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Phenotypic and dispersal plasticity are not alternative strategies for organisms to face thermal changes

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To buffer the effects of local environmental changes, organisms may modify their phenotypic traits (i.e. phenotypic plasticity) or disperse towards other potential habitats (i.e. dispersal plasticity). Despite extensive work studying either 'local phenotypic plasticity' or 'dispersal plasticity' independently, little is known about their potential covariation and interplay. These strategies are classically viewed as alternatives. However, this expectation has been challenged by theoretical work suggesting that they may instead evolve together under some environmental contexts. Here, we experimentally quantified morphological, movement and dispersal plasticity in response to thermal changes in 12 strains of the ciliate *Tetrahymena thermophila*. We showed that phenotypic and dispersal plasticity are not alternative strategies, with half of the strains expressing simultaneously all dimensions of plasticity in response to thermal changes. Furthermore, the extent of morphological and movement plasticity weakly but significantly differed between residents and dispersers. Interestingly, we found no covariation between these different plasticity dimensions, suggesting that they may evolve independently, which pleads for studying which environmental contexts favour the evolution of each. The fact that phenotypic and dispersal plasticity are not alternative strategies and may affect the expression of one another opens interesting perspectives about their joint evolution and the potential consequences of their interplay.

1. Introduction

Following an environmental change impacting their fitness, organisms may deal with the new local conditions through a variety of strategies, including phenotypic plasticity, genetic adaptation and adjustment of the environment [1–3]. Local phenotypic plasticity, the ability of a genotype to produce different phenotypes depending on the local environment, is expected to help organisms buffer the effects of environmental changes [1,4]. This widespread mechanism is known as key for ecological and evolutionary dynamics, allowing organisms to persist in changing or stressful environments [1,4–7]. For instance, the western white butterfly (*Pontia occidentalis*) adaptively changes its colour pattern depending on the season (i.e. polyphenism) [8], and the microalga *Dunaliella salina* changes cell shape and content to face temporal fluctuations of salinity [9].

Instead of dealing with new (unfavourable) local environmental conditions, organisms may avoid them by dispersing towards another habitat [10–12]. Dispersal plasticity (also called context-dependent dispersal) is a form of phenotypic plasticity in which the plastic trait is the decision of an organism to emigrate from the local patch to disperse towards other potential habitats [13–16]. For instance, five ciliate species of the genus *Tetrahymena* have

been shown to change their emigration decisions across abiotic gradients in microcosms, dispersal decisions that were correlated with the fitness sensitivity to these environmental gradients [13]. Likewise, the Asteraceae *Crepis sancta* and *Carduus nutans* produce seed dispersal morphs, hence plastically increasing emigration rates, both when nutrients are depleted [17] and under drought conditions [18], respectively. Dispersal plasticity can strongly affect local adaptation, metapopulation dynamics and meta-ecosystem functioning [19–23]. The first step of dispersal plasticity consists in whether to stay in the local environment or leave it by initiating dispersal (i.e. emigration decision [14,21]). This first step has been shown to potentially have as strong consequences on metapopulation dynamics and local adaptation as habitat choice at settlement can have [23,24].

Local phenotypic plasticity and dispersal plasticity may be considered as alternative mechanisms: why change your phenotype if you can change your environment, and vice versa [25]? On the one hand, adaptive dispersal plasticity allows organisms to leave unsuitable conditions to potentially find better ones in a landscape. These plastic dispersal decisions should decrease the range of environments experienced by individuals and may therefore mitigate the benefits of being locally phenotypically plastic. Dispersal plasticity should thus favour the evolution of phenotypically non-plastic specialists [23,26–30]. On the other hand, plastic changes in traits related to fitness in the local conditions should help organisms deal with local environmental changes and thus allow high environmental generalism. As locally plastic organisms should maintain their fitness across a wider range of environmental conditions, they should thus not need to adjust their dispersal decisions compared to non-locally plastic organisms.

However, recent theoretical models [31,32] suggest that local phenotypic plasticity and dispersal plasticity may sometimes evolve together. Especially, Scheiner's model [31] demonstrates that temporal variations in environmental conditions can favour the evolution of phenotypic plasticity, even in the presence of dispersal plasticity. Some empirical examples also challenged this view of phenotypic and dispersal plasticity being alternatives, looking at dispersal plasticity at immigration (i.e. habitat choice). The ambush bug *Phymata americana* uses a combination of morphological and dispersal plasticity to match its colour to the background in order to increase its prey capture success rate [33], while the chameleon prawn *Hippolyte varians* uses the same mechanism of matching colour to improve its camouflage to the perspective of predatory fishes [34]. The amphibious fish *Kryptolebias marmoratus* moves on land when the water becomes hypoxic, and thus remodels its gill in order to survive out of the water [35]. Thermal generalism is known to be associated with morphological plasticity in *Tetrahymena thermophila* [36], and the more generalist genotypes showed plastic immigration decisions in a habitat choice experiment [37]. Therefore, whether local phenotypic plasticity and dispersal plasticity should be evolutionary alternatives or should coevolve probably depends on the characteristics of environmental changes, potential genetic covariation between the two forms of plasticity, and the subsequent relative costs and benefits of these mechanisms [25].

Further, plastic phenotypic changes may not only serve for local adaptation but can occur together with the dispersal movement itself [14,38]. Phenotypic plasticity may indeed help organisms to perform dispersal movements and deal with environmental conditions encountered during or after dispersal [33,35]. In these cases, we can expect local phenotypic plasticity and dispersal plasticity to be complementary rather than alternative strategies and to thus co-occur. Dispersal is often associated with a suite of other phenotypic traits that enable dispersal or enhance dispersal success, also called dispersal syndrome [14,38,39]. Across taxonomic groups, dispersers are often larger, with a marked locomotion-oriented morphology, more active, and move faster than residents [40–42]. While these dispersal trait associations may result from genetic differences between residents and dispersers, phenotypic plasticity has also been shown to play a key role in the establishment of resident-disperser phenotypic differences [38,43,44]. This points out the possibility for important interplays between phenotypic and dispersal plasticity in driving ecological and evolutionary dynamics when organisms evolve both strategies.

In this study, we investigated whether local phenotypic plasticity and dispersal plasticity at emigration were alternative strategies or, on the contrary, were both expressed in response to environmental changes. We specifically investigated the first step of dispersal plasticity, namely the emigration decision (i.e. whether to stay in and deal with the local environment or leave it by initiating dispersal). To do so, we quantified morphological, movement and dispersal plasticity across 12 isogenic and clonally reproducing strains of the ciliate *T. thermophila* in response to thermal changes. This species has been used as a model system in cellular and molecular biology for decades [45] and shows high intraspecific variability in morphology, movement and dispersal rates [46,47]. Furthermore, the plasticity of both phenotypic traits and dispersal show high intraspecific variability [13,37,48], which thus provides the materials to investigate whether these two dimensions of plasticity covary. Morphology was characterized as cell size, a trait known to be linked to resource acquisition and metabolic rate [49], and cell shape that modifies surface area to volume ratio and thus sets protists energetic intake [50]. Linking local phenotypic plasticity to fitness was not the aim of this study and could not be done with our experimental design. However, in protists, cell size and shape are correlated with thermal performance curve parameters and intrinsic growth rates [50]. Further, morphological plasticity in response to local thermal fluctuations is associated with local performance in *T. thermophila* strains [36]. We therefore predicted morphological plasticity to be mainly associated with strain adapting locally rather than dispersing depending on environmental conditions. Movement was described as cell velocity, a trait often linked to dispersal in this species [47,51], and trajectory linearity [47]. For dispersal plasticity, we quantified temperature effects on emigration decisions [14,21]. Previous studies revealed a match between preference for emigration and immigration in this species [37,52]. Under the hypothesis that local and dispersal plasticity would be alternative strategies, we expected to see less dispersal plasticity in the most morphologically plastic strains. However, theoretical and empirical evidence reviewed above suggests that the two strategies may both be expressed in the same organisms exposed to the same environmental conditions, thus leading to the expectation of a co-occurrence of local phenotypic and dispersal plasticity within strains. Due to the expected link between movement traits and dispersal, we could expect movement plasticity (i.e. plastic changes in movement velocity and linearity) to be strongly associated with dispersal plasticity. We also investigated whether the plasticity of morphological and movement traits covary. Furthermore, we tested whether morphological and movement plasticity and their covariation differed depending on dispersal

decisions, as expected if dispersal itself or the associated changes of environmental conditions encountered drive phenotypic plasticity or if phenotypic plasticity promotes dispersal. We expected dispersers to show higher plasticity for traits related to movement and residents to show higher plasticity for traits related to local adaptation. We further investigated the effects of the rate and amplitude of the thermal change on local phenotypic plasticity and dispersal plasticity at emigration using a subset of the strains.

2. Material and methods

(a) Biological system

We used 12 isogenic strains (D1, D2, D3, D6, D8, D9, D10, D12, D13, D16, D17, D19; electronic supplementary material, table S1) of the ciliate *T. thermophila*, a 20–50 µm micro-eukaryote naturally living in freshwater ponds and streams in the Eastern United States [53,54]. The natural distribution of this species is restricted to the Eastern United States, where the strains were sampled, with a pattern of moderate endemism [54], in contrast to other related species that show global distribution [55]. The strains were initially collected in the field by Dr Paul Doerder (Cleveland State University, USA) around 20 years ago (representing approx. 20 000 clonal generations in the laboratory). They have been kept in the Station d'Ecologie Théorique et Expérimentale of Moulis (France) since 2014 where they have been maintained in 24-wells culture plates at 23°C and axenic media (0.6% Difco proteose peptone, 0.06% yeast extract) with transfer every approximately 10 days. These 12 strains represent a large part of the phenotypic [56–58] and genomic [59] diversity in the known natural isolates of *T. thermophila* (the strains differ from approx. 1000 to more than 1 000 000 SNPs from each other and are representative of six distinct genomic types [59]). These organisms are known to be able to change their dispersal decisions depending on thermal conditions [13,23,37,52]. Prior to experiments, strains were allowed to grow from 1 ml of stock culture into 10 ml of media in Falcon tubes for seven days to obtain sufficient volumes for experiments. All manipulations were performed in sterile conditions under a laminar flow hood.

(b) Experimental design

Firstly, we quantified the plasticity (i.e. both the magnitude and the direction of the phenotypic change) of cell morphology and movement in the absence of dispersal for each strain (figure 1A). We exposed the 12 isolated strains to two temperatures: 23°C (the standard temperature at which strains are maintained in the laboratory) and 35°C (a thermally stressful condition previously used for habitat choice experiments [23,37,52]) for 3 h. To make these results comparable with the dispersal assay regarding the physical content in which cells are cultured, this quantification of phenotypic plasticity was performed using standard dispersal systems as presented below, but preventing dispersal by keeping the corridors closed.

In parallel, we quantified dispersal plasticity in response to the same thermal change using standard 1.5 ml two-patch systems connected by a corridor (4 mm internal diameter, 2.5 cm long silicone tube) filled with growth medium (figure 1A). We inoculated one patch of these two-patch systems with 300 µl of the one-week-old culture (close to asymptotic density) and let cells disperse. After 3 h we closed the corridors to prevent further movements. This time is below the minimum generation time at the tested temperatures but leaves sufficient time for cells to disperse [13] with negligible back-and-forth movement between patches.

We performed five replicates per strain, temperature and dispersal treatment (n = 240) in two consecutive days (six strains with all treatments and replicates per block). To initiate the experiment, we placed the inoculated systems in their corresponding temperature (incubator PolEko SmartPro) after a 30 min acclimation period at 23°C. For all systems, we quantified cell morphology, movement and dispersal rate after 3 h by recording 15 s videos of 10 µl samples placed in counting slides (Kima precision cell) under dark-field microscopy (Axio Zoom V16, Zeiss). We quantified cell abundance, morphology and movement characteristics from the videos recorded using the BEMOVI R-package [60]. We calculated emigration rate as $N_{disp}/N_{resi} + N_{disp}$, where N_{disp} and N_{resi} are the abundances of dispersers and residents, respectively. Dispersal plasticity at emigration was then quantified as the effect of local temperature on dispersal rate (see below). We quantified cell morphology as cell size and cell shape (i.e. the ratio of cell major/minor axes). We characterized cell movement as velocity (i.e. the total distance travelled by cells divided by the duration of the trajectory) and trajectory linearity (ratio between the net distance travelled and the total distance effectively moved through a more or less tortuous way) [47]. Because strains reproduce clonally in laboratory conditions, differences in trait values between replicated environmental conditions for a given clonal strain result from the expression of phenotypic plasticity [48].

In a small complementary experiment, we investigated the co-occurrence and covariation of morphology, movement and dispersal plasticity depending on the rate and the amplitude of thermal changes. We used three of the 12 strains used in the main experiment (D2, D3 and D6). We exposed them for 3 h to three temperatures: 23°C, 35°C and 31°C, an additional temperature corresponding to the upper margin of 80% of the area under a Gaussian distribution representative of the averaged thermal tolerance curve in this species [23]. We quantified the influence of the rate of thermal changes on local phenotypic plasticity and dispersal plasticity by setting up either a fast (i.e. immediate thermal change with thermic inertia from 23°C to either 31°C or 35°C) or a gradual (i.e. gradual increase every five minutes over three hours from 23°C to either 31°C or 35°C) increase in temperature. An immediate increase in temperature corresponds to the commonly used treatment to characterize phenotypic plasticity with reaction norms (i.e. the relationship between a given trait and an environmental gradient). The rest of the design was similar to the one described above. We performed five replicates per strain, temperature, thermal change and dispersal treatment (n = 150).

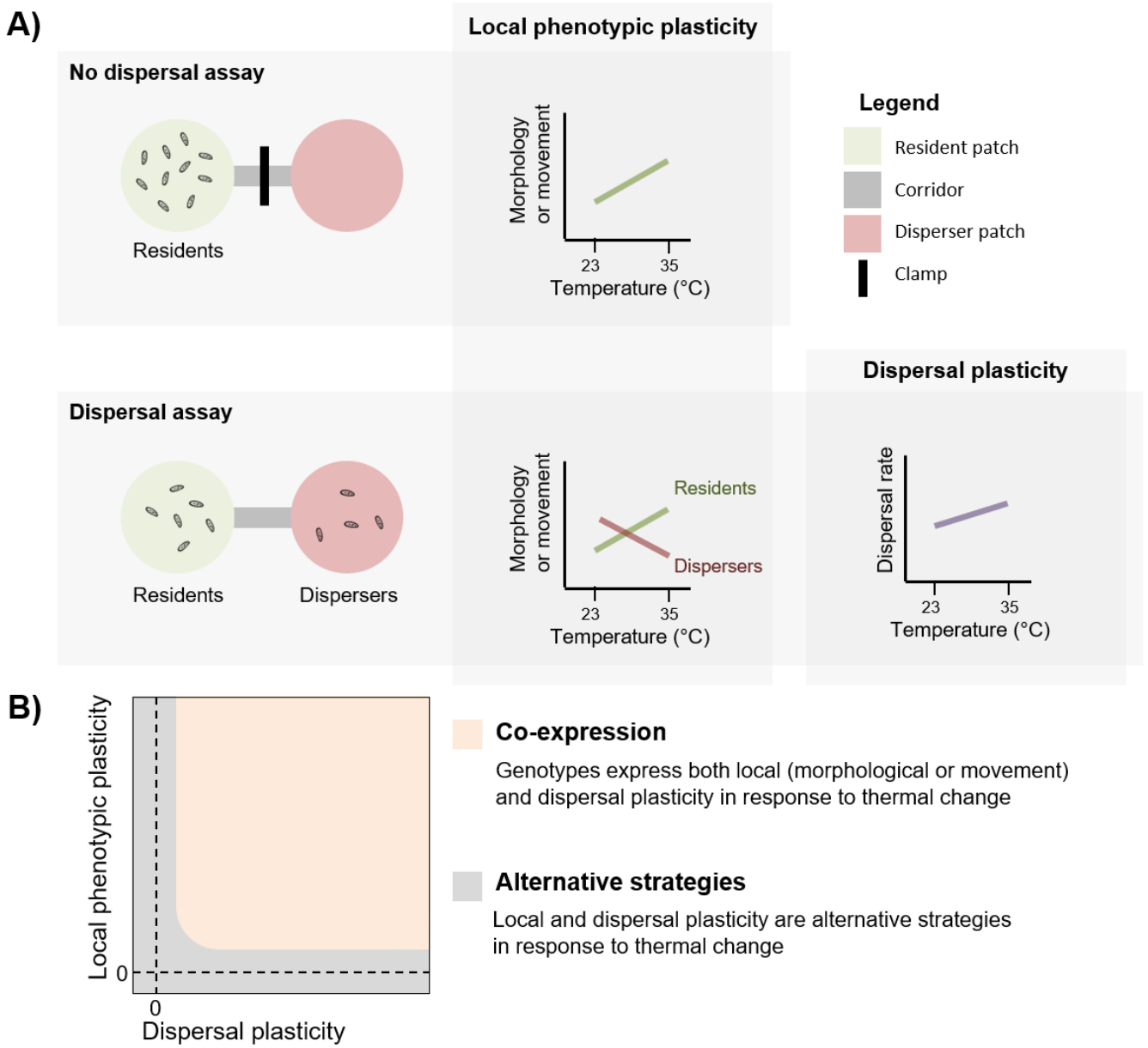

Figure 1. (A) Experimental setup to quantify local phenotypic plasticity (cell morphology and movement) and dispersal plasticity. We inoculated the two-patch systems with a culture of one of the 12 strains and placed them at either 23°C or 35°C. We measured morphological and movement traits in systems without dispersal (no dispersal assay; $n = 120$) and for residents and dispersers (dispersal assay; $n = 120$) and reconstructed reaction norms of cell morphology, movement and dispersal rate along the temperature axis. The reaction norms presented here are for illustration only (random slopes attributed for residents, dispersers and dispersal rate). (B) Predictions for the relationship between local plasticity (morphological and movement) and dispersal plasticity in response to thermal change. Genotypes expressing both local plasticity (morphological and/or movement) and dispersal plasticity would be in the orange zone of the plot (coexpression), while genotypes expressing either local or dispersal plasticity would be in the grey zone (alternatives). On zero the genotypes are not plastic at all.

(c) Data analysis

First, we quantified morphology, movement and dispersal plastic capacity for each strain along the thermal axis and investigated if these plasticity types co-occurred and covaried (figure 1B). To do so, we modelled for the main experiment, morphology and movement without dispersal and dispersal rates depending on temperature (two levels: 23°C and 35°C), strains (12 levels) and their two-way interactions. All morphological and movement traits were standardized (i.e. centred and scaled within traits) to allow effect size comparison between traits. We included our block-day as a random effect to account for temporal effect (two blocks). We verified model assumptions with the *DHARMa* package [61]. To correct for heteroscedasticity of the residuals, dispersal rates were modelled using a beta-binomial error distribution and a logit link function using the *glmTMB* function from the *TMB* package [62]. Morphological and movement traits were modelled using a Gaussian error distribution and the *lme4* package. *p*-values were adjusted with *holm* correction to account for multiple testing when appropriate. The significance of the effects was tested using Wald type III analysis of deviance with Likelihood-ratio tests. Adjusted r^2 for the whole model and each factor and interaction within were obtained with the *r.squaredLR* function of the *MuMIn* package [63]. Factor levels were compared by Tukey's HSD *post hoc* comparisons of all means, using the *emmeans* package [64], to extract the effect size of temperature on traits for each trait and each strain quantifying plastic capacities. We tested for correlation between pairs of raw

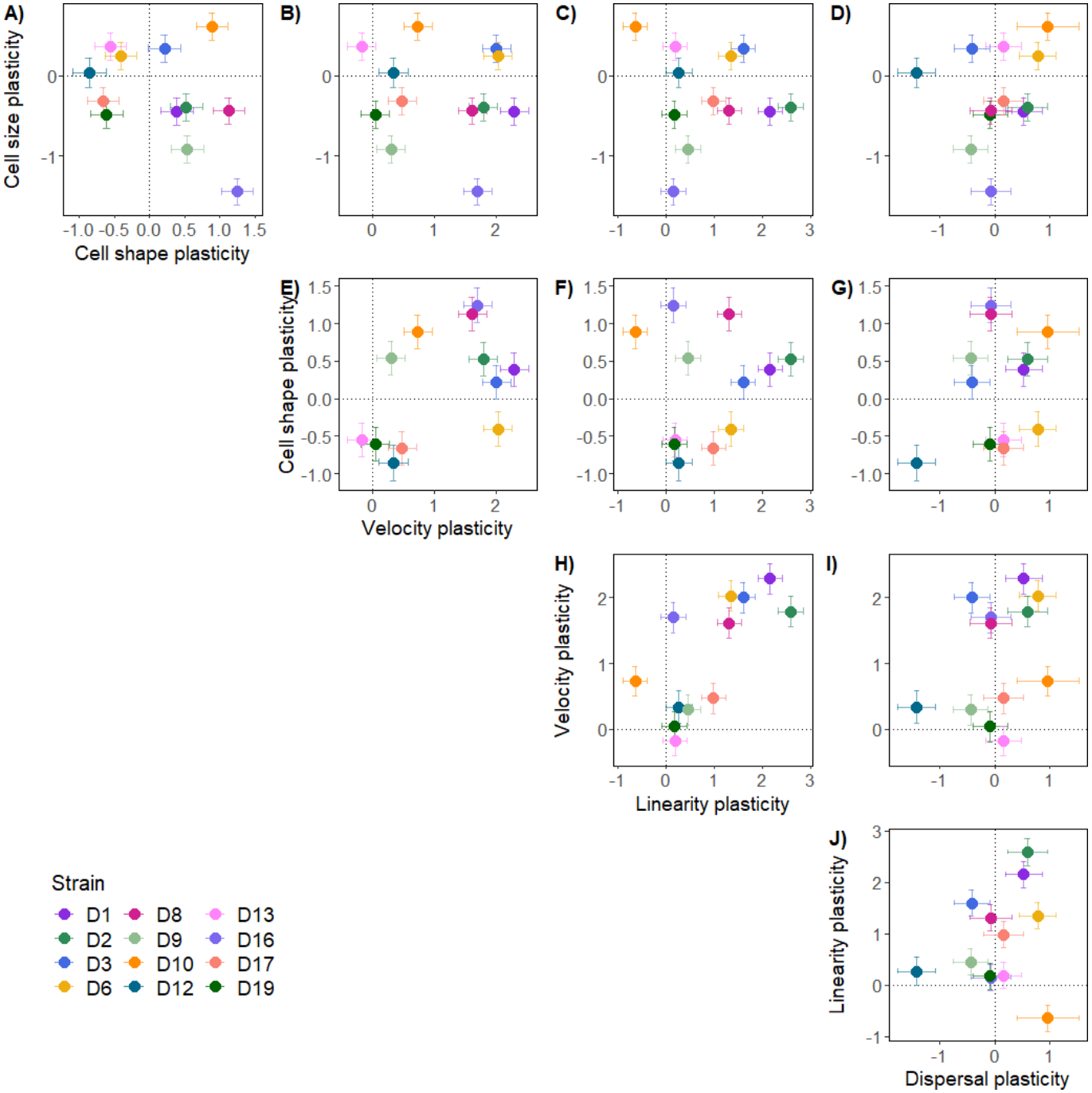

Figure 2. Co-occurrence of morphological (cell size and shape), movement (velocity and linearity) and dispersal plastic capacity. Points represent the absolute effect size of thermal change for each strain (absolute estimates ± 95% CIs). Horizontal and vertical dotted lines situate the effect size values corresponding to no plasticity. Points with errors non-overlapping these lines represent significant plasticity.

traits in each condition (electronic supplementary material, table S1 and figure S1), pairs of directional plasticity indices (effect sizes; hereafter, plasticities), and pairs of plastic capacities (absolute effect size) with the Spearman's rank test (*cor.test* function). The relationship between absolute values of plasticity allows us to investigate whether the different dimensions of plasticity are alternative or not, regardless of their direction, while the relationships between directional plasticity indices allows us to look at specific associations between these plastic strategies. All analyses were performed using R 4.2.1 [65].

For the complementary experiment, we modelled morphology and movement without dispersal, and dispersal rates depending on temperature (three levels: 23°C, 31°C and 35°C), strains (three levels) and their two-way interactions for each rate of thermal change. We then tested whether morphological, movement and dispersal plasticity (i.e. differences between standard and warmer temperatures) differed depending on the rate of thermal change (two levels: immediate and gradual), the amplitude of thermal change (two levels: 31°C and 35°C), the strains (three levels: D2, D3 and D6) and their two- and three-way interactions. We investigated whether correlations between the different plasticities changed when the thermal change was gradual compared to immediate change using Spearman's rank correlation.

Second, we tested whether the opportunity to disperse changed how strains performed morphological and movement plasticity compared to when dispersal was not allowed. To do so, we included dispersal status (three levels: without dispersal, residents in treatment with dispersal, and dispersers) in our models, and the two- and three-way interactions with temperature

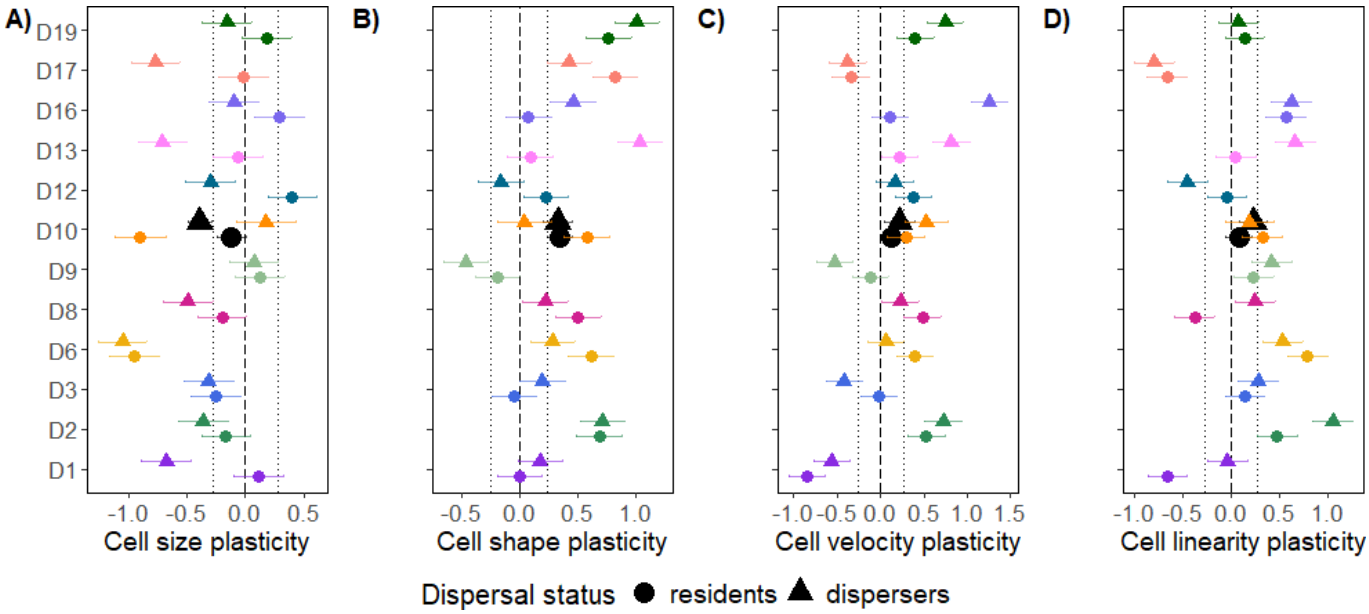

Figure 3. Changes of cell (A) size, (B) shape, (C) velocity and (D) linearity plasticity between residents and dispersers compared to when dispersal was not allowed. Points represent the effect size of thermal change for each strain (colours) and dispersal status (circles for residents, triangles for dispersers) recovered from the models (contrasts ± 95% CIs) and centred around the thermal plasticity without dispersal (horizontal dash line ± 95% CIs). Big black points represent the mean effects across strains. Points outside the range represent significant differences of plasticity for residents or dispersers compared to without dispersal, while deviation between circles and triangles illustrates differences of phenotypic plasticity between residents and dispersers within each strain.

and strain. The dispersal system identity was included as a random effect to account for biological non-independence. Effect sizes of temperature were computed within each level of dispersal status, as explained above.

3. Results

(a) Local phenotypic plasticity and dispersal plasticity are not alternative strategies

Plasticity varied between strains for all five tested variables (two-way interaction between temperature and strains for cell size: $\chi^2_{(11)} = 134.0$, p-value < 0.0001, $r^2 = 0.084$; for cell shape: $\chi^2_{(11)} = 117.6$, p-value < 0.0001, $r^2 = 0.169$; for velocity: $\chi^2_{(11)} = 163.8$, p-value < 0.001, $r^2 = 0.242$; for linearity: $\chi^2_{(11)} = 149.3$, p-value < 0.001, $r^2 = 0.236$; for dispersal rate: $\chi^2_{(11)} = 30.2$, p-value = 0.0002, $r^2 = 0.109$; electronic supplementary material, table S3 and figure S2). Half of the strains (6 out of 12) significantly expressed the five types of plasticity measured (all four local phenotypic plasticity and dispersal plasticity) (figure 2, electronic supplementary material, figure S3 and table S4). Five strains did not express significant dispersal plasticity, among which two were plastic for all the local phenotypic traits measured (D8 and D17) and the three others for traits related to morphology, but not to movement (D13, D16 and D19; figure 2; electronic supplementary material, figure S3 and table S4).

Despite co-occurrence of the different types of plasticity, there were no significant correlations between local phenotypic (morphology and movement) and dispersal plasticities (i.e. effect size of temperature on traits for each strain; electronic supplementary material, table S5 and figure S3) nor plastic capacities (i.e. absolute effect size; electronic supplementary material, table S6; figure 2). Similarly, we found no significant correlation between size and movement plasticity, but we did find a positive correlation between velocity and linearity plasticity (electronic supplementary material, figure S3, and tables S5 and S6) and a negative correlation between shape and linearity plastic capacities (figure 2; electronic supplementary material, table S6).

(b) Differences in morphological and movement plasticity between residents and dispersers

When dispersal was allowed, morphological and movement plasticity significantly differed compared to without dispersal, with effects that varied across strains and dispersal status (residents or dispersers in the dispersal treatment and individuals in the no-dispersal treatment; three-way interaction between temperature, strains and dispersal status for cell size: $\chi^2_{(22)} = 47.2$, p-value = 0.001; for cell shape: $\chi^2_{(22)} = 57.6$, p-value < 0.0001; for velocity: $\chi^2_{(22)} = 70.8$, p-value < 0.0001; for linearity: $\chi^2_{(22)} = 45.6$, p-value = 0.002; electronic supplementary material, tables S7 and S8). Dispersers tended to become smaller in response to increased temperature than individuals in the no-dispersal treatment, while size plasticity of residents was in most cases similar (figure 3). Mean plastic changes for the other traits were similar between residents, dispersers and individuals in the no-dispersal treatment due to high intraspecific variations in these plastic responses (figure 3). Morphological and movement plasticity significantly changed compared to the treatments without dispersal in 48% of the cases (23 out of 48 [12 strains × four traits]) for dispersers and 29% of the cases (14 out of 48) for residents (figure 3). These differences tended to lead to

new correlations between the different local phenotypic plasticities but no association was observed with dispersal plasticity (electronic supplementary material, tables S5 and S6).

(c) Effects of the rate and the amplitude of thermal change on morphological, movement and dispersal plasticity

Morphological plasticity was higher in case of a fast change in comparison with a gradual thermal increase and a higher thermal change (electronic supplementary material, figures S4 and S5, and table S9). These differences led to some changes in correlation between the different types of plasticity (electronic supplementary material, tables S10 and S11). As in the results above, marked differences in the response to thermal changes were observed between residents and dispersers (electronic supplementary material, figure S6 and table S12).

4. Discussion

Although local phenotypic plasticity and dispersal plasticity may be considered as alternative mechanisms [2,25], theoretical and empirical studies pointed out that they can instead both be expressed in the same organisms [31,33–35,66,67]. Accordingly, our experiment demonstrated that local phenotypic plasticity and dispersal plasticity were not alternative strategies in our study system: half of the twelve *T. thermophila* strains tested here expressed simultaneously morphological, movement and dispersal plasticity. This result highlights that these two dimensions of plasticity, instead of alternatives, may sometimes evolve together. For instance, dispersal plasticity may benefit from the expression of phenotypic plasticity to deal with environmental conditions encountered during or after dispersal [35]. Even though it can be conceptually helpful to separate dispersal and phenotypic plasticity, dispersal plasticity is a form of phenotypic plasticity where dispersal is the plastic trait. Both phenotypic and dispersal plasticity require environmental variability with somehow reliable environmental cues to evolve [25,31]. Future studies should investigate whether they may, in some organisms, share specific underlying mechanisms such as sensory structures, explaining why they could co-occur.

In spite of both phenotypic and dispersal plasticity being expressed in most of the strains tested, we did not find significant correlations between the plastic capacities of morphological, movement and dispersal. This suggests that these plasticity dimensions, despite co-occurring, might have evolved independently and be favoured by different environmental contexts. Indeed, although cell morphology, movement and dispersal were previously found to be correlated in *T. thermophila* [43,44,47,51,56], we did not find correlation between their plasticity or their plastic capacity. Especially the absence of correlation between thermal plasticity of cell movement and that of dispersal shows that these two dimensions of plasticity are not necessarily two sides of the same mechanisms. Cell movement plasticity could enhance dispersal plasticity but did not explain the changes in dispersal induced by thermal change. Indeed, strains with the highest dispersal plasticity also showed relatively weak movement plasticity. Dispersal and movement plasticity may represent different, partly independent responses to thermal change.

Further, some theoretical work predicts that the evolution of adaptive phenotypic plasticity should be more common than the evolution of adaptive dispersal plasticity because the mismatch between the phenotype and the new environment can be more easily resolved with changes in the local phenotype rather than by finding the right environment [25,31]. However, in our experiment some strains showed weak dispersal plasticity and high plasticity for other traits, other strains showed the opposite, and others had more intermediate values for all types of plasticity. These results suggest that some strains may be more specialized to deal with local conditions through plasticity, others might be more specialized to adjust their emigration decisions, and the rest might perform both strategies. Hence, we did not find evidence for the evolution of one of these strategies over the others.

Interestingly, we observed that the correlation between morphological and movement traits at 23°C disappeared under warmer thermal conditions (35°C). The loss of phenotypic integration—the pattern of functional, developmental and/or genetic correlation among phenotypic traits [68]—under stressful conditions was also observed in the perennial shrub *Lepidium subulatum* (Brassicaceae) [69]. Whether such differences in patterns of covariation between traits depending on environmental conditions [70] may constrain or on the contrary favour plasticity [69], and whether the resulting different dimensions of plasticity should correlate altogether or not are open questions. In addition, phenotypic traits tended to homogenize at the intraspecific level under warmer conditions while intraspecific variability was important at ambient temperature, and the direction of plastic phenotypic changes differed across strains (electronic supplementary material, figure S1). This trait homogenization at warmer temperatures could be a result of adaptive plasticity, whereby traits would converge towards specific values that may enhance thermal tolerance or of shared physiological effects at high temperatures. Such changes in intraspecific variability may have important consequences for ecological and evolutionary dynamics [71,72], especially if phenotypic plasticity leads to rapid trait changes that may help organisms to deal with environmental change. Here, we looked at morphological, movement and dispersal plasticity but many other traits could have responded to thermal change and covaried with the plastic traits studied. For instance, the expression of heat-shock proteins (HSPs) is an important part of most organisms' response to thermal change [73,74] and could be coupled with other adaptive mechanisms such as changes in morphology if linked to fitness.

We furthermore showed that the extent of phenotypic plasticity in morphological and movement traits significantly changed when strains were allowed to disperse compared to the no dispersal setup, and between dispersers and residents within the dispersal treatment. These results raise the question of a potential role of phenotypic plasticity in helping organisms to disperse and to improve settlement probability [75,76]. One striking example found in the literature is the expression of a caudal cilium,

thought to help swimming movement, in 10% of *T. thermophila* cells of the same strain under starvation, an environmental condition known to induce dispersal behavior [57]. Further, dispersers tended to be more morphologically plastic than residents (i.e. steeper reaction norms; electronic supplementary material, figure S2). Especially, dispersers tended to become smaller in response to increased temperature compared to residents and individuals in the no-dispersal treatment. This hints towards local phenotypic plasticity helping dispersal, and especially size plasticity. These plastic changes could be due to the dispersal movement itself, for instance because of dispersal costs or the expression of specific phenotypic attributes favouring dispersal, or indirectly due to differences in environmental conditions induced by dispersal [38,43,77]. Indeed, when organisms disperse depending on conditions along an environmental axis, variations along other environmental axes are often associated. In such case, the phenotypic changes observed may not be due to the environmental factor inducing dispersal plasticity but to other associated environmental changes. For instance, dispersal can lead to changes in local population densities, a biotic gradient along which traits may change [78], including dispersal decisions [79,80]. In addition to local/temporal changes in environmental conditions, phenotypic differences between dispersers and residents within an isogenic strain, hence without genetic variance, could also be due to non-genetic mechanisms (e.g. differences in gene expression, methylation, small RNA contents and other molecules transmitted by the cytoplasm between cells), to developmental noise, to residual phenotypic variance [81] or to the unique genome organization of ciliates. Indeed, ciliates possess two nuclei, one hosting their germline that is silent except during sexual reproduction, the other hosting their highly polyploid and fragmented somatic genome (about 45 copies of 181 chromosomes in *T. thermophila*), responsible for phenotypic expression. This somatic nucleus divides amitotically during the clonal phase, meaning that the chromosomes are not equally segregated and that cells do not receive the exact same number of copies [45,82]. This means that while the genomic sequence does not vary (at the exception of a few *de novo* mutations that should remain negligible within the short timeframe of the present experiment), the chromosomal copy number does, albeit some unknown regulatory mechanisms seems to avoid chromosomal loss [83]. Environmental variation, non-genetic mechanisms, developmental noise and chromosomal content can all induce phenotypic variance within isogenic strain potentially influencing dispersal, and/or creating differences in phenotypes between residents and dispersers as observed here and in other studies. However, this should be considered cautiously because the effect of dispersal status on phenotypic changes remained weak, with important differences between strains. Strain identity explained the most part of the phenotypic response observed, as in [56,84].

Here, we cannot determine whether the phenotypic changes occurred before the initiation of dispersal movements, at the transience phase, or after dispersal during the colonization of the initially empty patch. This pleads for the development of studies capturing the plastic changes occurring at each dispersal step through individual tracking. Indeed, one key element to understand what drives the co-occurrence between phenotypic and dispersal plasticity may lay in the relative timing of such plastic changes. One crucial aspect of plasticity that should be considered when investigating the relationships between phenotypic and dispersal plasticity is their timings [6,34,85]. If the environment changes faster than the time needed for phenotypic changes to take place (i.e. plasticity rate) [6,34,85] or at an amplitude higher than the capacity for plasticity, phenotypic plasticity may be insufficient for organisms to locally deal with new environmental conditions [6,67,85] and dispersal may be favoured. With thermal changes for example, we can expect high amplitude of thermal change compared to the organism's thermal limits [86] and fast thermal changes to favour dispersal plasticity at the expense of local phenotypic plasticity. In the complementary experiment, we showed that the rate and amplitude of thermal changes had a stronger effect on the expression of morphological plasticity than movement and dispersal plasticity. Indeed, the results hint at a latency time for morphological plasticity to be mounted in response to thermal change while plasticity in movement and dispersal were almost immediate. We also observed that immediate versus gradual changes in thermal regimes modified the covariation between some types of local plasticity but not between local phenotypic and dispersal plasticity. These complementary results suggest that, in our system, the rate and the amplitude of environmental changes might have limited impact on the interplay between local phenotypic and dispersal plasticity, but a deeper exploration of the rate and amplitude of environmental changes is necessary.

5. Conclusion

Our study demonstrated that local phenotypic plasticity and dispersal plasticity were not alternative mechanisms, and that how they associated with each other varied intraspecifically. Considering the potential interactions between local phenotypic and dispersal plasticity is therefore crucial for our understanding of ecological and evolutionary dynamics. For instance, phenotypic plasticity associated with adaptive dispersal plasticity may allow adaptive divergence by creating reproductive barriers between alternative phenotypes and reinforcing mate choice [32]. Initial plastic divergence could be followed by the evolution of reduced phenotypic plasticity with genetic differences between phenotypes (i.e. genetic assimilation) [32]. This reinforcement by morphological plasticity of phenotypic divergence caused by adaptive dispersal plasticity has been observed in salamanders (*Gyrinophilus porphyriticus*) [66]. Experimental evolution will be key to understand the relationships between these two processes and to assess whether phenotypic and dispersal plastic changes are adaptive or not [13,48]. In addition, further investigating the environmental scenarios under which either or both local and spatial strategies are selected for [31], especially focusing on the frequency and predictability of environmental changes [9,87–89] and the landscape composition [25], will be key to understand their evolution. Here, we focused on dispersal plasticity at emigration, a key first step of the dispersal process that has been shown to have important consequences on metapopulation dynamics and local adaptation [23,24]. Extending this investigation to dispersal plasticity at immigration, namely the choice of where to settle in a heterogeneous landscape, is a key future step to understand the relationship between phenotypic and dispersal plasticity and their ecological

and evolutionary consequences [90]. Further, changes in population and community contexts may modify how a focal species responds to environmental changes [91], and thus the relationship between phenotypic and dispersal plasticity [92].

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. All raw data and R scripts used for this study are available on the Zenodo repository [93].

Supplementary material is available online [94].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. M.T.: conceptualization, data curation, formal analysis, investigation, methodology, visualization, writing—original draft; L.D.: investigation, writing—review and editing; D.L.: investigation, supervision, writing—review and editing; S.J.: conceptualization, investigation, supervision, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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