Genetic variation in dispersal plasticity in an aquatic host-parasite model system

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Abstract

Dispersal plays a main role in determining spatial dynamics, and both theory and empirical evidence indicate that evolutionary optima exist for constitutive or plastic dispersal behaviour. Plasticity in dispersal can be influenced by factors both internal (state-dependent) or external (context-dependent) to individuals. Parasitism is interesting in this context, as it can influence both types of host dispersal plasticity: individuals can disperse in response to internal infection status but might also respond to the presence of infected individuals around them. We still know little about the driving evolutionary forces of host dispersal plasticity, but a first requirement is the presence of a genetic basis on which natural selection can act. In this study, we used microcosm dispersal mazes to investigate plastic dispersal of 20 strains of the freshwater protist *Paramecium caudatum* in response to the bacterial parasite *Holospora undulata.* We additionally quantified the genetic component of the plastic responses, i.e. the heritability of state- and context-depended dispersal. We found that infection by the parasite can either increase or decrease dispersal of individual strains relative to the uninfected (state-dependent plasticity), and this to be heritable. We also found strain-specific change of dispersal of uninfected *Paramecium* when exposed to variable infection prevalence (context-dependent plasticity) with very low level of heritability. To our knowledge, this is the first explicit empirical demonstration and quantification of genetic variation of plastic dispersal in a host-parasite system, which could have important implications for meta-population and epidemiological dynamics. We discuss some of the underlying mechanisms of this variation and link our results to the existing theoretical models.

Introduction

In recent years the study of dispersal has received an increasing interest (Bowler & Benton, 2005; Ronce, 2007; Clobert *et al.*, 2009; Kubisch *et al.*, 2014). Understanding why animals move and disperse within a landscape has in fact become critical in a world driven by environmental changes (Parmesan & Yohe, 2003; Thomas *et al.*, 2004). Constitutive or plastic dispersal, broadly defined as the movement of individual between different habitat patches (Ronce, 2007), is a fundamental and complex trait driving metapopulation and spatial dynamics (Hanski & Hanski, 1999; Baguette *et al.*, 2012). Namely, plastic dispersal can be influenced by changes in the internal condition of the individual (state-dependent dispersal) or by environmental factors (context-dependent dispersal) (Clobert *et al.*, 2009).

State-dependent dispersal depends on the phenotypic variation of single individuals, so that they will be more or less propense to disperse or even migrate (Narayanan *et al.*, 2020) because of their internal condition (Clobert *et al.*, 2009). Sex (Greenwood, 1980), body size (Hanski & Woiwod, 1993) and condition (Binning *et al.*, 2017), and developmental stage (Bowler & Benton, 2005) can also play a role in influencing dispersal. Context-dependent dispersal is usually correlated to the environment the organisms live in. Here, individuals can decide to leave or stay in the patch by gathering information about the patch quality from some environmental cues characteristic of the patch itself (Clobert *et al.*, 2009; Fronhofer *et al.*, 2018). Cues may be linked to abiotic or spatial factors such as food availability (Massot & Clobert, 1995; Kim, 2000), patch isolation (Conradt *et al.*, 2001; Bowler & Benton, 2005) or patch size (Stamps *et al.*, 1987; Kindvall & Petersson, 2000), but also relate to biotic interactions (Kubisch *et al.*, 2014). Examples of these interactions are within species density (Harrison, 1980; Roland *et al.*, 2000; Poethke & Hovestadt, 2002; Bowler & Benton, 2005; Rodrigues & Johnstone, 2014), sex ratio (Lawrence, 1987, 1988), relatedness (Ronce *et al.*, 2001) as well as between species dynamics (Poethke *et al.*, 2010). Despite their ubiquity and impact on demographic dynamics and evolution, still little is known about how the interaction with parasites and predators affects dispersal plasticity, and what the consequences might be for epidemiology and co-evolution of host and parasite (May & Anderson, 1983; Poethke *et al.*, 2010; Binning *et al.*, 2017). As such, parasites effect on dispersal is of particular interest since parasitism can simultaneously influence dispersal by state-dependent or context-dependent plasticity.

State dependence is the simplest: the host can be either infected or uninfected, and thus the behaviour of the host can differ between infection status. The parasite can influence dispersal by changes in the host that can be morphological or physiological (Binning *et al.*, 2017). However, the outcome of the infection on dispersal behaviour is not straightforward. Parasites infections are usually costly and, as a consequence of host exploitation, they can decrease dispersal levels (Heeb *et al.*, 1999; Fellous *et al.*, 2011; Debeffe *et al.*, 2014; Horky *et al.*, 2014; Welicky & Sikkel, 2015; Norgaard *et al.*, 2019; Baines *et al.*, 2020). For example, the

freshwater fish *Squalius cephalus* parasitized by the larva of the bivalve *Anodonta anatine* show reduced movement and dispersal caused by an increased energetic demand in the immune system to fight off the infection (Horky *et al.*, 2014). Although considering the specific case of natal dispersal (i.e. dispersal from the natal to a non-overlapping adult range), a monitoring study of wild population of the roe deer *Capreolus capreolus*, found that parasite abundance of three different orders of worms (Strongylida, Trichocephalida, Ascaridida) reduced state-dependent dispersal (Debeffe *et al.*, 2014). Alternatively, dispersal behaviour can be positively correlated with infection (Brown & Brown, 1992; Suhonen *et al.*, 2010; Brown *et al.*, 2016; Shaw & Binning, 2016). For example, in the damselfly *Calopteryx virgo* (Suhonen *et al.*, 2010), males have been found to disperse more when the immune system was activated. Possibly, males need to forage more as a consequence of an activated immune system, and so they also move and disperse more in search for food. Other examples of increased dispersal can be found when parasites directly manipulate host behaviour to gain a fitness benefit (Curtis, 1993; Thomas *et al.*, 2002; Lion *et al.*, 2006; Martini *et al.*, 2015). The Asian citrus psyllid (*Diaphorina citri*) infected by *Candidatus* Liberibacter asiaticus show increased dispersal and parasite manipulated flight patterns (Martini *et al.*, 2015). Kin selection can also be a major driver in increasing dispersal. Individuals may leave their patch to prevent relatives to be infected, even if dispersal is not favoured by other conditions (Hamilton & May, 1977; Poethke *et al.*, 2010; Iritani & Iwasa, 2014; Iritani, 2015; Deshpande *et al.*, 2020). In other cases, parasites do not seem to affect dispersal behaviour of their hosts (Mayer *et al.*, 2015; Nelson *et al.*, 2015; Csata *et al.*, 2017; Taggart *et al.*, 2018). In Australian Sleepy Lizards (*Tiliqua rugosa*) infected by ticks, dispersal is unaffected, potentially because *T. rugosa* has adapted to some level of parasite load in its natural system (Taggart *et al.*, 2018).

Context-dependent dispersal can happen in response to the presence of natural threats in the population, as shown by theoretical (Poethke *et al.*, 2010) and empirical work (de la Peña & Bonte, 2014; Otsuki & Yano, 2014). In a model for a predator-prey system, Poethke *et al.* (2010) predict that, for dispersal plasticity to be selected for, the presence or absence of predators need to have a high spatio-temporal correlation (i.e. the patch future conditions need to be highly predictable). In this way, the prey can take the appropriate "decision" to leave or stay in the patch. This condition is also found in a model considering host-parasite context-dependent plasticity (Deshpande *et al.*, 2020). A recent study analysing multiple taxa of invertebrates and vertebrates showed that chemical predator-related cues can induce dispersal, along with alterations in resource availability (Fronhofer *et al.*, 2018). It is likely that such cues are relevant also in host-parasite models, where strategies of infection-avoidance behaviour by uninfected individuals are well known (Behringer *et al.*, 2006; Beltran-Bech & Richard, 2014; Curtis, 2014; Lopes *et al.*, 2016; Stroeymeyt *et al.*, 2018). However, empirical studies that explicitly consider host dispersal plasticity as a function of parasite density are rare (French & Travis, 2001).

Dispersal and dispersal-related traits have a genetic basis, as reviewed extensively by Saastamoinen *et al.*, 2018, and they can rapidly evolve (Phillips *et al.*, 2006; Taylor & Buckling, 2011; Weiss-Lehman *et al.*, 2017; Zilio *et al.*, 2020). However, also plastic responses such as dispersal plasticity have a genetic basis underlined by additive genetic components which could respond to selection (de Jong, 2005; Pigliucci, 2005; Garland & Kelly, 2006; Laitinen & Nikoloski, 2019). Reinforcing this idea, a recent meta-analysis highlighted the crucial role of plasticity relative to genetic differentiation in determining phenotypic divergence between populations (Stamp & Hadfield, 2020). Thus, plastic level of dispersal can be expected to readily evolve in response to biotic pressures and environmental changes. To date, only very few studies have investigated whether this requirement is met for state- and context-dependent dispersal plasticity. In fact, how plastic dispersal varies between different genotypes due to a parasite challenge is rarely evaluated in empirical studies (Suhonen *et al.*, 2010; Fellous *et al.*, 2011), or the genetic diversity is treated as a random effect (Csata *et al.*, 2017). Also, the number of strains evaluated is usually small, making it difficult to draw any strong conclusion on the genetic component of plastic dispersal of infected hosts.

In this study, using microcosm dispersal mazes, 20 strains of *P. caudatum* were tested for dispersal in the presence and absence of its parasite, the bacterium *Holospora undulata*. From previous studies on this protist it was observed that parasitic infection reduces dispersal (Fellous *et al.*, 2011). The objective of this study was to test whether this negative effect was general, or whether strains varied in infection-state dependent dispersal. Variation in context dependency was investigated by comparing the dispersal of uninfected hosts over a range of population-level infection prevalence. Inspection of this natural variation in dispersal plasticity and the estimation of heritability, allowed us to make projections as to whether these traits may respond to parasite-mediated selection.

Materials and methods

Study system

Paramecium caudatum is a freshwater filter-feeding protist commonly found in stagnant waters of the Northern hemisphere (Wichterman, 2012). Like all ciliates, paramecia have a macronucleus for somatic gene expression and a germ-line micronucleus, used for sexual reproduction. *Holospora undulata* is a gram-negative alpha-proteobacterium that infects the micronucleus of *P. caudatum* (Fokin, 2004). It can be transmitted vertically when the host divides or horizontally at host death. Infectious spores are immobile and therefore rely on host movement or water current for their own dispersal. Infection reduces *P. caudatum* survival (Restif & Kaltz, 2006) and dispersal (Fellous *et al.*, 2011; Nørgaard *et al.*, 2020).

Experimental procedure

In this experiment we assessed dispersal of 20 strains of *P. caudatum* from different geographical regions

(provided by S. Krenek, TU Dresden, Germany; Table S1, Supplementary Information). Each strain was infected with an inoculum of *H. undulata,* prepared from a mix of infected stock cultures in the lab. All infections in these stock cultures originate from a single isolate of *H. undulata* established in 2001 and serves as the reference genome for this species (Dohra *et al.*, 2013). The 20 strains were grown as mass cultures and then divided into two blocks, each consisting of three assay replicates per strain (20 strains x 2 blocks x 3 assay replicates = 120 replicates). Four days after infection, the prevalence of infected individuals in each population was measured to test if the infection by *H. undulata* had established in the tube. In parallel, three uninfected controls for each strain were maintained (total of 180 replicates). Using the methods described in Nørgaard et *al* (2020), dispersal of infected and uninfected control replicates was tested three weeks post-infection, when population size (mean: 190 mL^{-1} ± 9 SE; 95% range [172; 208]) and infection prevalence (mean: 26.8 % ± 2.1; 95% range [3.1; 90.7]) had settled naturally in each experimental replicate. Shortly, the dispersal arena consisted of three 50-mL Falcon tubes, one in the middle connected to two lateral tubes. The connection between tubes could be opened or closed by the experimenter. Each tube was filled with 25 mL of fresh medium so that both connections were established. The connections were then blocked and ~20 mL of culture containing infected or uninfected control were put in the middle tube. The lateral tubes received 20 mL of *Paramecium*-free medium. Connections were then opened, and the *Paramecium* allowed to disperse to the lateral tubes. After 3h, the connections between tubes were closed. Samples were taken from the middle tube (500 µl) and the combined lateral tubes (3 mL). We then counted the number of individuals (dissecting microscope, 40x) and made lacto-aceto-orcein fixations (Görtz & Wiemann, 1989) of the *Paramecium* from infected replicates to determine their infection status (Phase contrast, 1000x). From the cell counts and the information on infection status, we estimated the total population density and infection prevalence (i.e., what was added to the middle tube at the beginning of the assay), as well as the proportion of infected and uninfected dispersers for each replicate (referred to as per-3h 'dispersal rate' or dispersal, hereafter). Furthermore, a swimming behaviour assay was performed. From each strain (infected and control replicates), 1 infected and 1 uninfected individual were isolated, and then grown in a 2mL Eppendorf for 8 days. The resulting 40 monoclonal cultures (20 strains x 2 infection status) were then checked to confirm the infection status. Swimming behaviour was assayed by placing 200-µL samples (containing 10-20 individuals) on a microscope slide and recording individual movement trajectories (Perfex SC38800 camera; 15 frames per second; duration 10 s). For each sample, average swimming speed (µm/s) and swimming tortuosity (standard deviation of the turning angle distribution, describing the extent of swimming trajectory change) were determined using video analysis with the “BEMOVI” package (Pennekamp *et al.*, 2015).

Statistical analysis

All the statistical analysis was conducted in R version 3.6.3 (R Core Team, 2019). To analyse variation in dispersal we used generalized linear mixed effect models (GLMMs) with binomial error distribution (logit

link) of the "lme4" (Bates *et al.*, 2015) and "car" (Fox & Weisberg, 2019) package.

For state-dependent dispersal, we compared the dispersal of the infected fraction (from infected replicates) with the dispersal in the uninfected control replicates. Using the completely uninfected control replicates as the reference (rather than the uninfected fraction in infected replicates) avoided any confounding effects that may arise from context-dependent dispersal of uninfected individuals in the infected tubes. The explanatory fixed factors were *Paramecium* strain identity, infection status (infected or uninfected control) and the strain x infection status interaction. Experimental block was considered as random factor. An overall effect of state-dependent plasticity would be indicated by a significant effect of infection status, and the genetic basis for plasticity by a significant strain x infection status interaction.

For context-dependent dispersal, we analysed the dispersal of uninfected *Paramecium* from the infected replicates. One strain (C105) was removed due to the lack of replication. The explanatory fixed factors were strain identity, infection prevalence and the strain x infection prevalence interaction. A significant effect of infection prevalence would indicate general context-dependent dispersal plasticity, while a significant strain x infection prevalence interaction would indicate genetic variation in this plasticity. Finally, we also added population density as a covariate to take into account this potential additional type of context-dependency (Fellous *et al.*, 2012; Deshpande *et al.*, 2020), and we considered experimental block as random factor. We excluded 11 replicates from the state- and context-dependent analysis because no infection was detected.

We quantified the heritability of our traits of interest using the following procedure for both state- and context-dependent dispersal (two identical, independent analysis). Given the non-Gaussian nature of the traits and to do not overestimate heritability, we chose a Bayesian framework to run the quantitative genetic models (de Villemereuil *et al.*, 2016, 2018). We used the "MCMCglmm" package (Hadfield, 2010) with binomial variable distribution. To obtain variance component estimates, state- and context-dependent dispersal variance was partitioned into four and five random effects respectively, corresponding to the explanatory factors of our GLMMs. The MCMC chains were run over 1 million iterations (initial burning = 10.000 iterations, thinning = 1000 iterations), and to obtain posterior distribution estimates from the data (Morrissey *et al.*, 2014) we specified parameter expanded priors (V = 1, nu = 0.02). From the obtained values, we used the specifically designed "QGparams" function for non-Gaussian traits in the "QGglmm" package (de Villemereuil *et al.*, 2016) and calculated narrow sense heritability of the plastic response from the formula $h^2 = V_A / V_P$. Heritability corresponded to the relative contribution of the additive genetic variance of the interaction term (i.e. V_A; strain x infection status interaction for the state-, and strain x infection prevalence interaction for the context-dependent dispersal) to the sum of all variance components (V_P).

The data from the video analysis were used to link behavioural traits to observed levels of dispersal. First, we ran two separate multiple linear regressions to test if the measured behavioural traits (average swimming

speed and average swimming tortuosity) varied as a function of strains and status. Second, we tested for correlations between these two swimming traits and mean dispersal (observed for each strain x infection status combination). Only 17 of the 20 strains were analysed due to isolated replicates not reproducing and thus missing data.

Results

State-dependent dispersal

We observed strong differences in state-dispersal among strains (χ^2 = 133.2, df = 19, $p < 0.001$), ranging from 1% (SE ± 0.003) to 33% (SE ± 0.03). The analysis further revealed a marginally significant effect of infection status (χ^2 = 2.9, df = 1, p= 0.086), even though the overall levels of infected and uninfected dispersal were very similar (average infected: 13.4 % SE ± 2.4; average uninfected control: 12.0 % SE ± 2.7). We found a genetic basis for state-dependent dispersal with the strains having different levels of plasticity in response to the infection (Figure 1A). The interaction between strain and infection status on dispersal was indeed highly significant (χ^2= 64.7, df= 1, $p < 0.001$). Confirming how such plastic response in state-dependent dispersal could respond to selection and evolve, the heritability of the interaction between strain and status was 8.58% (95 % CI [0.0006; 0.17103]) and explained almost a third of the model variance (r^2 = 0.32). The differences in dispersal between uninfected control and infected groups in Figure 1B highlights how parasite infection had different effects depending on the strain, and therefore the genetic identity of the host. In 4 out of the 20 strains, infection and dispersal were clearly negatively correlated (Figure 1B, strains above 0 on the left having higher dispersal when uninfected than infected). For three strains, infection and dispersal were clearly positively correlated, while strains overlapping 0 (Figure 1B) show little to no difference in state-dependent dispersal plasticity.

Context-dependent dispersal

The strains had significant differences in their context-dependent dispersal levels (χ^2 = 94.7, df = 18, $p <$ 0.001), which ranged from 2% (SE ± 0.04) to 38% (SE ± 0.01). However, the strains reacted differently across infection prevalence gradients (infection prevalence x strain interaction: χ^2= 28.9, df = 18, p = 0.049), with either higher or lower dispersal at increased level of infection in the population, as showed by the different slopes in Figure 2A and 2B. Although marginally significant, the interaction term explained 23% of the model variance and had a very low heritability ($h^2 < 0.001$, 95% CI [10^{-7}, 10^{-4}]). Neither the main effect of infection prevalence (χ^2 = 0.9, df = 1, p = 0.348) nor population density (χ^2=0.5, df = 1, p = 0.497) affected the dispersal of uninfected *Paramecium*; adding or removing this latter term from the model did not change the results (χ^2=0.45, df = 1, p = 0.497).

Swimming behaviour

Analysis of swimming speed revealed a significant effect of strain identity ($F_{16,16} = 2.5$, $p = 0.038$) and infection status ($F_{1,16} = 40$, $p < 0.001$). Namely, mean swimming speed was higher in uninfected control groups (mean 677 µm s^{-1}, ± 238 SE) compared to infected (360 µm s^{-1}, ± 135 SE) (Figure 3). Swimming tortuosity was not significantly affected by strain or infection status ($p > 0.5$). Neither swimming speed ($F_{1,31} = 0.53$, $p = 0.47$) nor swimming tortuosity ($F_{1,31} = 0.51$, $p = 0.48$) were good predictors of dispersal.

Discussion

Here, we investigated the amount of dispersal plasticity and its genetic basis in an experimental host-parasite model system. We found that different host strains express different levels of both state- and context-dependent dispersal in response to parasite infection and parasite prevalence in the population. In other terms, the correlation of dispersal plasticity with the infection status or infection prevalence depended on the host strain. Interestingly, state-dependent dispersal showed significant additive genetic variance and heritability, whereas context-dependent dispersal had a low genetic component. These results indicate a genetic basis for parasite-related dispersal plasticity that could be selected upon. This may lead to the evolution of complex dispersal phenotypes and reaction norms, with overall consequences for patterns of local adaptation, epidemiology and metapopulation dynamics.

State-dependent plasticity

In our analysis we observed that the dispersal outcome was affected by the identity of the host strains (Figure 1A-B). Previous studies reported that infection by *H. undulata* reduced dispersal in *P. caudatum* (Fellous *et al.*, 2011; Nørgaard *et al.*, 2020), which was related to a reduction in survival and reproduction. The explanation for a reduced dispersal can easily be connected to the negative effect that a parasite may have on its host locomotory ability (Horky *et al.*, 2014; Binning *et al.*, 2017). Infection may cause direct mechanical and physiological damage to its host (virulence). In fact, the host has to face the energetic demand of mounting an immune response and clear (or resist) the infection, reducing the potential to disperse. Further, the parasite may steal host resources (Mideo, 2009), and dispersal may become more costly (Lopes, 2014; McElroy & Buron, 2014; Risely *et al.*, 2018). The observed positive correlation of some strain is less intuitive. The theoretical model of Deshpande *et al.* (2020), predicts that positive parasite induced state-dependent dispersal can evolve as a consequence of kin selection. Dispersal of infected individuals is promoted to prevent relatives to be affected. Also, we cannot exclude that increased dispersal is a consequence of parasite manipulation to enhance its own growth, reproduction and transmission (Lion *et al.*, 2006; Martini *et al.*, 2015; Binning *et al.*, 2017). Higher level of host dispersal may allow the parasite to infect not only at the local scale, but also to encounter new suitable habitats and host populations and spread globally (Kamo & Boots, 2006). Still, we do not know whether the plasticity we observed is an adaptive response.

Context-dependent plasticity

In a recent meta-experiment, Fronhofer et al. (2018) demonstrated that context-dependent dispersal is driven by chemical predator signals in various organisms, including *P. caudatum*. It is therefore reasonable to think that this applies to infection-driven context-dependent dispersal in this system, in line with our results. However, similarly to the state-dependent analysis, the effect of infection prevalence on dispersal was strain specific, with a small number of strains showing a negative response to infection prevalence. This highlight the presence of a genetic basis for context-depend dispersal. Theoretical models (Deshpande *et al.,* 2020; Poethke *et al.,* 2010) predict that, for the prey or parasite to make the appropriate "dispersal decision" to leave or stay in the patch, there needs to be high predictability about the environmental future conditions. More specifically, in a host-parasite system, prevalence could be a good predictor of a patch future condition only at high parasite virulence (Deshpande *et al.*, 2020). In our system, *Holospora undulata* generally reduces survival and reproductive success of *P. caudatum* (Restif & Kaltz, 2006; Nørgaard *et al.*, 2020), which is also reflected by differences in population density between uninfected control and infected microcosms in the present experiment (density uninfected control: 288 mL^{-1}, ± 103; infected: 139 mL^{-1}, ± 91 SE). Hence, we might speculate that some strains could have had an evolutionary history with a highly virulent parasite in the wild. Population density is considered one of the most prominent cues for context-dependent dispersal (Harrison, 1980; Bowler & Benton, 2005; Rodrigues & Johnstone, 2014), and density may even be used as a proxy cue for infection prevalence (Deshpande et al 2020). For example, a high infection prevalence in a patch may be expected to be associated with low density, and *vice versa*, making density a "mirror cue" of prevalence. Population density has been also reported to influence dispersal in *P. caudatum* (Fellous *et al.*, 2012). However, in our study we did not find evidence for such density dependence. More controlled experimental setups may be employed to elucidate this question, with artificially manipulated infection prevalence and host densities (via controlled mixing of infected and uninfected individuals prior to dispersal, for example along a prevalence and/or density gradient). To investigate in more detail the underlying nature of the dispersal cue, we may envisage the use of filtered parasite inocula, in order to leave only potential chemical cues to influence the dispersal decision of *Paramecium* (see Fronhofer *et al.*, 2018).

Swimming behaviour

We observed reduced swimming speed in the infected groups (Figure 3), which is a frequently observed outcome of parasitic infections (McElroy & Buron, 2014; Binning *et al.*, 2017). In *P. caudatum*, a negative correlation between speed and dispersal has been previously observed (Zilio *et al.*, 2020). Reduced speed could also have the side effect of reducing predator avoidance. However, we did not observe any clear correlation between dispersal and swimming speed or tortuosity in our study. Thus, it is difficult to infer any clear mechanistic explanation for the different dispersal rates measured. It is possible that we did not consider some other aspect of swimming behaviour that may be influenced by the presence of *H. undulata.*

For example, some parasites are known to affect the position of its host in the water column (Cezilly *et al.*, 2000). Although this is yet to be formally tested in this system, this could have influenced the ability of *P. caudatum* to find the connection corridor between the test tubes in the dispersal arena.

Is plasticity selected for?

We observed genetic variation in both state-dependent and context-dependent dispersal plasticity, however context-dependent dispersal presented low heritability compared to state-dependent. Under parasite-mediated selection, we may therefore expect little evolutionary response of context- compared to state-dependent dispersal. Only state-dependent dispersal seems to have the genetic potential for the evolution of plastic dispersal phenotypes and reaction norms.

Heritability values range broadly in wild and laboratory populations (Mousseau & Roff, 1987; Weigensberg & Roff, 1996; McFarlane *et al.*, 2014; Salles *et al.*, 2020). The low estimated value of context-depend dispersal may reflect different causes apart from low additive genetic variance, large environmental or residual effects. If the trait is linked to fitness (avoiding the risk of infection), directional selection is expected to erode genetic variation with corresponding low heritability (Kruuk *et al.*, 2000). Thus, the trait may have already been selected under different circumstances (e.g. high or low parasite prevalence), leading to the observed pattern. Alternatively, low heritability may occur when the trait has a complex genetic architecture, as it is likely for context-depend dispersal which includes many physiological and behavioural components. As a result, the co-variation of additive genetic and residual variance may suffer from a lack of power and limit direct effects on heritability (Stirling *et al.*, 2002). Genetic architecture can influence evolutionary outcomes (Holloway *et al.*, 1990), and the effect of a parasite on the host could also depend on its past evolutionary history. Evolved resistance or tolerance to some level of parasitic infection can explain why, in some cases, we do not see any effect of infection on dispersal (Taggart *et al.*, 2018). In our data, we have an estimate of resistance in the form of parasite prevalence at 4 days after infection (Table S1). However, it seems that there is no correlation between resistance and the difference in dispersal (data not shown). Yet, the strains that we used in our analysis come from many different geographical regions (Table S1), and there might be some aspects that we are unaware of, that cause some strains to be less subject to the effect of parasite infection on dispersal. Seminal work on our *Paramecium-Holospora* system illustrated the presence of different compatibilities between parasite isolate and host clones (Skoblo *et al.*, 1996), and the potential for variation and evolution of resistance. Still, as mentioned before, explanation different from an adaptive perspective might apply.

Understanding the possible trade-offs between dispersal plasticity and other traits could be crucial to predict plasticity evolution. In fact, plasticity seems to be more important (twice as much) compared to genetic

345 differentiation in causing adaptive phenotypic and spatial divergence between populations (Stamp &
346 Hadfield, 2020). Also, the absolute values of dispersal and plasticity could play a role in determining
347 evolutionary outcomes; individuals with high plasticity but low dispersal could be disadvantaged against a
348 parasite, while individuals with high dispersal but low plasticity could be favoured. On an epidemiological
349 side, different plasticity levels could influence disease spatial dynamics. This may lead to non-intuitive
350 predictions and feedbacks, as shown by Deshpande et al (2020).
351

352 ## Conclusions

353 Dispersal is crucial in determining patterns of local adaptation, epidemiology and metapopulation dynamics
354 (Hamilton & May, 1977; Hanski & Hanski, 1999; Ronce *et al.*, 2001; Baguette *et al.*, 2012). In this study we
355 showed genetic variation in both state-dependent and context-dependent parasite-driven dispersal
356 plasticity, with the two traits potentially under selection. However, we do not know how much of the
357 observed variation between our strains is adaptive. An interesting follow up study could be to test how
358 plasticity changes after the strains experience different selective pressures. Further work is needed to help
359 better comprehend the main drivers of plasticity, especially for context-dependent dispersal, which, in host-
360 parasite systems, remains poorly understood. Our study provides the first empirical demonstration of the
361 genetic basis of dispersal plasticity in a host-parasite system.
362

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367 ## Author contributions

368 GZ, LN, NZ and OK conceived the study. GZ, LN, NZ, CGB and OK performed the experimental work. GZ, GP,
369 and OK performed the statistical analysis. All authors interpreted the results. GZ, GP and OK wrote the first
370 draft of the manuscript and all authors commented on the final version.

371 ## Competing interests

372 The authors declare no competing financial interests.

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608 # Figures

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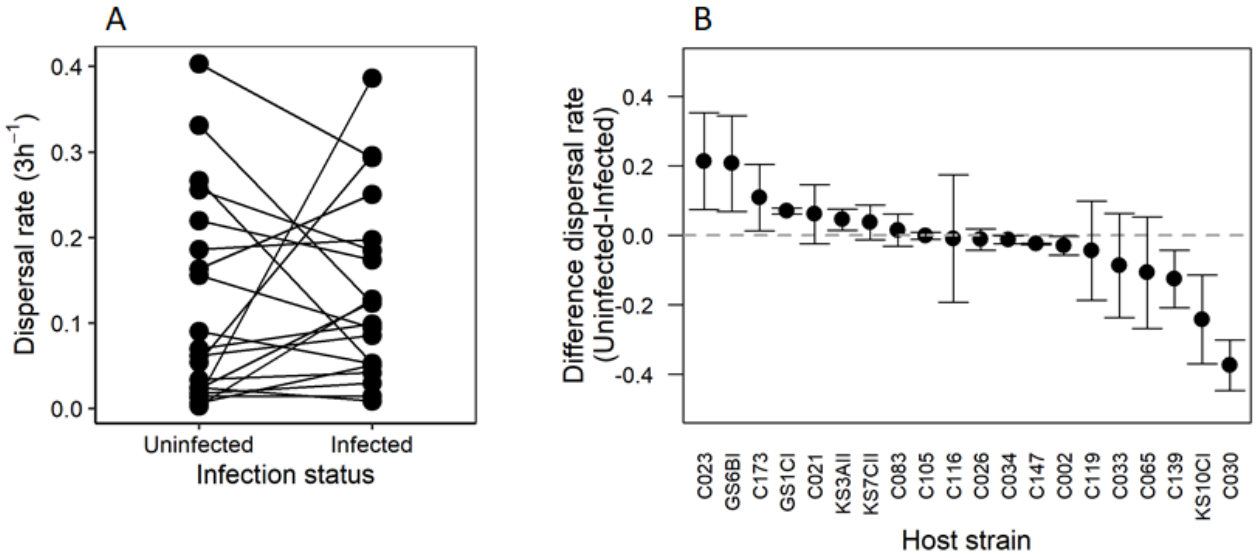

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611 **Figure 1.** (A) State-dependet dispersal of infected and uninfected host strains. Each point represent the mean dispersal
612 value of a specific strain, the lines connect the the two infection status of the same strain. (B) Difference in dispersal
613 rate between uninfected and infected populations. On the vertical axis is the difference between the mean percentage
614 of dispersal in the uninfected and the infected group for each strain (black points ± SE). Strains with mean values above
615 0 (dashed grey line) have higher dispersal when uninfected, whereas strains with mean values below 0 have higher
616 dispersal when infected.

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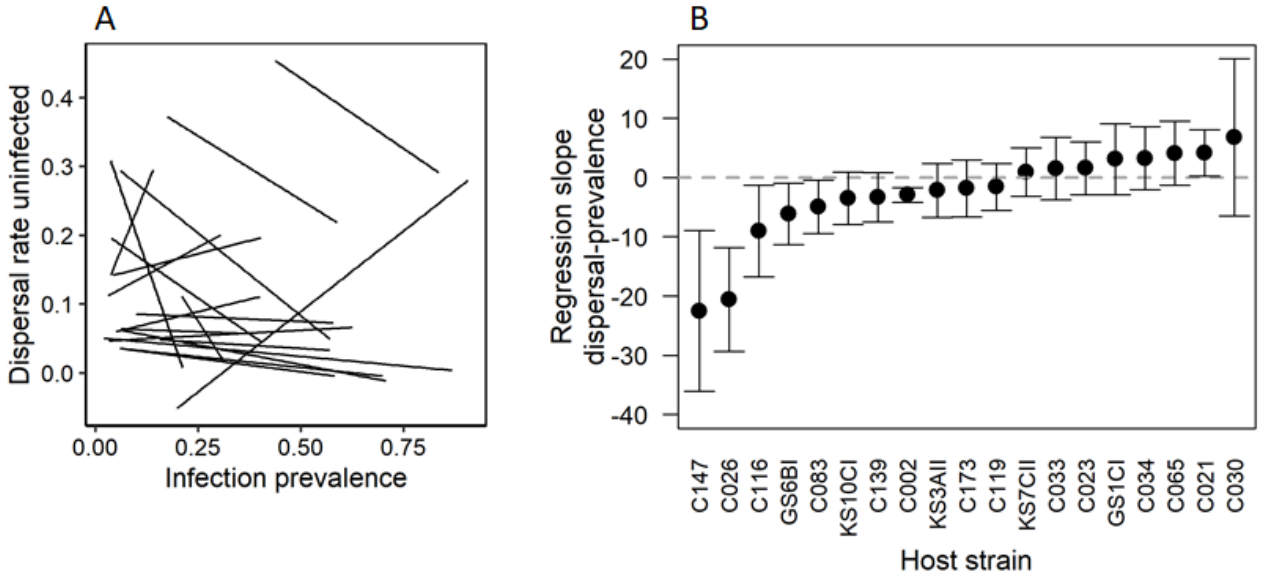

618

619 **Figure 2.** (A) Context-dependet dispersal of uninfected host strains in response to different level of parasite infection
620 prevalence in the population. Regression lines are calculated separately for each strain, (B) Slopes from the model for
621 each strain calculated in logit (black point ± SE). Positive or negative slopes (above or below 0, dashed grey line), indicate
622 a higher or lower dispersal in response to increasing presence of infected hosts.

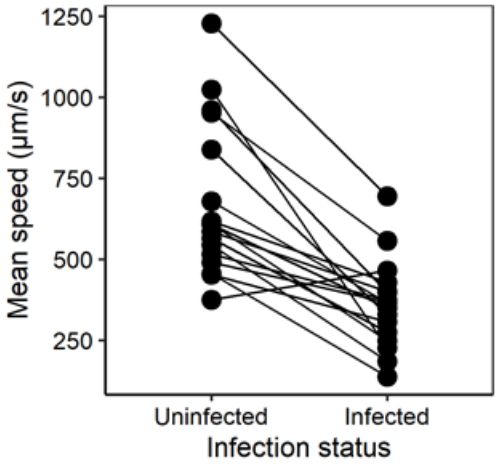

Figure 3. Swimming speed of infected and uninfect strains of *P. caudatum*. Each point represent the mean speed per strain, the line connects the different infection status for each strain.

Supplementary Information

Table S1. Host strain identity, country of origin and infection prevalence (number of infected individuals) at day 4 post inoculation.

Strain	Country	Day 4 infection prevalence
C002	Germany	0.73
C021	Germany	0.87
C023	Germany	0.66
C026	USA, Louisiana	0.62
C030	Austria	0.81
C033	China	0.2
C034	Germany	0.39
C065	Sweden	0.23
C083	USA, Indiana	0.18
C105	Spain	0.14
C116	France	0.57
C119	Peru	0.89
C139	Japan	0.32
C147	Japan	0.56
C173	Greece	0.73
GS1CI	Germany	0.67
GS6BI	Germany	0.81
KS10CI	Germany	0.76
KS3AII	Germany	0.81
KS7CII	Germany	0.74

Table S2. ANOVA results from GLMM models for (a) state- and (b) context-dependent dispersal. Block was always included as random factor.

(a) State-dependent dispersal				
Random effect:	Var±SD:			
Block	0.07±0.26			
Fixed effects:	*d.f.*	χ^2	p	
Strain	19	133.2	<0.001	***
Infection status	1	2.9	0.086	.
Strain x Infection status	19	64.7	<0.001	***

(b) Context-dependent dispersal				
Random effect:	Var±SD:			
Block	0±0			
Fixed effects:	*d.f.*	χ^2	p	
Strain	18	94.7	<0.001	***
Infection prevalence	1	0.9	0.348	
Population density	1	0.5	0.497	
Strain x Infection prevalence	18	28.9	0.049	*

Table S3. ANOVA results from linear models analysing swimming behaviour, (a) speed, (b) tortuosity and (c) their correlation with dispersal.

(a) Speed				
	d.f.	F	p	
Strain	16	2.4	0.038	*
Infection status	1	39.9	<0.001	***
(b) Tortuosity				
	d.f.	F	p	
Strain	16	0.44	> 0.5	
Infection status	1	0.06	> 0.5	
(c) Dispersal				
	d.f.	F	p	
Speed	1	0.8	0.373	
Tortuosity	1	0.2	> 0.5	