Dispersal and eco-evolutionary dynamics in antagonistic species interactions

Authorship

Giacomo Zilio[1, 2, a], Jhelam N. Deshpande[1], Alison B. Duncan[1], Emanuel A. Fronhofer[1], Oliver Kaltz[1, a]

Giacomo Zilio[1, 2, a], giacomo.zilio@cefe.cnrs.fr,
ORCID ID: https://orcid.org/0000-0002-4448-3118

Jhelam N. Deshpande[1], jhelam-nitin.deshpande@umontpellier.fr,
ORCID ID: https://orcid.org/0000-0003-3185-4589

Alison B. Duncan[1], alison.duncan@umontpellier.fr,
ORCID ID: https://orcid.org/0000-0002-6499-2913

Emanuel A. Fronhofer[1], emanuel.fronhofer@umontpellier.fr, ORCID ID:
https://orcid.org/0000-0002-2219-11 784X

Oliver Kaltz[1, a], oliver.kaltz@umontpellier.fr,
ORCID ID: https://orcid.org/0000-0002-7154-0456

[1] Institut des Sciences de l'Evolution - Montpellier (ISEM), University of Montpellier, CNRS, IRD, Montpellier, France

[2] Centre d'Ecologie Fonctionelle et Evolutive (CEFE), University of Montpellier, CNRS, Montpellier, France.

[a] Corresponding authors: giacomo.zilio@cefe.cnrs.fr, oliver.kaltz@umontpellier.fr

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Highlights

Dispersal is a life-history trait of fundamental importance in single- and multi-species systems.

Several studies indicate its central role for antagonistic species interactions, modulating ecological and evolutionary processes, epidemiology, spatial dynamics and patterns of local adaptation.

Dispersal itself can evolve, but only recently theoretical and experimental research has recognized the profound implications of this second-order evolutionary process for antagonistic interactions, including host-parasite, host-parasitoid and predator-prey.

We therefore call for more detailed investigations of dispersal evolution and its impact on critical interaction traits, such as virulence and resistance, and the potential for eco-evolutionary feedbacks.

Abstract

Dispersal fuels the interplay between ecology, evolution and adaptation across spatial and temporal scales. Dispersal also determines the encounter between natural enemies and can produce eco-evolutionary feedbacks with potentially profound consequences for the geographic distribution and genetic diversity of antagonistically interacting species. Although both dispersal and interaction traits, such as virulence or resistance, evolve, their concurrent evolution and impact for these dynamics remain understudied. We advocate for a more comprehensive framework, integrating dispersal, interaction and life-history trait evolution in a multi-species context. This integration may substantially alter our current vision of coevolutionary dynamics, and influence projections of range expansion, biological invasions or spreading epidemics, which is particularly relevant with ongoing global change and habitat alteration.

Main text

Dispersal evolution and antagonistic species interactions

Dispersal is responsible for transporting individuals and their genes across spatially structured populations [1]. It can therefore drive demographic and evolutionary dynamics involved in various spatial processes from local adaptation to range expansions or biological invasions [2–4]. In recent years, the field of spatial ecology has increasingly recognised this role of dispersal as an engine of eco-evolutionary feedbacks [5–7]. Moreover, dispersal itself can evolve [2,8,9] thereby additionally affecting ecological patterns. An iconic example is the evolution of increased dispersal during range expansion of the cane toad in Australia [10] which has coined the term **spatial selection** (see **Glossary**) as an analog to natural selection. Putting dispersal into the context of other correlated traits, the evolution of entire **dispersal syndromes** [11] is known to lead to faster [12] and more diversified range expansion outcomes [13].

While we have advanced our understanding of dispersal and its evolution in a single specie context**,** it is less clear how it might be affected by other species in a community [14,15]. Clearly, interactions with natural enemies are of particular interest: Antagonistic species interactions such as host-parasite, host-parasitoid or predator-prey systems are ubiquitous in nature [16,17]. Antagonists impose strong demographic and evolutionary pressures on their target species [18–21] with effects on single host individuals scaling up to the (meta)population, metacommunity or global level [22]. There is a long-standing history of studying epidemiology [23,24] and host-parasite coevolution from a metapopulation perspective [25,26], from the study of local adaptation [27] to more recent work on the eco-evolutionary dynamics underlying the spatial and temporal variation typical of **antagonistic species** interactions [28]. Dispersal is certainly recognised as a key element in this research, but unlike in metapopulation ecology, questions of evolution focus on **interaction traits** such as resistance, infectivity or **virulence,** and do not consider dispersal evolution. Indeed, most conceptual and empirical work identifies gene flow of the interacting species as one main engine of coevolution and local adaptation [29,30] or virulence evolution [31], and dispersal is typically considered a fixed parameter [32,33] rather than an evolvable trait with a genetic basis [9].

Here, we provide a synthesis of the current state of the art and research trends regarding these questions, bringing together rather separated research communities, working on dispersal evolution in the presence of natural enemies (spatial ecology) and interaction trait evolution (host-parasite evolutionary ecology). For simplicity we concentrate on the evolution of constitutive dispersal, even though the evolution of plastic dispersal behaviour represents an important response to changes in external environmental conditions (see Box I). We highlight how dispersal evolution modulates ecological and epidemiological patterns and affects (co)evolutionary trajectories. Given the implications for biodiversity, biological control, conservation and epidemiology, we argue for future investigations into (i) how dispersal coevolves in antagonistic species interactions, (ii) how dispersal evolution modulates the evolution of interaction traits, such as virulence, transmission, infectivity or resistance, and (iii) how the joint evolution of these different traits feeds back on population-level patterns and processes (spread of epidemics, local adaptation).

Evolution of dispersal with fixed interaction traits

Demographic fluctuations and dispersal evolution

In ecology, antagonistic species interactions are of particular interest because of their top-down effects impacting population densities. Most often predators, parasites and parasitoids (**aggressors**) decrease the abundance of their prey and hosts (**defenders**). In turn, low defender availability reduces the aggressor's abundance, thereby generating characteristic demographic fluctuations of aggressor and defender through time [34,35]. The presence of aggressors in a defender population can be seen as a decline in habitat quality and, with spatio-temporal variability in this quality, increased dispersal becomes advantageous, despite potential risks and costs of dispersal [36]. The higher the efficiency of the aggressors, the higher the fitness cost for the defenders. This corresponds to stronger demographic fluctuations and thereby a higher extinction risk for the defenders, leading to stronger selection for dispersal [37–41]. Conversely, low-efficiency aggressors introduce little cost for the defenders, produce weaker fluctuations and reduce selection for dispersal. Similarly, commensalism or mutualism produce negligible fluctuations and have little to no impact on dispersal evolution [38]. Fluctuations in defender populations may lead to the evolution of high dispersal in the aggressor [42]. Taking this one step further, several models studied at dispersal coevolution, in which the evolution of increased dispersal of one species (defender)

alters population dynamics and drive the evolution of dispersal in the antagonistic species (aggressor), creating eco-evolutionary feedbacks [43–45]. Chaianunporn & Hovestadt [39] found that the aggressors counter-respond by evolving higher dispersal to keep up with the high-dispersal defenders. Other studies have addressed the evolution of plastic dispersal responses (Box I) rather than constitutive dispersal. This includes various types of context-dependent dispersal, such as adaptive dispersal behaviours in the presence of enemy signals [46] or altered dispersal of already infected hosts [47].

Impact of habitat heterogeneity and additional trophic levels

The evolution of high dispersal, but also of long-distance seasonal migrations (Box II), results from an evolutionary response to the expected decline in local habitat quality which can be due to the presence of aggressors. However, dispersing individuals may also encounter novel environments and other challenges. Abiotic habitat heterogeneity is usually thought of as a counteracting force that reduces and constrains dispersal [39]. Heterogeneity can be due for example to different resource productivity [41] or variation in home ranges/clusters of the aggressors [48]. In the latter, aggressors gather together and localise in specific zones, creating spatial heterogeneity. This leads to selection against dispersal in defenders to avoid abandoning aggressor-free refugees. The same logic applies to heterogeneity in resource distribution; dispersal is counter-selected if it implies moving from a high-resource environment into a patchy, novel environment that may be lacking resources. Nonetheless, the evolution of increased dispersal can still occur under habitat heterogeneity when, despite the risks, the advantage of dispersal outweighs the costs of staying in a poor and detrimental environment [39]. Dispersal evolution has also been incorporated into more complex models including additional trophic levels. This is the case when the defenders, and not their aggressors, can interact with the available resources [41,49]. Spatial and temporal variation in resource productivity affect population dynamics and may also generate a selective pressure for dispersal evolution. Chaianunporn & Hovestadt [50] show how the introduction of a third species (hyperparasitoids) attacking aggressors reduces the pressure imposed by the latter on defenders, which then evolve lower levels of dispersal. In a study investigating tritrophic plant-pollinator-parasitoid systems instead, intermediate levels of dispersal evolved in each species [51].

In summary, in the majority of these models, dispersal essentially evolves in response to increased local extinction risk imposed by the enemy. Importantly, interaction traits (e.g., "handling time" or "efficiency") are considered set parameters that influence population dynamics, but they are not directly linked to the dispersal traits via allocation rules or genetic trade-offs. Consequently, constraints on dispersal evolution in theoretical models mainly result from ecological costs, such as the risk of death during travel [36].

Evolution of interaction traits with fixed dispersal

Dispersal and local adaptation

While the above models investigate dispersal evolution for different interaction parameters, the majority of host-parasite research is focused on interaction trait evolution in a metapopulation setting, in which the parasite has to colonise patches of hosts. In this context, dispersal is less of a trait than a mere effector of gene flow, reshuffling the distribution of genetic variation in space and time and mediating geographic mosaics of coevolution [52]. A classic example is local adaptation: In spatially structured populations, parasites are expected to adapt to the locally most common host genotypes, and vice versa, hosts to their parasites [53–55], leading to geographic pattern of host or parasite adaptation, depending on which of the two players evolves faster. Gene flow represents one way to increase evolutionary potential, by introducing the right genes at the right time. Thus, all else being equal, the player with the higher gene flow has a higher chance of being ahead in the coevolutionary race and therefore adapt locally [56]. Meta-analyses have corroborated the role of gene flow as a driver of local adaptation in natural populations [29,30], and microcosm experiments have demonstrated its effect on local (co)evolutionary rates by artificially manipulating the amount of bacteria or phages dispersing between host populations [57,58]. While certain levels of gene flow can facilitate the maintenance of polymorphism for hosts and parasites, large-scale gene flow will tend to shortcut coevolutionary processes [59], or even swamp local patterns and override habitat-specific optima of attack and defence [60].

The case of parasite virulence evolution

More recently, spatial structure and dispersal have become of interest in studies of virulence evolution, broadly defined as a decrease in host fitness. Theoretical models typically assume a trade-off between virulence and transmission [61], where increased exploitation of host

resources increases parasite transmission, but also shortens host life-span and thereby the infectious period [62,63]. To balance these costs and benefits intermediate levels of virulence are expected to evolve in a spatially homogeneous population [64,65]. With spatial structure, optimal virulence depends on dispersal [66]. Under low dispersal, parasite transmission becomes limited by local host availability, and thus increased self-shading (i.e., increased kin competition) favours less virulent, prudent parasites [67,68]. In contrast, high dispersal reduces local self-shading and guarantees transmission to new hosts on a global scale to more distant patches. Thus, with sufficient supply of susceptible hosts, more virulent parasites are favoured in spatial grids [69,70] or at the front of advancing epidemics [71]. Experimental tests in microcosm populations [72–74] corroborated these theoretical predictions by artificially manipulating spatial structure/connectedness (but see [75]). Data from natural populations are scarce and interpretations of observed patterns potentially difficult [76,77]. Indeed, in real-life situations, the biology of dispersal becomes relevant. For example, while theory often makes no specific assumptions about how parasites move, many parasites may actually rely on their host for dispersal. If parasites travel with their hosts, additional costs of dispersal may arise, for instance if infected hosts disperse less due to the exploitation by the parasite. Implementing such a dispersal-virulence trade-off into models may therefore produce lower, rather than higher, virulence at the front of an epidemic wave [78], a prediction consistent with observations of an expanding bacterial parasite in the North American house finch [79].

Concurrent evolution of dispersal and interaction traits

Correlated trait responses

The two previous sections described research in fairly isolated scientific communities, treating different, but potentially complementary issues. A straightforward step bridging this gap is to consider the simultaneous evolution of both dispersal and interaction traits (Figure 1). Of course, this is not an entirely new idea. The concerted evolution of dispersal and other traits (e.g., physiology, morphology, life-history and behaviour) is known as a dispersal syndrome in spatial ecology [80]. It has been well investigated for single species, namely in the context of range expansion and biological invasions [12]. In multi-species scenarios, with natural enemies, interaction traits become part of the equation, raising questions of how changes in

interaction traits affect dispersal evolution (and vice versa), whether syndromes exist for both aggressors and defenders, and whether such syndromes might even coevolve.

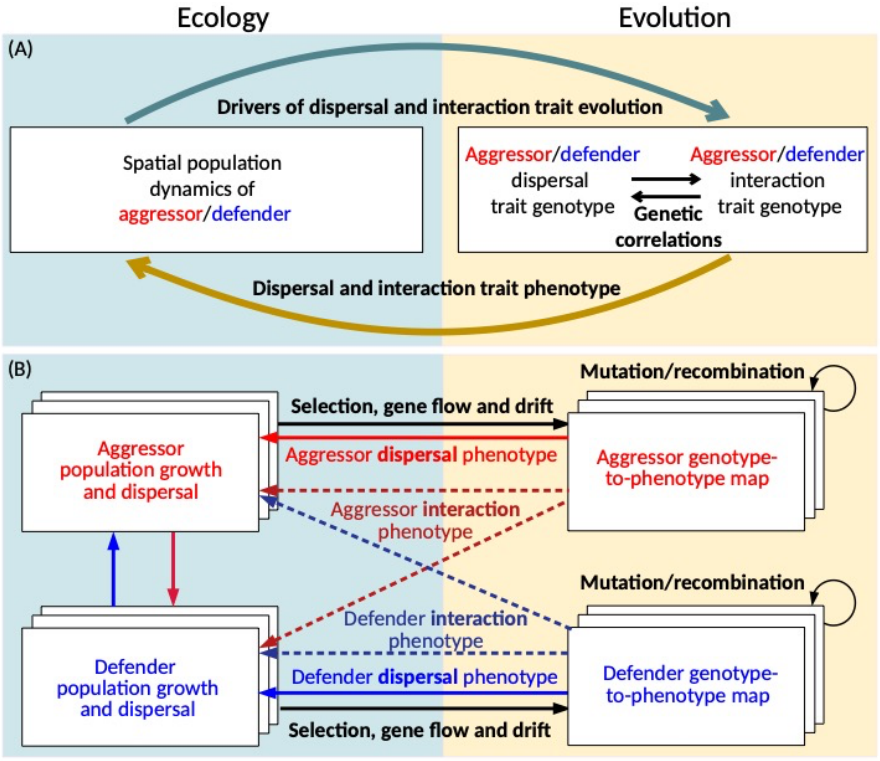

Figure 1. Eco-evolutionary feedbacks between the spatial dynamics of aggressors/defenders (e.g., host-parasite, host-parasitoid, or predator-prey) and the (co)evolution of dispersal and interaction traits. (A) The spatio-temporal ecological dynamics and patterns set the scene for all evolutionary drivers of dispersal and interaction traits (eco-to-evo arrow). Vice versa, the resulting dispersal and interaction trait phenotypes determine demographic rates (evo-to-evo arrow). (B) A more detailed view of this eco-evolutionary feedback loop reveals the relevance of basic demographic rates (birth, death, dispersal) for understanding ecology, which sets the scene for resulting selection, gene flow and drift. These evolutionary forces will lead to trait evolution, which will be modulated by genotype-phenotype maps, mutation and recombination. Importantly, if ecological and evolutionary dynamics are synchronous, that is, play out on similar time scales, this may lead to complex emergent system properties (for an in-depth discussion see [7]).

Answers to these questions depend on the genetic correlations between the different traits and their correlated responses to selection. Several experimental studies have addressed this issue for various biological systems (Table 1). On the defender side, selection for high dispersal reduced predator avoidance in flour beetles [81] or resistance of a protist to infection by a bacterial parasite [82]. Similarly, selection for increased predator defence led to a reduction in motility and dispersal of a green alga [83], while there was no clear link between dispersal of bacteria and their resistance to bacteriophages [84]. On the aggressor

side, selection for increased dispersal of protists or earlier dispersal in a spider mite was negatively associated with their capacities of prey exploitation [85,86]. Along the same lines, adaptation of a bacterial parasite facilitating the dispersal of infected hosts (*Paramecium caudatum*) was associated with lower virulence and reduced investment in horizontal transmission [87]. Conversely, evolution of motility traits of a pathogenic bacterium was positively or negatively associated with changes in virulence expressed in an insect host, depending on the movement selection environment [88]. These few examples already suggest that dispersal and interaction traits do indeed show correlated responses to selection, with trade-offs appearing more often than not. Still, the sign and magnitude of these relationships might vary from system to system, and critically depend on how the organisms move and disperse. Moreover, proper syndromes involve more than two traits, meaning that trait relationships may be even more complex and require multivariate approaches, both in terms of experimental tests and statistical analyses [82].

Table 1. Empirical examples of selection on dispersal and/or interaction traits and correlated responses.

Defender	Aggressor	Focal	Selected Trait	Correlated Trait	Trait relationship	Refs
Galleria mellonella	*Pseudomonas aeruginosa*	Aggressor	High Dispersal	High Virulence	Positive	[88]
Paramecium caudatum	*Holospora undulata*	Aggressor	High Dispersal	Low Virulence	Negative	[87]
Tribolium castaneum	*Amphibolus venator*	Defender	High Dispersal	Low Resistance	Negative	[81]
Paramecium caudatum	*Holospora undulata*	Defender	High Dispersal	Low Resistance	Negative	[82]
Pseudomos syringae	Phage community	Defender	High Dispersal	Resistance	No association	[84]
Bacterial community	*Tetrahymena pyriformis*	Aggressor	High Dispersal	Low Foraging	Negative	[85]
Tetranychus urticae	*Phytoseiulus persimilis*	Aggressor	Early Dispersal	Little Exploitation	Negative	[86]
Chlamydomonas reinhardtii	*Paramecium tetraurelia*	Defender	High Resistance	Low Dispersal	Negative	[83]

Ecological and epidemiological consequences

What are the consequences of concerted multi-trait evolution? For example, the evolution of dispersal syndromes [12,89] is generally expected to accelerate range expansion (Figure 2A-C). However, the inclusion of interaction traits likely produces shifts in multi-trait space, thereby modifying range expansion speed (Figure 2D-F). Using an aquatic model system (*Paramecium caudatum* and a bacterial parasite), Zilio *et al.* [82] studied the dynamics of spatially advancing front populations experimentally. Hosts from infected front populations evolved higher resistance, but also showed lower rates of dispersal and lower population growth rate (in the absence of the parasite) than their uninfected counterparts. This would

imply that parasite-mediated selection leads to reduced expansion speed (Fig. 2F), over and above the speed limits already imposed by negative demographic effects, such as reductions in host density [90]. In contrast, in natural populations of the invasive cane toad, both higher parasite resistance and higher dispersal have been described at the range front [91,92], suggesting that resistance evolution does not interfere with range expansion speed in this system (but see [93]). The scenario depicted in Figure 2D assumes strong parasite-mediated selection, for example with a wide-spread and highly prevalent parasite closely tracking the expanding host population. However, in alternative scenarios, less resistant (and more dispersive) host variants might outrun the parasite geographically (Fig. 2G), creating enemy-free space at the range front, consistent with the enemy-release hypothesis [94]. With reduced selection imposed by the parasite, range expansion speed might remain relatively unaffected by trade-offs with resistance (Figure 2I).

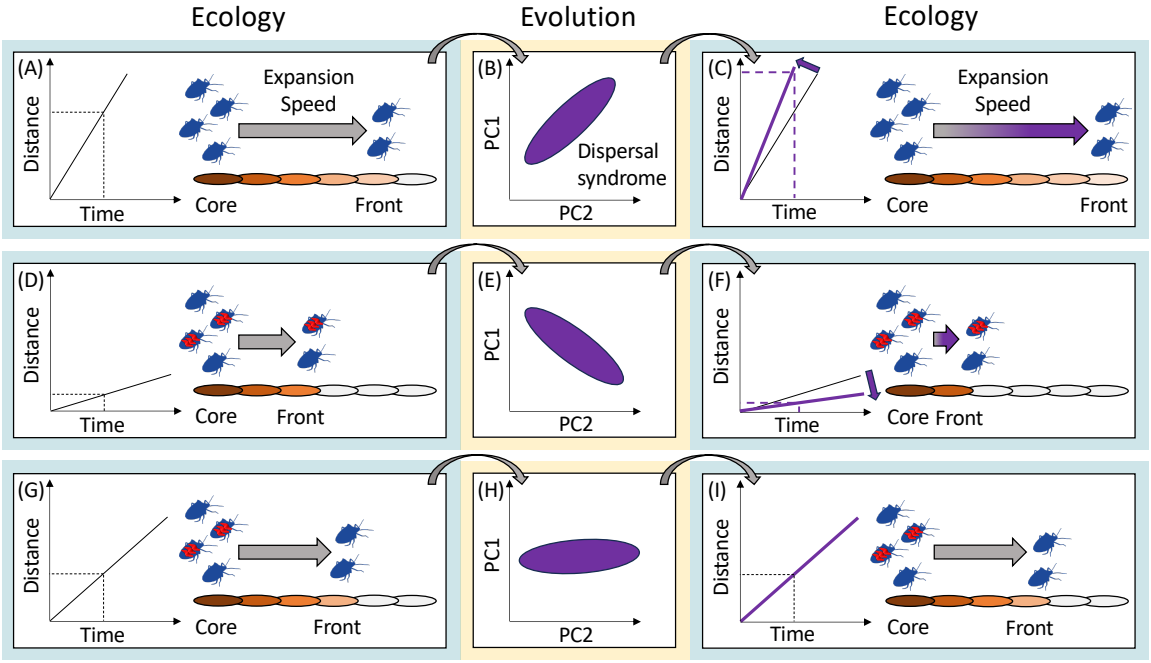

Figure 2. Hypothetical eco-evolutionary feedback loops occurring during a range expansion of a host defender (blue silhouette), in the absence or presence of a parasitic aggressor (red silhouette). (A-C): In the absence of the aggressor (A), ecological conditions set the speed of the range expansion from core to front, expressed as the distance covered over time. At the range front, correlated evolutionary trait change (e.g, dispersal and life-history) produces a dispersal syndrome (B), illustrated as an ellipsis in multivariate space (PCA plot) . The evolution of the dispersal syndrome then feeds back on the ecology, by increasing range expansion speed (C). (D-F): If an aggressor is closely tracking the expanding defender population, range expansion speed is reduced, due to negative demographic effects imposed by the aggressor (D). Evolution of dispersal syndromes at the front now includes interaction traits, and for example, produces a different dispersal syndrome due to a

dispersal-resistance trade-off (E), which further slows down range expansion speed (F). (G-I): If the aggressor is lagging behind the expanding defender front, it only has a small effect on range expansion speed (G). Due to weak aggressor-mediated selection at the front, there are no clear changes in dispersal syndromes (H) and range expansion speed may remain unchanged (I).

Different scenarios of eco-evolutionary feedbacks might also exist in advancing epidemic waves of a parasite. As mentioned above, certain models of parasite evolution predict the evolution of virulent variants at the front of an epidemic, given enough supply of susceptible hosts [71,95]. However, if virulence trades off with dispersal capacity, the opposite prediction might hold [78,79]. Indeed, reduced virulence was found in the bacterial parasite *Holospora undulata,* while travelling with infected hosts in experimental range front populations of *Paramecium caudatum* [87]. Again, how this evolutionary response affects the speed of spatial spread of infection may depend on ecological conditions. For example, if opportunities for host dispersal are rare, such low-virulence variants may have a higher chance of reaching a new patch together with infected hosts and thus enjoy faster expansion, compared to high-virulence variants. However, though moving faster between populations, low-virulence variants may have intrinsically lower rates of local transmission, because of a virulence-transmission trade-off. This might cause less intense infection outbreaks in newly colonised patches. Furthermore, in certain host-parasite systems, a reduction in virulence may be linked to a shift from horizontal to vertical transmission [87,96]. Such a prudent strategy may be favoured in situations where infected hosts arrive in an environment with low host availability. This might contribute to additional reductions in virulence and less interference with host dispersal, and potentially even favour transition towards mutualism in the long run [97].

The emerging picture

These simple illustrating examples indicate that predicted outcomes will not only depend on the underlying genetic architecture of the relevant traits, but also on ecological parameters, such as the connectedness between core and front populations or the type of dispersal of host and parasite (Figure 1). Thus, care should be taken with intuitive predictions, and formal investigations of multi-player and multi-trait evolution scenarios are required. One such theoretical study [98] revisited host-parasite local adaptation, this time allowing for simultaneous evolution of interaction traits and dispersal in the antagonists. On short time

scales, the model predicts low dispersal to evolve in the locally adapted player and high dispersal in the locally maladapted player. This is analogous to evolution in spatially heterogeneous environments, where adaptation to abiotic conditions favours low dispersal, thereby limiting encounters with unsuitable environments. Over longer time scales, host-parasite coevolution produces alternating periods of local adaptation and maladaptation, thereby promoting an arms-race like coevolution of ever-increasing dispersal rates in both host and parasite. These results indicate a more complex picture than that described by classic models without dispersal evolution, predicting that the locally adapted species is the one having the higher dispersal [32]. In another study, addressing host-parasite coevolution from a different angle, Ledru et al. [99] used a spatially explicit simulation model allowing the concurrent evolution of interaction traits and dispersal in both aggressors and defenders. They find that joint evolution of the traits led to a stable coexistence of parasites and mutualists, thereby facilitating the transition from parasitism to mutualism. Their model features an eco-evolutionary feedback loop acting via host density and interspecific competition [99]. Note that neither of these two models assume genetic correlations between interactions traits and dispersal.

Concluding remarks and future perspectives

Dispersal affects the dynamic interplay between ecology, evolution and adaptation across spatial and temporal scales (Figure 1). For single species, a conceptual framework has emerged, allowing eco-evolutionary feedbacks to be assessed under the specific assumption of dispersal as a trait under selection. Here we showed that this also holds in a multi-species context, for interactions between natural enemies. Thus, we argue that dispersal should be considered a trait in its own right, with a genetic basis, together with the diversity of traits defining the interaction between aggressors and defenders (resistance, infectivity, virulence) and other life-history traits. Such a framework may also apply to other biotic interactions such as mutualisms [100,101]. In this view, dispersal might not only evolve concurrently with these traits within each player, but also coevolve between players. This calls for novel theoretical approaches, explicitly specifying assumptions of defender and aggressor dispersal and adopting a multi-trait perspective, incorporating the relevant genetic correlations and trade-offs, including the relationships of dispersal with interaction traits and with other traits such as competitive ability or reproduction [80,85,102–104] (Figure 1). The theoretical and

empirical work reviewed here suggests that such an integrated approach can substantially affect predictions about virulence evolution, the coevolutionary dynamics of local adaptation or the evolution of dispersal syndromes. In turn, these evolutionary changes can be expected to feed back onto the speed of spread of epidemics or the geographic ranges of the interacting players.

On the empirical side, a multi-trait perspective calls for appropriate experiments, allowing covariances to be assessed in multivariate analyses [82] by taking the different measurements on the same experimental unit (individual/clone/cohort). For natural populations, such multi-trait data could be combined with classic field surveys as well as with recent phylodynamic techniques to infer links between spatial structure, dispersal and eco-evolutionary processes [102,105–107]. Alternatively, for sufficiently small organisms, theory can be complemented with laboratory microcosm experiments, in which organisms can disperse naturally [87,108–111] and where the spread of infection and evolution can be tracked over longer time scales. One advantage of this approach is that general trends can be inferred by replicating landscapes and treatments under controlled conditions.

Given the global alteration of habitats with increased opportunities for dispersal and interactions with other organisms to occur [112–114], future theoretical and empirical work on the simultaneous evolution of dispersal and interaction traits stands to increase in relevance. Despite considerable progress in different fields, we still know little about how the spatial dynamics of evolutionary processes over short timescales characterise many ecological patterns, especially when considering antagonist species interactions. An extended conceptual framework may also include a refined vision of landscape modifiers and realistic spatial network topologies (modular, riverine, etc) known to affect epidemiological dynamics [115], or explicit gene-regulatory network approaches [116]. Considering a more realistic representation of these eco-evolutionary dynamics (Figure 1) is therefore crucial, and will help gain new insight on the predictability of their outcomes [117] and guiding management strategies.

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Text Boxes

Box I. Plastic dispersal behaviour and reaction norms

Plasticity and the evolution of dispersal reaction norms [118] is one possible adaptive response against aggressors. Defenders may plastically modify dispersal to escape aggressors (context-dependent dispersal), avoiding extreme reductions in fitness and constitutive costs of higher dispersal [36]. In many situations, adaptive phenotypic plasticity represents a fast and flexible solution, particularly under variable but nonetheless predictable environmental conditions [119,120]. In host-parasite systems, theory has shown that infection prevalence and kin competition can be the main determinant of the evolution of plastic dispersal in infected and susceptible hosts, respectively [121,122]. In these models, infected individuals evolve higher dispersal when there is a chance of parasite release or recovery during dispersal, the costs of dispersal are low relative to susceptible individuals, and the parasite is highly virulent. Dispersal of infected individuals can additionally reduce the risk of infecting susceptible related individuals [47]. The role of kin competition in driving the evolution of dispersal plasticity was additionally demonstrated in a model investigating predator-induced dispersal in a prey [37]. Empirical studies show the presence of both state- and context-dependent dispersal in natural and laboratory conditions. Increased dispersal probability in response to chemical predator cues has been reported in a multi-species experiment [123], consistent with similar findings in other organisms [124,125]. Similarly, infection-avoidance behaviour from uninfected individuals has been observed in natural host-parasite systems [126]. Parasite infection may also induce morphological or physiological changes in the host, triggering modification in dispersal [127], i.e. state-dependent dispersal.

Box II. Evolution of seasonal migration

Seasonal migration differs from dispersal in that it is characterised by the movement of a large number of individuals gathering together, usually occurring at a broad geographical scale between high and equatorial latitudes, corresponding to summer reproduction and wintering areas (for details on seasonal migration see [128]). These long-distance, round-trip movements are widespread among different taxa and have profound consequences for antagonistic species interactions [129–131]. In host-parasite systems, migration may cause the spread of infectious disease such as in migratory birds carrying avian influenza viruses

[132,133] or for other viruses [134]. Wintering or stop-over sites may represent hot-spots where parasites can easily spread in high-density host populations already weakened by the stressful and energy-demanding migration [135,136]. Migratory species should therefore be under strong selective pressure to evolve effective defensive strategies, and recent works suggest that parasite prevalence, infection state and intensity can indeed influence host behaviour and migratory decisions [137–139]. Despite the lack of an exhaustive understanding of how parasites affect and shape ecological and evolutionary trajectories of migration, several studies indicate that parasites can favour host migratory movements and the evolution of migration as means to reduce parasite spread [140]. The "migratory escape" hypothesis suggests that migration helps individuals to move away from highly parasitized areas or infected individuals [141], or even to separate susceptible juveniles from infectious adults [142]. Another possibility is "migratory culling", i.e. due to the physiological costs imposed by parasites, infected individuals are likely to lag behind and perish during a migration, reducing the infection prevalence in the population [143]. Lastly, hosts may recover from infection during the migratory phase across different environments [144] thus favouring the evolution of migration, depending on the fecundity or survival costs of infection. Overall, these findings suggest that long-distance movement decreases infection risk and that recovery from parasite infection during the spatial spread could influence and drive the evolution of migration.

Glossary

Aggressors: the organisms that negatively affect the other organism's fitness, such as predators, parasites and parasitoids

Antagonistic species: biological relationships in which two organisms benefit one to the detriment of the other, examples are prey-predator, host-parasitoid and host-parasite interactions.

Artificial selection: experimental approach whereby organisms are selected, propagated and bred based on certain phenotypic traits

Coevolution: reciprocal selection and evolution of two or more interacting species

Defenders: the organisms whose fitness is negatively affected by the the other organism, such as prey and hosts

Dispersal: movement of individuals with potential consequences for gene flow.

Dispersal syndrome: concurrent evolution of dispersal, physiology, life-history traits or behaviour.

Experimental evolution: replicate populations evolving under the same controlled conditions to investigate evolutionary processes

Interaction traits: traits whose expression is affected by interactions with another species such as virulence or resistance.

Resistance: reduction in the harm caused by other organisms, in a host-parasite context the host ability to limit parasite burden

Spatial selection: the spatial sorting of individuals due to differential dispersal and dispersal-related traits, followed by random mating and resulting in evolutionary changes.

Virulence: reduction in host fitness (via reduced birth or increased death) due to parasite exploitation and reproduction

Outstanding Questions

- How does dispersal evolution affect interaction trait evolution (and vice versa) in antagonistic species?

- How does the genetic relationship between dispersal and interaction traits impact antagonistic species (co)evolution and dispersal syndromes?

- What are the consequences of concurrent evolution of dispersal and interaction traits for eco-evolutionary dynamics?

- Does dispersal coevolve between antagonistic species?

- How do dispersal and interaction traits evolve in spatial networks with realistic topologies?

- Can the integration of joint evolution of dispersal and interaction traits help in making predictions and guide management strategies in increasingly changing environments?