.81	0.19

593

594 **Figure captions**

595 **Fig. 1** Schematic representation of the life cycle of the freshwater mussel *Margaritifera*  
596 *laevis*. Glochidia released from female mussels infect the gills of masu salmon  
597 *Oncorhynchus masou masou* in the local habitat (local infection process). After a 40–  
598 50-day parasitic period, juvenile mussels detach from host fish and invade into  
599 unoccupied, parasite-free habitats through host dispersal (landscape-level process).

600

601 **Fig. 2** Plastic dispersal response of masu salmon *Oncorhynchus masou masou* to  
602 infection by larval parasites of *Margaritifera laevis*. Shaded areas with dotted lines  
603 denote average dispersal kernels. Solid and dashed lines indicate dispersal kernels for  
604 large (80<sup>th</sup> percentile body size) and small (20<sup>th</sup> percentile body size) fish hosts,  
605 respectively. Uninfected individuals showed little variation in dispersal (a). In contrast,  
606 infected individuals exhibited strong size-dependence in dispersal (b).

607

608 **Fig. 3** Size-specific effects of dispersal on host growth rates ( $\log(FL_{50}/FL_0)$ ). Solid and  
609 broken lines represent predicted values of the linear mixed effect model for large (80<sup>th</sup>  
610 percentile body size) and small (20<sup>th</sup> percentile body size) fish hosts, respectively.

611 Bubbles indicate individual fish and their sizes are proportional to fork length during the

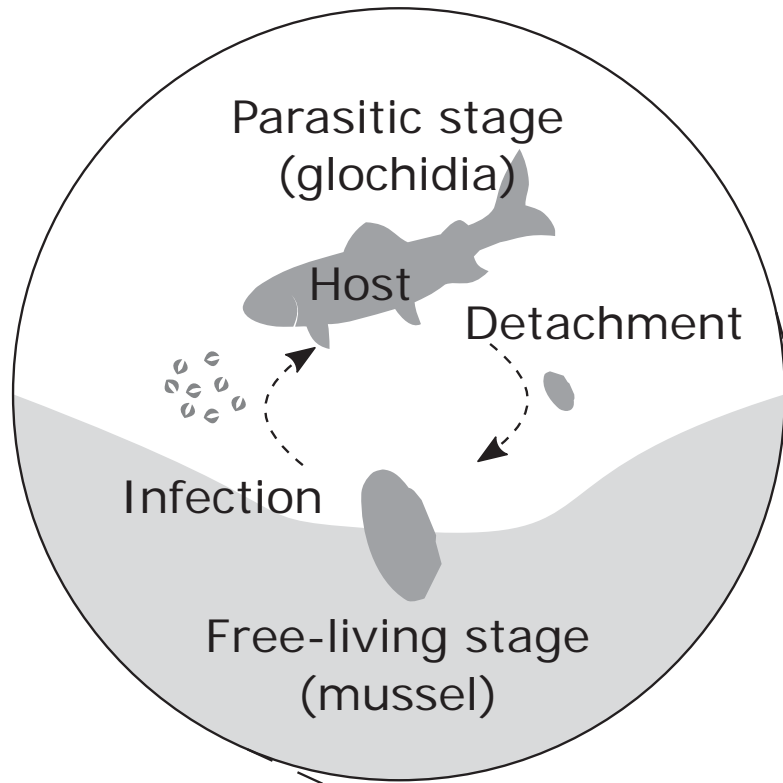
612 first capture session ( $FL_0$ ).

613

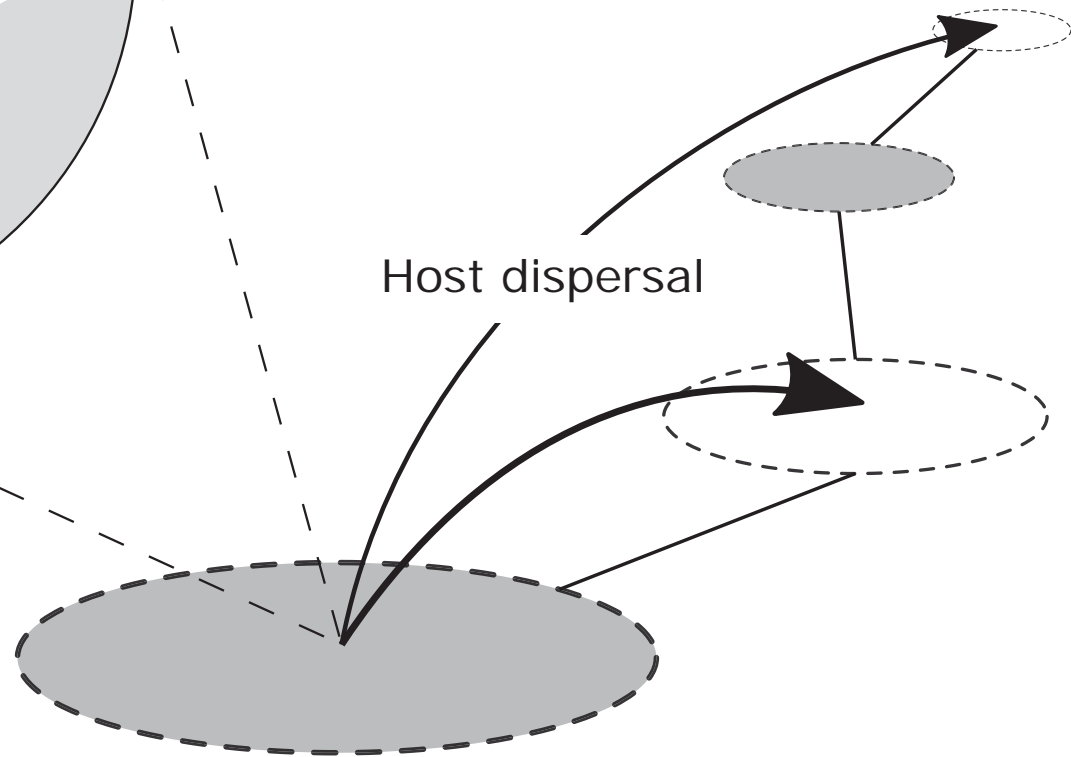
614 **Fig. 4** Contour plots of simulated parasite persistence times (a, b) and occupancy  
615 (proportion of parasite-occupied patch; c, d) in a 100-patch linear landscape with host  
616 carrying capacities of 4 (a, c) and 8 fish/patch (b, d). Brighter colors represent longer  
617 persistence time (a, b) or greater occupancy (c, d). Increasing values on the  $\gamma$  ( $x$ -axis)  
618 denote increasing average dispersal distance, whereas increasing values on the  $\delta$  ( $y$ -axis)  
619 represent stronger size-dependence in dispersal (larger individuals become more  
620 dispersive whereas smaller individuals become less dispersive). Other parameter values  
621 were as follows: colonization rate,  $C = 0.005$ ; extinction probability,  $E = 0.01$ ;  
622 environmental stochasticity in host population dynamics,  $\sigma_\varepsilon = 0.18$ ; and survival during  
623 dispersal,  $s = 0.87$ . Open and filled dots represent observed dispersal scenarios with  
624 weak ( $\gamma = \beta_0$ ,  $\delta = \beta_2$ ; see Fig. 1a) and strong ( $\gamma = \beta_0 + \beta_1$ ,  $\delta = \beta_2 + \beta_3$ ; see Fig. 1b) size-  
625 dependence, respectively. See Table 1 for estimated parameters.

626

# Local infection process



- Parasite-occupied patch
- Parasite-free patch

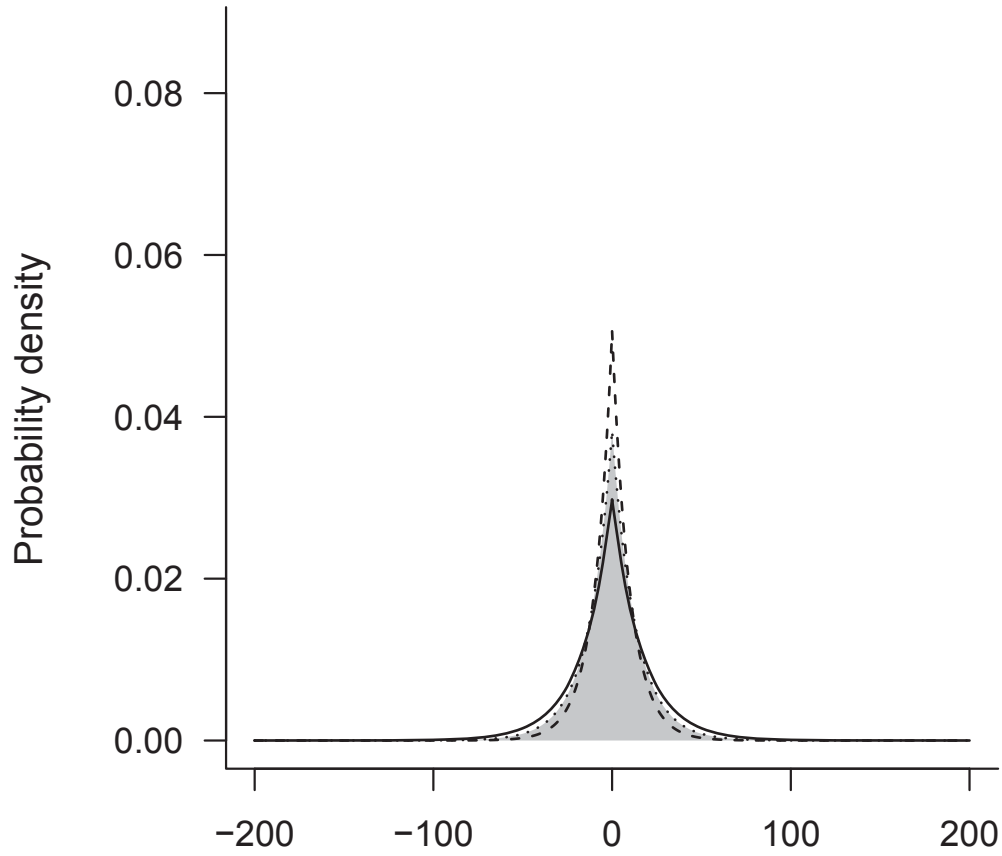


# Landscape-level process

Fig. 1 Terui et al.

Fig. 2 Terui et al.

(a) Uninfected



(b) Infected

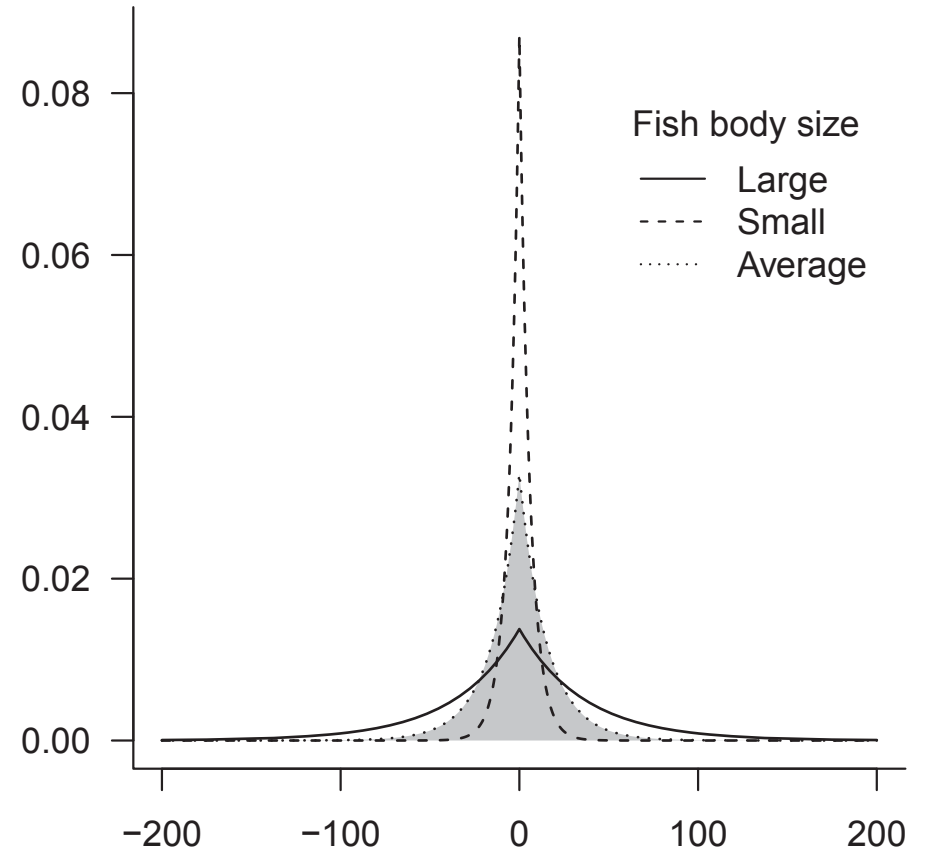


Fig. 3 Terui et al.

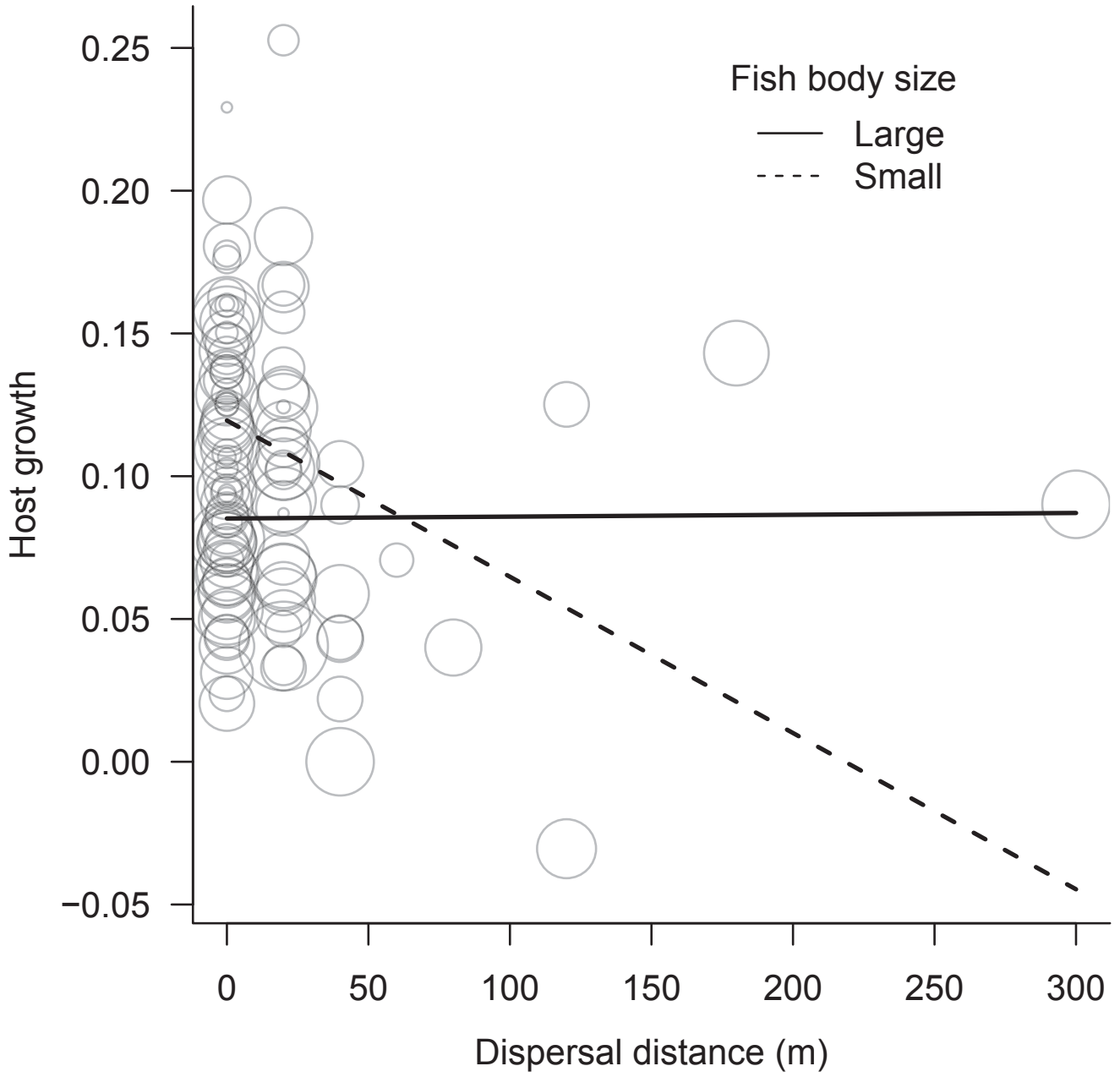




Fig.4 Terui et al.

