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Research

Fragmentation and the context-dependence of dispersal syndromes: matrix harshness modifies resident-disperser phenotypic differences in microcosms

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Habitat fragmentation, the conversion of landscapes into patchy habitats separated by unsuitable environments, is expected to reduce dispersal among patches. However, its effects on dispersal should depend on dispersal syndromes, i.e. how dispersal covaries with phenotypic traits, because these syndromes can drastically alter dispersal and subsequent ecological and evolutionary dynamics. Our comprehension of whether environmental factors such as habitat fragmentation generate and/or modify dispersal syndromes (i.e. conditional dispersal syndromes) is therefore key for biodiversity forecasting. Here we tested whether habitat fragmentation modulates dispersal syndromes by experimentally manipulating matrix harshness, a critical feature of habitat fragmentation, in ciliate microcosms. We found evidence for dispersal syndromes involving multiple traits linked to morphology (elongation and size), movement (velocity and linearity) and demography (growth rate and maximal population density). More importantly, these syndromes were modified by matrix harshness, with increased differences between residents and dispersers in morphology and movement traits, and decreased differences in growth rate as the matrix became increasingly harsh. Our findings thus reveal that habitat fragmentation can mediate the intensity and form of dispersal syndromes, a context-dependence that could have important consequences for ecological and evolutionary dynamics under environmental changes.

Keywords: dispersal, fragmentation, informed decision, metapopulation, phenotypic plasticity

Introduction

Habitat loss and fragmentation caused by human activities are considered major threats to biodiversity (Fahrig 2003, Haddad et al. 2015, Newbold et al. 2015). In addition to reducing the overall quantity of habitat, the conversion of natural ecosystems into agricultural or urban areas turns landscapes into increasingly patchy habitats separated by unsuitable environment (Fahrig 2003, Cote et al. 2017). As a consequence, habitat fragmentation is expected to increase the costs of dispersal and reduce movements among patches (Travis and Dytham 1999, Schtickzelle and Baguette 2003,

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Fahrig 2007, Delgado et al. 2014, Haddad et al. 2015, Cote et al. 2017). Since dispersal plays a major role in ecological and evolutionary dynamics (Hanski 1998, Lenormand 2002, Bowler and Benton 2005, Ronce 2007, Abbot et al. 2011, Clobert et al. 2012, Jacob et al. 2017), our ability to forecast population and species responses to environmental changes crucially depends on our understanding of how habitat fragmentation affects dispersal (Olivieri et al. 1990, McPeek and Holt 1992, Travis 2001, Duputié and Massol 2013, Cote et al. 2017).

Increasing empirical evidence for extensive within population variability in dispersal is causing a re-evaluation of the simplifying assumptions of uniform dispersal in current dispersal theory (reviewed by Bowler and Benton 2005, Ronce 2007, Edelaar et al. 2008, Clobert et al. 2012, Travis and Dytham 2012, Travis et al. 2012, Jacob et al. 2015a). Indeed, dispersal often depends on a variety of internal factors (i.e. phenotype-dependent dispersal) and environmental conditions (i.e. context-dependent dispersal; Holt 1987, Bowler and Benton 2005, Cote and Clobert 2007, Clobert et al. 2009, Cote et al. 2010, 2017, Schtickzelle et al. 2012, Stevens et al. 2014). This dependence of dispersal to multiple factors can lead to dispersal syndromes, defined as the covariation of dispersal with other phenotypic traits (Cote and Clobert 2007, Fjerdingstad et al. 2007, Clobert et al. 2009, Cote et al. 2010, 2017). Such covariation might result from genetic correlations or parallel selective responses to environmental factors between dispersal and phenotypic traits, which both lead to more dispersive individuals having different phenotypic traits compared to residents (Ronce and Clobert 2012, Stevens et al. 2014). Traits associated with dispersal might otherwise change in a labile way according to the environmental conditions encountered (i.e. phenotypic plasticity; Clobert et al. 2001, Bowler and Benton 2005, Benard and McCauley 2008, Cote et al. 2017). Labile changes might, for instance, occur through adaptive phenotypic plasticity where individuals change their phenotype when dispersing in order to increase the success of dispersal. Dispersal syndromes can have an important impact on evolution through spatial selection (Stevens et al. 2014, Cote et al. 2017). For instance, newly colonized habitats after local extinction or during range expansion will mostly be occupied by dispersive individuals which may have specific dispersal syndromes (Haag et al. 2005, Phillips et al. 2006, Niitepõld et al. 2009, Shine et al. 2011). This process that can speed up species range shifts and expansions (Thomas et al. 2001, Phillips et al. 2006, 2010, Shine et al. 2011, Lombaert et al. 2014, Cote et al. 2017). Furthermore, dispersal syndromes have been suggested to increase metapopulation size and persistence (Shima et al. 2015). More recently, Jacob et al. (2019a) demonstrated that variability in dispersal syndromes can be as important for metapopulation dynamics as spatiotemporal variability in environmental conditions.

Theoretical and empirical studies have pointed out that such multi-dependency of dispersal can change the predictions of dispersal consequences for ecological and evolutionary dynamics. For instance, dispersal syndromes can affect the dynamics of species range shifts (Phillips et al. 2006, 2010, Duckworth and Badyaev 2007, Shine et al. 2011), population genetic structure (Edelaar et al. 2008, Jacob et al. 2015a, 2017, Nicolaus and Edelaar 2018), evolution of ecological specialization (Holt 1987, Holt and Barfield 2015, Jacob et al. 2017) or metapopulation responses to habitat fragmentation (Hanski 1999, Hanski and Gaggiotti 2004, Clobert et al. 2009, Bonte et al. 2012, Cote et al. 2017). This makes our comprehension of the causes and consequences of variability in dispersal syndromes critical for biodiversity forecasting, especially in light of the increasing fragmentation of habitats (Travis and Dytham 2012, Travis et al. 2012, Haddad et al. 2015, Santini et al. 2016, Cote et al. 2017). Habitat fragmentation should indeed modify the selective pressures acting on dispersal and dispersal-related traits and therefore might generate variability in dispersal syndromes (Cote et al. 2017). For instance, Shima and Swearer (2009) found that the size and growth rate of dispersing marine reef larvae depended on the conditions experienced during dispersal between reefs. At the proximate level, such variability in dispersal syndromes might result from fragmentation changing the patterns of spatial selection on dispersal phenotypes, for instance increasing the costs of dispersal and therefore filtering out the non-specialized dispersers (Cote et al. 2017). In addition, fragmentation might also drive the form and intensity of phenotypic plasticity seen before, during or after dispersal, therefore modifying the patterns of covariation between dispersal and other phenotypic traits. Are dispersal syndromes conditional such that they vary depending on the environment? What are the mechanisms underlying such context-dependency? These are important questions to answer to improve our understanding of the drivers of variability in dispersal syndromes.

Here, we tested whether habitat fragmentation modulates dispersal syndromes by manipulating the harshness of the matrix separating habitats patches, a fundamental but overlooked component of habitat fragmentation that mediates functional connectivity (Bestion et al. 2019, Fahrig 2003, Haddad et al. 2015). Matrix harshness should raise the costs of dispersal and might consequently modulate dispersal syndromes. Since the causes and consequences of dispersal syndromes might differ depending on their underlying mechanisms, we quantified the effects of fragmentation on dispersal syndromes both in the presence and absence of genetic variability (i.e. genetically diverse versus isogenic clonal populations; see below). In the absence of genetic variability, only phenotypic plasticity (whether adaptive or not) could generate dispersal syndromes since all individuals bear the same genotype. In the presence of genetic variability, differences in dispersal propensity between phenotypically different genotypes may generate dispersal syndromes by phenotypic segregation, in addition to the effects of phenotypic plasticity.

We used microcosms of the ciliate protist *Tetrahymena thermophila* as a model system to experimentally test the abovementioned predictions (Jessup et al. 2004, Benton et al. 2007,

Altermatt et al. 2015, Jacob et al. 2017, 2018). Dispersal syndromes have been previously documented in this species, involving traits related to morphology, movement and demography (Fjerdingstad et al. 2007, Pennekamp et al. 2014, 2018, Jacob et al. 2016, 2018, 2019). We quantified dispersal syndromes as the difference between residents and dispersers in morphology (cell size and elongation), movement (velocity and linearity) and demography (growth rate and maximal population density), and compared these syndromes between two treatments: a control matrix versus a harsh matrix (i.e. no resources). The availability of several clonally reproducing strains of this species (hereafter called 'genotypes') allows us to contrast patterns of dispersal syndromes in both the absence and presence of genetic variability, by using either isogenic clonal genotypes kept isolated or mixing several genotypes. First, to test the importance of phenotypic plasticity in generating dispersal syndromes and how fragmentation modifies these plastic syndromes, we quantified the differences between residents and dispersers in the absence of genetic variability (i.e. genotypes kept separated in isogenic cultures making dispersers and residents to share identical genetic background). Second, to test for the importance of genetically-based phenotypic segregation in addition to phenotypic plasticity, we measured these same dispersal syndromes in the presence of genetic variability by performing the same experiment but with the five genotypes mixed in the same dispersal systems.

Methods

Culture conditions and genotypes

Tetrahymena thermophila is a 30–50 µm unicellular eukaryote, a ciliated protozoon naturally living in freshwater ponds and streams in North America (Collins 2012, Doerder and Brunk 2012). We used five genotypes, originally sampled from different locations in North America (D3, D4, D6, D13 and D17; Zufall et al. 2013, Pennekamp et al. 2014), which reproduce clonally in our culture conditions (Elliott and Hayes 1953, Bruns and Brussard 1974). Cells were maintained in axenic rich liquid growth media (0.6% Difco proteose peptone, 0.06% yeast extract) at 23°C (Schtickzelle et al. 2009, Chaine et al. 2010, Altermatt et al. 2015). All manipulations were performed in sterile conditions under a laminar flow hood.

Dispersal experiment

To allow dispersal and then quantify phenotypic traits of residents and dispersers, we used standard two-patch microcosms consisting of two habitat patches (1.5 ml standard microtubes) connected by a corridor (4 mm internal diameter silicon tube, 2.5 cm long) and filled with growth medium (Fjerdingstad et al. 2007, Schtickzelle et al. 2009, Chaine et al. 2010, Pennekamp et al. 2014, Jacob et al. 2015b, 2016). We manipulated matrix harshness by filling corridors with either nutrients (i.e. control matrix) or water without resources (harsh matrix), as used in a previous study (Fronhofer et al. 2018). To do so, all two-patch systems were initially filled with water (in patches and corridor), and nutrients were added in each patch while keeping corridors closed to avoid diffusion of nutrients in the corridors. For the control matrix treatment, corridors were then opened and the two-patch systems homogenized in order to let nutrients diffuse in the corridors. In the harsh matrix treatment, corridors were opened, but systems were not homogenized, which allowed us to maintain a low concentration of resources in the corridors for more than four hours (Supplementary material Appendix 1 Fig. A1).

We performed this dispersal experiment both in the presence and absence of genetic variability. We used each of the five genotypes kept isolated to quantify dispersal syndromes in the absence of genetic variability, and used a freshly mixed culture of the five genotypes at equal density to generate genetic diversity. We performed five replicates for each experimental condition (five genotypes alone plus a mixed culture, each tested under two matrix harshness treatments). Cells were placed on one side of the two-patch systems (i.e. 'start patch'; standard density of inoculated cells = 40 000 cells ml^{-1}) and corridors were opened for four hours to allow dispersal towards the initially empty neighbour patch. After four hours of potential dispersal, the corridors were closed to separate residents (cells remaining in the start patch) from dispersers (cells that actively moved to the target patch). We chose to let cells disperse for four hours in order to guarantee limited population growth during the experiment. This species indeed shows a latency time before growth initiates after transfer in a new tube (latency time in this experiment: mean ± SE = 33.04 ± 4.04 h), meaning that population growth is negligible during four hours and thus does not affect quantifications of dispersal rates and phenotypic traits (Pennekamp et al. 2014, Jacob et al. 2018).

Quantification of phenotypic traits and growth of residents and dispersers

Immediately after the four hours dispersal period, we used a standardized procedure to measure cell density and phenotypic traits in residents and dispersers. From each culture, we pipetted five samples (10 µl each) into chambers of a multichambered counting slide (Kima precision cell 301890), and immediately took digital pictures under dark-field microscopy (Schtickzelle et al. 2009, Pennekamp and Schtickzelle 2013, Pennekamp 2014). Population density and cell phenotypic traits in cultures were measured based on an automatic analysis of pictures (Pennekamp and Schtickzelle 2013) using IMAGEJ software (ver. 1.47; Schneider et al. 2012). We also took a 20 s video from each patch under dark-field microscopy to measure cell movement characteristics using BEMOVI R-package (Pennekamp et al. 2015). This package tracks moving particles in the videos through an image processing workflow involving IMAGEJ software (Pennekamp et al. 2015).

We quantified dispersal rate as the proportion of cells present in the target patch (i.e. $N_{target}/(N_{start}+N_{target})$). From pictures, we quantified two morphologic traits of residents and dispersers: cell size (cell surface area on pictures) and elongation (ratio of cell major/minor axis; Fjerdingstad et al. 2007, Jacob et al. 2016). From videos, we quantified the velocity and linearity of cell movement trajectories. Cell velocity is defined as the total distance travelled by cells divided by the duration of the trajectory, and linearity is the ratio between the net distance travelled (Euclidian distance between start and end positions) and the total distance effectively moved through a more or less tortuous way such that higher values indicate straighter trajectories. This index is sometimes referred as NGDR (net to gross distance ratio) or straightness index. It may be biased by large location errors and/or when very long movements are recorded (Almeida et al. 2010), but none of these issues occur in the system we used.

Finally, we quantified growth rate and maximal population density of residents and dispersers as demographic traits characterizing reproductive strategies. To do so, we transferred 10 µl of resident or disperser cells (~100 cells) from each patch into 96-well plates (250 µl wells) filled with growth medium, with five replicates for each resident and disperser patch. Population growth was quantified through absorbance measurements at 550 nm recorded every three hours for two weeks using a microplate reader. Absorbance, as classically used in cell culture research, is significantly and linearly correlated with cell density within the range of densities observed under our culture conditions (Pennekamp 2014, Jacob et al. 2017). To avoid any bias due to slight variability in absorbance measures, and thus allow the predicted logistic growth curves to accurately match the observed data, we smoothed the absorbance data using general additive model (gam package; Hastie 2018), a non-parametric method that does not require any assumption regarding the shape of the curve. We then used the grofit package (gcfit function; Kahm et al. 2010) to fit a spline-based growth curve and compute the growth rate as the maximum slope of population growth through time, and the maximal population density as the density reached at the plateau (Jacob et al. 2017, 2018).

Statistical analyses

We quantified dispersal syndromes by computing the difference of trait values between dispersers and residents ($Trait_{disperser} - Trait_{resident}$) for each of the six traits measured (i.e. cell size, elongation, velocity, linearity, growth rate and maximal population density; centred and scaled with respect to the full dataset to provide comparable metric scale among phenotypic traits; base package, scale function; <www.r-project.org>). This led to one value of resident-disperser phenotypic difference for each trait and each two-patch dispersal system.

We then tested for the effect of matrix harshness on dispersal rate and dispersal syndromes. First, when all cells bore the same genotype (i.e. isolated genotypes), we tested whether resident-disperser differences can occur and whether these syndromes depend on matrix harshness. We used each phenotypic difference as the dependent variable in separate ANOVA models (stats package; lm function; <www.r-project.org>). Matrix harshness treatment, genotype and their interaction were used as explanatory factors. Second, we tested whether these resident-disperser differences changed depending on the presence or absence of genetic variability and on matrix harshness. To do so, we performed an ANOVA with each phenotypic difference as the dependent variable, and 'genetic variability' (i.e. isogenic versus mixed culture), matrix harshness and their interaction as explanatory factors. Note that using traits as dependent variables instead of disperser-resident differences, with dispersal status, matrix harshness and their interaction as fixed factors, and genotype as a random factor, lead to qualitatively similar conclusions (results not shown). In addition to the phenotypic and growth traits, we performed the same analyses as presented above for dispersal rate.

Finally, we drew the architecture of dispersal syndromes involving all six traits quantified in this study using a principal component analysis (ade4 package; dudi.pca function; <www.r-project.org>). We tested whether matrix harshness affects the syndromes of multiple dispersal-related traits by using an ANOVA with principal component scores as dependent variables and matrix harshness, dispersal status (i.e. resident versus disperser) and their interaction as explanatory factors.

With the exception of dispersal rate that were logit transformed, in all cases models followed assumptions of normality of residuals and homoscedasticity. Estimation of variance attributed to each explanatory factor was performed through variance partitioning analysis (relaimpo package; calc.relimp function with the lmg metric; Lindeman et al. 1980, Groemping 2006). Interactions were removed from the models when non-significant ($p > 0.05$).

Results

Dispersal rate and matrix harshness

As expected, matrix harshness generally reduced dispersal rate (Fig. 1a, Table 1), and we found intraspecific variability in the magnitude of this effect (genotype by matrix harshness interaction; Table 1), with three out of the five genotypes showing a decrease of dispersal rate in the presence of a harsh matrix (Supplementary material Appendix 1 Fig. A2). Dispersal rate decreased similarly in the harsh matrix treatment in the presence of genetic variability (i.e. mix of genotypes; Fig. 1a, Table 2).

Intraspecific variability and plastic dispersal syndromes

In the absence of genetic variability, i.e. in isogenic cultures where only phenotypic plasticity can lead to dispersal

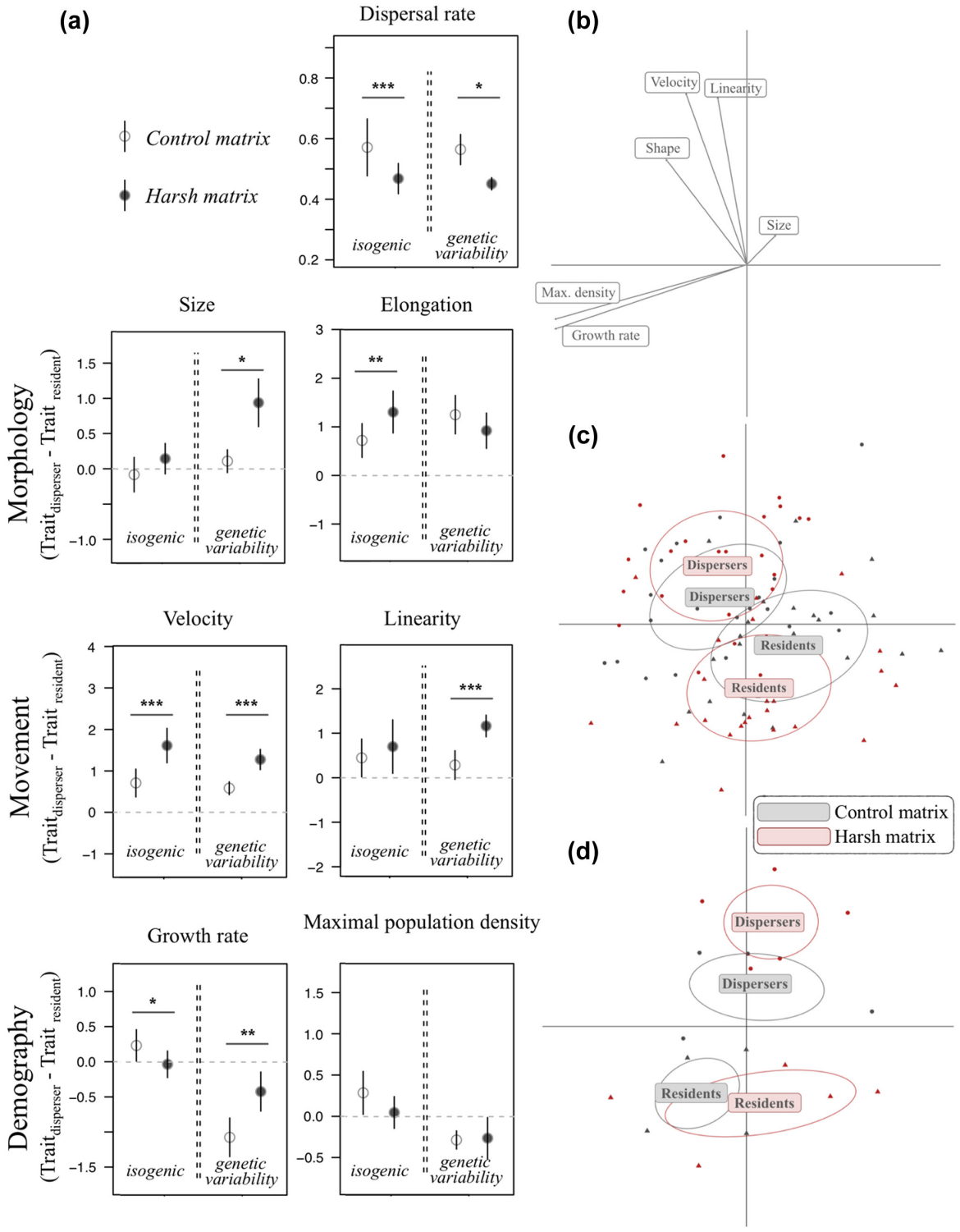

Figure 1. Effects of matrix harshness on dispersal rate and dispersal syndromes. (a) Matrix harshness effects on dispersal rate and disperser-resident differences in the absence of genetic variability ('isogenic', i.e. mean value over the five genotypes, each measured in isogenic culture; details in Supplementary material Appendix 1 Fig. A2) and presence of genetic variability ('genetic variability', i.e. mix of cells from the five genotypes; 'genetic variability'). Grey dots represent the control matrix, black dots represent the harsh matrix. Mean ± SE are shown; see statistics in Table 1, 2. Supplementary material Appendix 1 Fig. A2 for traits for each individual genotype. (b) Organisation of morphological, movement and demographic traits in principal components. Grey arrows show the contribution of traits to each axis of the principal component analysis. (c) Differences between residents and dispersers in the multiple-trait space depending on matrix harshness in the absence of genetic variability and (d) in the presence of genetic variability. Circular dots for dispersers, triangles for residents.

Table 1. Intraspecific variability and context-dependency of plastic dispersal syndromes, quantified separately for each of the five genotypes kept isolated. Statistics and variance partitioning of genotype (i.e. among genotypes differences), matrix harshness treatment and their interaction are shown for each response variable (dispersal rate and the six morphologic, mobility and growth syndromes). Significant effects are highlighted in bold, and adjusted p-values after Bonferroni correction (n = 7) are provided.

	Sum Sq	df	F	p	p.adjust	R^2
Dispersal rate						
Genotype	**17.304**	**4,40**	**15.803**	**<0.001**	**<0.001**	**0.43**
Matrix harshness	0.536	1,40	2.447	0.124	0.870	0.14
Matrix × Genotype	**4.674**	**4,40**	**4.269**	**0.003**	**0.019**	**0.10**
Morphology						
Cell size						
Genotype	0.884	4,44	0.8152	0.522	1	0.07
Matrix harshness	0.642	1,44	2.3682	0.131	0.917	0.05
Matrix × Genotype						
Cell elongation						
Genotype	**13.726**	**4,44**	**6.564**	**<0.001**	**0.002**	**0.33**
Matrix harshness	**4.273**	**1,44**	**8.173**	**0.006**	**0.045**	**0.10**
Matrix × Genotype						
Movement						
Velocity						
Genotype	2.838	4,44	0.994	0.421	1	0.06
Matrix harshness	**10.153**	**1,44**	**14.215**	**<0.001**	**0.003**	**0.23**
Matrix × Genotype						
Linearity						
Genotype	**15.550**	**4,44**	**3.463**	**0.015**	0.106	**0.237**
Matrix harshness	0.806	1,44	0.718	0.401	1	0.012
Matrix × Genotype						
Demography						
Growth rate						
Genotype	1.953	4,44	2.572	0.051	0.356	0.17
Matrix harshness	**0.905**	**1,44**	**4.769**	**0.034**	0.241	**0.08**
Matrix × Genotype						
Carrying capacity						
Genotype	2.403	4,44	2.580	0.050	0.352	0.18
Matrix harshness	0.707	1,44	3.036	0.088	0.619	0.05
Matrix × Genotype						

syndromes (Fig. 2a), we found that dispersers were more elongated than residents, moved faster and tended to show straighter movement (elongation, velocity and linearity respectively; Fig. 1a, Supplementary material Appendix 1 Table A1), but did not differ significantly in their size, growth rate and maximal population density. We furthermore found that the degree of difference between residents and dispersers in cell elongation and linearity differed between genotypes (genotype effects in Table 1, Supplementary material Appendix 1 Fig. A2).

These morphological and movement differences between residents and dispersers depended on matrix harshness: the differences between residents and dispersers in elongation and velocity were greater in the harsh matrix than in the control matrix treatment (matrix harshness effect in Table 1, Fig. 1a, Supplementary material Appendix 1 Table A1). Phenotypic traits in residents did not significantly change depending on matrix harshness (all $p > 0.1$; Fig. 1b–d), meaning that a change in dispersal syndromes between matrix treatments in all cases resulted from phenotypic changes in dispersers. Finally, genotypes did not significantly differ in how resident-disperser phenotypic differences changed with matrix harshness (non-significant genotype by matrix interactions; Table 1), showing that there is no significant intraspecific variability in how dispersal syndromes change with fragmentation within the set of genotypes we used here.

The effect of matrix harshness on phenotypic differences between residents and dispersers resulted in context-dependent dispersal syndromes (Fig. 2b). A principal component analysis revealed that the six traits quantified in this study organise along three orthogonal dimensions. A first dimension comprised of reproductive traits (growth rate and maximal population density), a second comprised of phenotypic elongation and movement (cell shape, velocity and linearity), and the third component including cell size (Fig. 1b; total variance explained = 77.1%; PC1: 31.6%, PC2: 28.7% and PC3: 16.9%). We found a significant disperser status by matrix harshness interaction on scores on the second principal component (estimate ± SE = 1.32 ± 0.40; $t = 3.30$; $p = 0.001$; Fig. 1c–d), but not on PC1 and 3 (respectively 0.52 ± 0.53; $t = 0.97$; $p = 0.33$ and 0.04 ± 0.40; $t = 0.10$; $p = 0.92$).

Interestingly, although phenotypic traits in residents did not significantly differ between control and harsh matrix treatments, matrix harshness had a significant effect on PC2

Table 2. Occurrence and context-dependency of dispersal syndromes in the presence of genetic variability. Statistics and variance partitioning of genetic variability (i.e. presence versus absence of genetic variability), matrix harshness treatment and their interaction are shown for each morphologic, mobility and growth trait. Significant contributions to dispersal syndromes are highlighted in bold, and adjusted p-values after Bonferroni correction (n = 7) are provided.

	Sum Sq	df	F	p	p.adjust	R^2
Dispersal rate						
Genetic variability	0.039	1,57	0.077	0.783	1	0.001
Matrix harshness	**3.389**	**1,57**	**6.593**	**0.013**	0.089	**0.10**
Matrix × Gen.var.						
Morphology						
Cell size						
Genetic variability	**2.018**	**1,57**	**7.030**	**0.010**	0.073	**0.10**
Matrix harshness	**1.602**	**1,57**	**5.581**	**0.022**	0.151	**0.08**
Matrix × Gen.var.						
Cell elongation						
Genetic variability	0.045	1,57	0.058	0.810	1	0.001
Matrix harshness	2.804	1,57	3.612	0.062	0.437	0.06
Matrix × Gen.var.						
Movement						
Velocity						
Genetic variability	0.442	1,57	0.699	0.407	1	0.001
Matrix harshness	**11.261**	**1,57**	**17.814**	**<0.001**	**0.001**	**0.24**
Matrix × Gen.var.						
Linearity						
Genetic variability	0.194	1,57	0.160	0.691	1	0.003
Matrix harshness	1.923	1,57	1.59	0.213	1	0.03
Matrix × Gen.var.						
Demography						
Growth rate						
Genetic variability	**7.139**	**1,56**	**29.886**	**<0.001**	**<0.001**	**0.28**
Matrix harshness	0.905	1,56	3.789	0.057	0.396	0.01
Matrix × Gen.var.	**1.765**	**1,56**	**7.3887**	**0.009**	0.061	**0.08**
Carrying capacity						
Genetic variability	**1.623**	**1,57**	**6.472**	**0.014**	0.096	**0.10**
Matrix harshness	0.568	1,57	2.264	0.138	0.965	0.03
Matrix × Gen.var.						

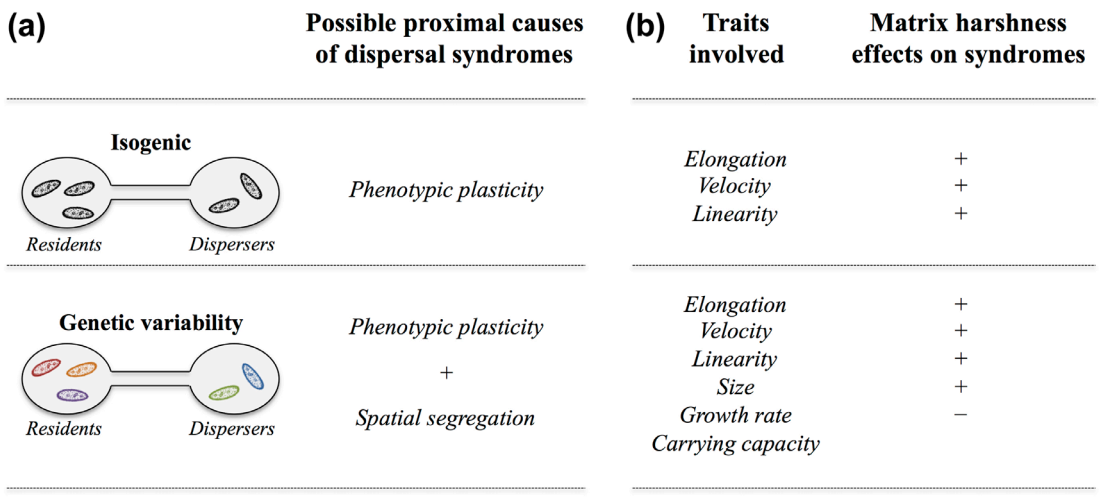

Figure 2. (a) Schematic illustration of the dispersal systems in the absence of genetic variability (inoculated either with isogenic cultures where all cells bear the same genotype) or in the presence of genetic variability (mix of cells from the five different genotypes) and possible proximal causes of dispersal syndromes (five replicates for each experimental condition). (b) Results of the experiments for the different dispersal-related traits and how these syndromes were affected by matrix harshness (+ for an increased difference between residents and dispersers, − for a decrease, blank when not significant).

scores in residents (0.76 ± 0.29; $t = 2.66$; $p = 0.01$). This suggests that negligible context-dependence of dispersal-related traits taken individually might still result in context-dependent syndromes of multiple traits.

Genetic variability and dispersal syndromes

The differences between residents and dispersers found in the absence of genetic variability (cell elongation, velocity and linearity; Fig. 2) did not increase nor decrease significantly when quantified in the presence of genetic variability (genetic variability effect in Table 2). However, additional phenotypic differences between residents and dispersers not seen in the absence of genetic diversity were revealed in the presence of genetic variability. Specifically, dispersers were bigger and showed slower growth rate and lower maximal population density than residents in the presence of genetic variability (genetic variability effect in Table 2, Fig. 1).

Matrix harshness led to an increase of the resident-disperser differences in cell size, a decrease in the differences of growth rate, and did not significantly affect maximal population density differences in the presence of genetic variability (Table 2, Fig. 1). Interestingly, additional analyses on the phenotypic traits in residents revealed that while matrix harshness did not significantly affect phenotypic traits of residents in the absence of genetic variability, cell linearity and growth rate in residents tended to decrease when facing a harsh matrix in the presence of genetic variability (linearity: estimate $\pm$ SE $= -0.73 \pm 0.30$, $t = 2.47$, $p = 0.040$; growth rate: -0.80 ± 0.36, $t = 2.24$, $p = 0.055$; all other $p > 0.1$; Fig. 2b).

The corridors in the two-patch systems used here contains ~300 µl (4 mm internal diameter, 2.5 cm long), meaning that homogenization of nutrients in the corridors in the control matrix treatment lead to a dilution of the patches resource concentration by a factor 1.1. The expected effect of such 1.1 dilution would be of $0.41 \pm 0.30\%$ for dispersal rate, of $3.39 \pm 1.92\%$ for growth rate and of $4.24 \pm 1.99\%$ for carrying capacity (data from Jacob et al. in press). Furthermore, we found that matrix harshness did not significantly affect phenotypic traits of residents in isogenic cultures, as would be expected if matrix harshness effects resulted from a dilution of local patch quality instead of matrix harshness itself. Altogether, we can therefore confidently argue that such small dilution factor cannot explain the effects found in the present study.

Discussion

In this study, we experimentally manipulated the harshness of the matrix separating habitats in microcosms of the ciliate *Tetrahymena thermophila* to test whether and how habitat fragmentation modulates dispersal syndromes. In line with previous studies (Fjerdingstad et al. 2007, Pennekamp et al. 2014, 2018, Jacob et al. 2016, 2019), we provided evidence for dispersal syndromes involving multiple traits linked to morphology (elongation and size), movement (velocity and linearity) and demography (growth rate and maximal population density). The evidence for such multi-dimensional dispersal syndromes echoes numerous previous studies that define dispersal as a combination of multiple traits, beyond a simple tendency to disperse (Clobert et al. 2009, Stevens et al. 2014, Cote et al. 2017, Beckman et al. 2018, Jacob et al. 2019a). Interestingly, differences between residents and dispersers in cell elongation and movement occur even when residents and dispersers share identical genetic background, meaning that dispersal syndromes might result from phenotypic plasticity. Furthermore, we found matrix harshness to decrease dispersal rate, as expected since habitat fragmentation is expected to raise the costs of dispersal (Schtickzelle and Baguette 2003, Fahrig 2007, Bonte et al. 2012, Haddad et al. 2015, Cote et al. 2017). Most importantly, we demonstrated that dispersal syndromes are context-dependent, with phenotypic differences between residents and dispersers that changed depending on matrix harshness (Fig. 2).

Proximate causes of dispersal syndromes

In the absence of genetic variability, meaning that all cells in a dispersal system have the same genotype, we found that dispersers were more elongated, moved faster and tended to show a straighter movement than residents. These three traits are classically associated with dispersal in this species: elongated cells of *T. thermophila* indeed show greater swim speed and straighter movements, and are thought to be more efficient dispersers because of reduced resistance during movements (Stein and Bronner 1989, Fjerdingstad et al. 2007, Schtickzelle et al. 2009, Pennekamp et al. 2014, Jacob et al. 2016). Importantly, these phenotypic differences occurred in the absence of genetic variability: isogenic cultures where residents and dispersers share identical genetic background. This finding shows that these dispersal-related traits (i.e. cell elongation, velocity and linearity) resulted from plastic changes in cell phenotypic traits that might occur before, during or after dispersal, and not from spatial segregation during dispersal of genotypes differing in their phenotypic traits. Interestingly, such plastic dispersal syndromes might modify the direction or distance of dispersal movements or alleviate dispersal costs. Consequently, they might greatly affect metapopulation dynamics and response to environmental changes, for instance by increasing recolonization rates and thus the stability of metapopulations, or fastening range expansions (Clobert et al. 2009, Lande 2015, Cote et al. 2017).

Whether such plastic dispersal syndromes result from adaptive phenotypic plasticity (the induction of a specific dispersal phenotype to increase dispersal success) or non-adaptive changes triggered by costs experienced during dispersal (Bonte et al. 2012, Cote et al. 2017) is an open question. Here we found that dispersers were more elongated and move more linearly and faster than residents, traits that are thought in this species and others to increase dispersal success (Phillips et al. 2006, 2010, Shine et al. 2011, Pennekamp et al. 2014, Jacob et al. 2016, Cote et al. 2017). Further investigation should test whether this potentially

adaptive phenotypic plasticity occurs 1) before dispersal, with specialized phenotypes produced through plasticity that disperse preferentially, 2) during dispersal, with phenotypic plasticity mediating the filtering effect of matrix harshness or 3) after dispersal, where phenotypic changes result from the process of colonizing a new environment.

Interestingly, in the presence of genetic variability (i.e. mix of genotypes), dispersers differed from residents not only in their elongation, velocity and linearity, but also in their size, growth rate and maximal population density. These additional traits are often found linked to dispersal. Body size is considered one of the main traits linked to dispersal in metapopulation and metacommunity theory (Cohen et al. 2003, Stevens et al. 2014, Dahirel et al. 2015). Furthermore, dispersal is often expected to tradeoff against competitive ability and thus related to reproductive characteristics (Hastings 1980, Leibold et al. 2004, Travis et al. 2012, Bonte and Dahirel 2017). The rise of these additional resident-disperser differences in the presence of genetic variability might be explained by a spatial segregation of genetically-based phenotypic traits: that is when the most dispersive genotypes carry different and heritable phenotypic traits compared to less dispersive individuals. Genotypes in this species have indeed repeatedly been found to differ regarding to multiple phenotypic traits (Supplementary material Appendix 1 Fig. A3; Fjerdingstad et al. 2007, Schtickzelle et al. 2009, Chaine et al. 2010, Jacob et al. 2015b, 2016, 2018). In such a case, dispersal syndromes based on body size and reproductive characteristics could lead to evolution by spatial selection, for instance leading to increased fecundity at range margins or during range expansions (Phillips et al. 2006, Shine et al. 2011, Fronhofer and Altermatt 2015). Alternatively, differences between residents and dispersers occurring only in the presence of genetic variability might result from phenotypic plasticity if this plasticity differ among genotypes or depends on e.g. kinship (Cote et al. 2007, Chaine et al. 2010, Cote and Clobert 2010, Jacob et al. 2016). Quantifying the level of heritability of phenotypic traits involved in dispersal syndromes and the (epi)genetic and transgenerational mechanisms underlying their variability is needed to improve our understanding of the evolution and eco-evolutionary consequences of dispersal.

Conditional dispersal syndromes

Further than providing evidence for multiple traits involved in dispersal syndromes, we showed that these syndromes depend on matrix harshness, one fundamental dimension of habitat fragmentation (Bestion et al. 2019, Fahrig 2003, Cote et al. 2017, Legrand et al. 2017). First, we found that matrix harshness can strengthen dispersal syndromes: the difference between residents and dispersers in cell size, elongation, velocity and linearity increased when dispersers had to cross a harsh matrix. This suggests that habitat fragmentation might increase plastic change of phenotypic traits toward more specialized dispersive phenotypes, and intensify the sorting of phenotypic variability during dispersal. Second, we found that matrix harshness decreased the difference between residents and dispersers in growth rate. Together, these results illustrate the diversity of effects that environmental changes can have on dispersal, and thus the importance of our understanding of dispersal drivers for the accuracy of ecological and evolutionary forecasting (Travis and Dytham 2012, Travis et al. 2012, Santini et al. 2016, Cote et al. 2017).

The consequences of context-dependent dispersal syndromes for ecological and evolutionary dynamics should differ depending on the proximal causes underlying these syndromes. For example, an increased investment in plastic dispersal traits might increase dispersal success and thus buffer against increased costs of dispersal induced by habitat fragmentation (Cote et al. 2017). Plastic dispersal syndromes might therefore favour metapopulation persistence under habitat fragmentation, and help maintain functional connectivity in fragmented landscapes. Conversely, when the difference between residents and dispersers result from the spatial segregation of genetically determined traits, dispersal syndromes might also increase metapopulation persistence facing habitat fragmentation if individuals with specialized dispersal phenotypes engage preferentially in dispersal (Cote et al. 2017). When dispersal syndromes result from the spatial segregation of genetically determined traits, environmental changes such as fragmentation and the resulting selective pressures acting on dispersal-related traits (McPeek and Holt 1992, Olivieri et al. 1995, Travis 2001, Duputié and Massol 2013) could generate a diversity of local dispersal syndromes throughout a landscape (Cote et al. 2017).

Conclusions

The role of dispersal and dispersal syndromes in population and species response to environmental changes and in turn the effects of these environmental changes on the evolution of dispersal have been the focus of considerable research effort in the past decades (Olivieri et al. 1990, McPeek and Holt 1992, Travis 2001, Travis and Dytham 2012, Duputié and Massol 2013, Travis et al. 2013, Bonte and Dahirel 2017, Cote et al. 2017). Dispersal is increasingly recognized as a complex process, a complexity that can affect both the evolution of dispersal and its consequences for ecological and evolutionary dynamics (Bowler and Benton 2005, Ronce 2007, Edelaar et al. 2008, Clobert et al. 2012, Travis and Dytham 2012, Travis et al. 2012, Jacob et al. 2015a, 2017). For instance, a recent study using microcosms of *T. thermophila* experimentally demonstrated that variability in dispersal syndromes can be as important for metapopulation dynamics as spatiotemporal variability of environmental conditions (Jacob et al. 2019a). Such evidence points out we need to further explore intraspecific variability in both dispersal and its underlying mechanisms. Here we experimentally demonstrated that the intensity and nature of dispersal syndromes can be driven by habitat fragmentation. Investigating the consequences of dispersal syndromes that vary depending on habitat fragmentation for gene flow in metapopulations, species range shifts or eco-evolutionary feedbacks under

environmental changes remain key questions for future investigation.

Experimental or semi-natural systems such as the microcosms used in this study, provide an unique opportunity to answer the above-mentioned questions (Jessup et al. 2004, Benton et al. 2007, Legrand et al. 2012, Altermatt et al. 2015, Jacob et al. 2017). These systems are by nature simplified and their utility is in deciphering the mechanisms and causality underlying natural processes in highly controlled and replicated experimental conditions, which would be otherwise impossible under natural conditions (Jessup et al. 2004, Srivastava et al. 2004, Benton et al. 2007). Ciliates are organisms usually covered with cilia that provide them high mobility to catch food and move from one location to another. Many species are able to orient in their environment through for instance taxic responses (reviewed by Fenchel 1987), including *T. thermophila* that is able to adjust dispersal decisions relative to kinship (Chaine et al. 2010), population density (Pennekamp et al. 2014, Jacob et al. 2016), resources (Jacob et al. 2019a) and temperature (Jacob et al. 2017, 2018). In the microcosms used in this study, patches are separated by 2.5 cm corridors, or over 1000 times the size of a *T. thermophila* cell, and each patch is of sufficient size and contains ample resources to allow populations to follow classical logistic population growth and end-up with very large population sizes (up to ~500 000 cells per patch). Furthermore, residents and dispersers in these microcosms differ in their phenotype, with for instance dispersers being more elongated, moving faster and in a more linear way (Pennekamp et al. 2014, 2018, Jacob et al. 2016, 2019). Finally, we previously demonstrated that these dispersal movements influence local adaptation by generating gene flow among patches and affecting population differentiation (Jacob et al. 2017). The movements we quantify in these microcosms therefore match the classical definition of dispersal movements: movements between populations than can lead to gene flow (Clobert et al. 2004, 2012, Ronce 2007).

This experimental study using microcosms shows that habitat fragmentation can modify dispersal syndromes through both spatial selection and phenotypic plasticity. These results suggest we need to integrate the mechanisms and variability of dispersal syndromes into ecological and evolutionary theory. Future investigation should now explore the generality of the patterns we found and whether the relative importance of plasticity and spatial selection for variability in dispersal syndromes differs depending on species life history strategies. For instance, whether terrestrial, freshwater and marine organisms show common patterns of context-dependent dispersal syndromes or in contrast differ in this regard represent promising avenues to improve our understanding of how biodiversity will respond to environmental changes (Berg et al. 2010, Travis et al. 2013, Urban et al. 2016, Cote et al. 2017, Legrand et al. 2017).

Data availability statement

Data are available from the Dryad Digital Repository: <http://dx.doi.org/10.5061/dryad.d2547d7z8> (Jacob et al. 2019b).

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Author contributions – SJ designed the project, with inputs from NS. SJ and EL performed the experiments, with the help of TMJ. SJ analysed the data and wrote the manuscript. NS contributed to revisions and all authors approved its final version.

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Supplementary material (available online as Appendix oik-06857 at <www.oikosjournal.org/appendix/oik.06857>). Appendix 1.

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