Landscape structure drives eco-evolution in host-parasite systems.

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Abstract

As all biological and many artificial systems, hosts and their parasites are most often spatially structured. Such spatial structure becomes evident when one considers, for example, cities connected by commuters or habitat islands connected by dispersing individuals. Within this spatial context, parasites may change through time due to rapid evolutionary processes, including mutation and selection, which strongly impacts how epidemics unfold. As a consequence, host-parasite systems can only be understood in the light of evolution and in their spatial context. However, past work has largely ignored relevant spatial complexity. How parasites evolve in realistically complex landscapes remains therefore unclear, hampering the translation of theoretical predictions to real ecological systems. Therefore, we here develop an eco-evolutionary metapopulation model of host-parasite dynamics in which hosts and parasites disperse through realistically complex spatial networks. Parasite virulence, a key parasite life-history trait that impacts host fitness (here: reproduction), is able to evolve. Our model therefore captures feedback loops between host demography and parasite evolution in space. In order to gain a general understanding of parasite eco-evolution in space, we analyse our model for spatial networks that represent characteristic terrestrial (represented by random-geometric graphs; RGG) and riverine aquatic (represented by optimal channel networks; OCN) landscapes. We find that parasites may evolve higher virulence in characteristically riverine compared to terrestrial networks. More generally, we explore how a key host trait, dispersal, modulates virulence evolution within these networks. Consistent with previous work, we show that parasite virulence evolution is driven by kin selection, because dispersal and landscape structure both impact patterns of relatedness. Our model yields readily testable predictions, including that terrestrial parasites should be more virulent than aquatic parasites at low dispersal rates and vice versa as dispersal increases. These differences in evolved virulence directly lead to differences in system stability, with more virulent parasites more often leading to their own extinction. Thus, in this study we highlight the role of landscape structure in driving eco-evolutionary dynamics of parasites.

Introduction

All biological and many artificial systems are fundamentally spatially structured. Spatial structure can be represented by networks (Urban and Keitt, 2001) where in ecology and evolution, nodes are habitat patches and links capture dispersal of organisms between these locations. Spatial network structure (topology) is key for understanding ecological patterns and processes such as the spread of perturbations (Gilarranz et al., 2017) and infections (Green et al., 2006; Davis et al., 2008; Chinazzi et al., 2020) as well as range expansions (Rayfield et al., 2023) and the distribution of biodiversity in space (Carrara et al., 2012). Importantly, spatial network structure also drives evolutionary processes: Evolution is impacted directly via gene flow within the network, but it can also be modulated indirectly via selection and drift due to the redistribution of population densities and genotypes within the spatial network (Fronhofer and Altermatt, 2017). Dispersal within the spatial network structure of landscapes is therefore a key eco-evolutionary driver (Govaert et al., 2019; Fronhofer et al., 2023).

Here, we focus on the spatial eco-evolutionary dynamics of one of the most pervasive interactions and, at the same time, maybe one of the most relevant to human and organismal health more generally: parasitism. The spatial network structure of host-parasite systems is critical for understanding epidemiological and (co)-evolutionary dynamics (Parratt et al., 2016; Penczykowski et al., 2016; White et al., 2018). For example, increased spatial clustering has been shown to reduce epidemic spread (Lilley et al., 2018), spatial network heterogeneity may lower epidemic thresholds (Colizza and Vespignani, 2008) and landscape properties impact rates of disease spread along river networks (Carraro et al., 2018). Incorporating complex landscape structures can help predict occupancy and persistence of infection (Hess, 1996): Jousimo et al. (2014) have shown in the *Plantago* (plant)-*Podosphaera* (fungal) natural host-parasite system, that increased resistance likely driven by greater gene flow in *Plantago* at highly connected habitat patches, leads to reduced parasite occupancy, when compared to less well connected patches, whereas from a purely ecological perspective higher dispersal should lead to greater parasite occupancy in more connected metapopulations (Hess, 1996).

While in epidemiology complex spatial structures have often been studied, the consequences of such spatial structures for the evolution of host-parasite systems, and how these novel evolutionary dynamics feed back on epidemiology, has not been studied as extensively. Instead, in evolutionary epidemiology, simplified spatial structures, such as lattice contact networks (Boots and Sasaki, 1999; Lion and Boots, 2010) and homogeneous (Gandon and Michalakis, 2002) or spatially implicit island models (Wild et al., 2009), have been used to understand the mechanisms driving host-parasite (co)-evolution. Particularly, previous work (Messinger and Ostling, 2009) has focused on the impact of such simplified spatial structures on virulence, a key parasite life history trait that can evolve (Frank, 1996; Cressler et al., 2016) and impact

host demography (May and Anderson, 1978; Hudson et al., 1998). Virulence is defined as the extent to which parasites harm their hosts either by reducing host survival (Anderson and May, 1982) or fecundity (O'Keefe and Antonovics, 2002; Abbate et al., 2015). Parasite virulence is assumed to be a by-product of the fact that parasites need to exploit their hosts in order to transmit more (virulence-transmission trade-off hypothesis Anderson and May 1982; Alizon et al. 2009). Models of simplified spatial structures have highlighted the evolutionary mechanisms, particularly, self shading (Boots and Sasaki, 1999) and kin selection (Wild et al., 2009; Lion and Boots, 2010), that drive the evolution of parasite virulence. Generally, spatial structure can limit the availability of resources (hosts) to parasites, and also lead to genetic structuring (relatedness patterns; Lion et al. 2011) which can, in turn, lead to the evolution of reduced virulence as an altruistic strategy. Thus, while the role of spatial structure in driving the evolution of parasite virulence is widely recognised in general (Messinger and Ostling, 2009; Kerr et al., 2006), how realistically complex landscapes impact its evolution is not known.

As a proof of concept, we here consider the evolution of parasite virulence across biomes in characteristically terrestrial versus riverine aquatic landscapes. Interestingly, spatial networks, whether terrestrial or freshwater aquatic have specific properties due to the physical processes that govern landscape genesis. Specifically, all riverine aquatic landscapes exhibit statistical properties that can be captured by optimal channel networks (OCNs) which are generated based on geomorphological processes that shape rivers globally (Rinaldo et al., 2014; Carraro et al., 2020). These networks are dendritic and patches vary strongly in the number of links they have to other patches, which can impact, for example, metapopulation and metacommunity dynamics (Carrara et al., 2012). Similarly, terrestrial landscapes are intrinsically modular, that is, patches can be classified into modules which are highly connected within each other but sparsely connected to other modules because of biological constraints on dispersal distances. This can be captured by random-geometric graphs (RGGs Gilarranz, 2020). Modularity has consequences, for example, for the spread of perturbations in space (Gilarranz et al., 2017).

Specifically, we develop an individual-based discrete-time metapopulation model of host-parasite dynamics in which parasite virulence evolves (Fig. S1). Hosts and parasites are asexual and have non-overlapping generations. Host dispersal is natal, and hosts disperse to neighbouring patches within their spatial network (carrying their parasite) with a fixed dispersal probability (d). Within a patch, host populations grow and compete for resources leading to logistic dynamics (Beverton and Holt, 1957). Death followed by infection of the new host generation follows a Nicholson Bailey type model (Nicholson and Bailey, 1935). We assume that the virulence trait can undergo mutation. Virulence (v) acts on the fecundity of the host and increasing virulence implies increasing transmission ($\beta(v)$; β_{max} gives maximum transmission rate, s slope of trade-off function). Since terrestrial (RGGs; Fig. 1 A) and riverine aquatic (OCNs; Fig. 1 B) landscapes have differing average numbers of links between patches (degree), we design

additional control landscapes (including random, modular and grid landscapes) in order to disentangle the role of average degree versus network topology (e.g., modularity and heterogeneity).

Specifically, we ask the following questions: 1) How does evolutionarily stable virulence differ between terrestrial and aquatic landscapes and what are the spatial network properties that drive these differences? 2) What are the underlying evolutionary mechanisms that lead to differing evolutionarily stable virulence in terrestrial vs. aquatic systems? Finally, 3) What are the ecological consequences of evolved virulence in terrestrial vs. aquatic landscapes?

Results and discussion

Landscape structure impacts the evolution of parasite virulence

Patterns of virulence evolution in terrestrial and riverine aquatic landscapes

We find that evolved parasite virulence differs qualitatively between terrestrial (Fig. 1 A) and riverine aquatic (Fig. 1 B) landscapes: depending on host dispersal (Wild et al., 2009), virulence can evolve to higher values in riverine aquatic landscapes (Fig. 1 C; for a more comprehensive analysis, see Supplementary Material Fig. S2–S3). More precisely, aquatic landscapes show lower virulence than terrestrial landscape at low dispersal, but higher virulence for a relatively wide range of intermediate dispersal rates.

More generally, these evolutionary effects of landscape type hold for greater maximum transmission ($\beta_{max} = 10$) and for different shapes of the virulence-transmission relationship (saturating: s = 0.5; linear: s = 1; accelerating: $s = 2$; Fig. S2). However, for lower transmission ($\beta_{max} = 4$) and host population intrinsic growth rate ($\lambda_0 = 2$; Fig. S3) virulence evolution depends only on host dispersal and not on landscape structure. This is likely because in our model high virulence and transmission rates lead to strong oscillations in host-parasite densities (Deshpande et al., 2021). Going out of this dynamical regime (see Fig. S4) reduces the differences between the two landscapes because lower amplitude oscillations do not induce local extinctions of the host, thus host availability is not limited.

What network properties explain parasite virulence evolution?

Since the processes that generate terrestrial (Gilarranz, 2020) and aquatic (Rinaldo et al., 2014) landscapes are fundamentally different, these networks differ in a range of properties, including their average degree, that is, the average number of links between patches (see Fig. 2 A). Using a series of control landscapes where we can fix the average degree while varying other properties (see Fig. S5 and S6 for details) we show that the results presented in Fig. 1 are not due to mere differences in the average number of links. Rather, virulence evolution in OCNs is mainly driven by the presence of large numbers of

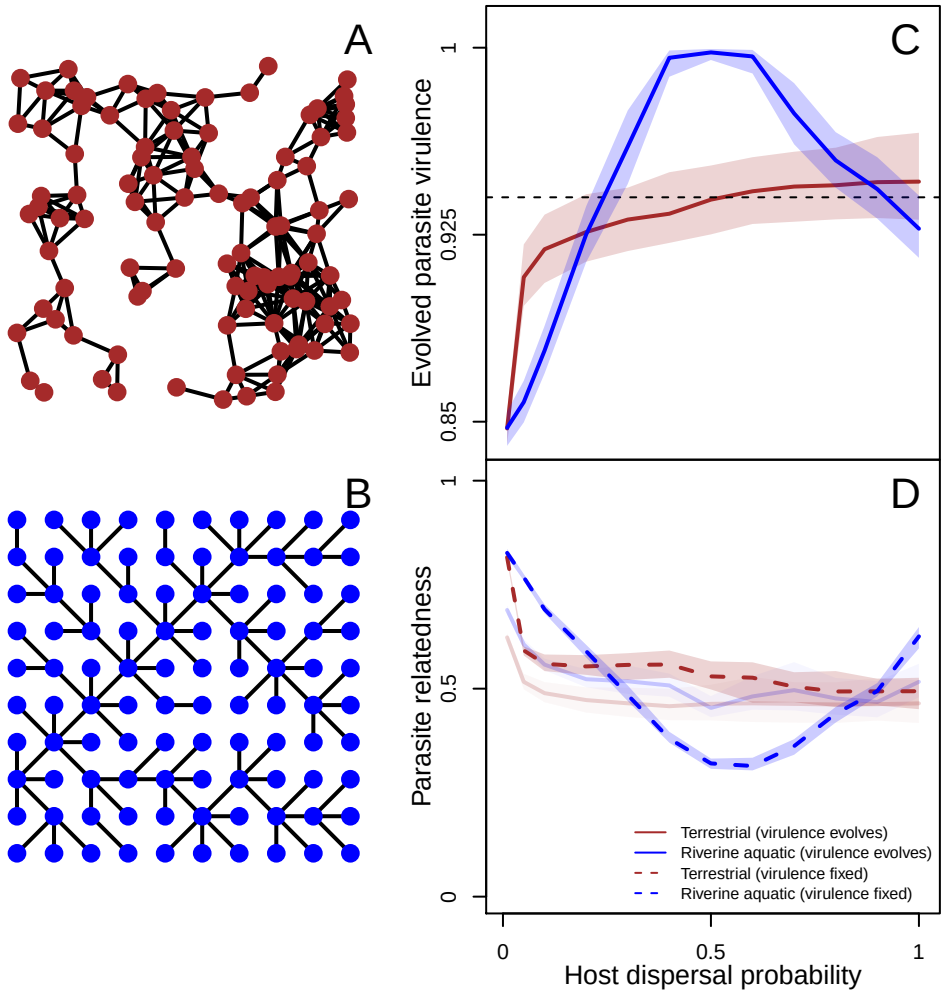

Figure 1: Terrestrial (A) and riverine aquatic landscapes (B) lead to differences in virulence evolution (C) driven by diverging patterns of parasite relatedness (D) in the two networks. A: A representative terrestrial landscape modelled as a random-geometric graph (RGG). B: A representative riverine aquatic landscape modelled as an optimal channel network (OCN). C: Evolved parasite virulence as a function of host dispersal in aquatic (blue) and terrestrial (brown) landscapes. The solid line represents the median and shaded regions the inter-quartile range of the evolved virulence trait over simulations in 1000 realisations of each landscape type, median over all parasites at the last simulation time step ($t = 2500$). D: Average parasite relatedness as a function of host dispersal in terrestrial (brown) and aquatic (blue) landscapes within a patch. The dashed line indicates the measured relatedness for an ecologically fixed virulence of $v = 0.94$. We plot the average parasite relatedness over the last 100 time steps within a patch over all patches over 100 replicates. Details of landscape generation and calculation of parasite relatedness are found in the Methods and Supplementary Information. Model parameters of the focal scenario: intrinsic growth rate of the host $\lambda_0 = 4$, intraspecific competition coefficient of the host $\alpha = 0.01$, maximum parasite transmission $\beta_{max} = 8$, shape of trade-off curve $s = 0.5$, searching efficiency of parasite $a = 0.01$.

headwaters, that is patches that are only connected to one other patch (Fig. 2 A; Fig. S5). Our results for RGGs critically depend on networks being spatially structured and, to a lesser degree, being modular (Fig. S6).

Landscape structure drives parasite virulence evolution via kin selection

Clearly, network topology is an important driver of parasite virulence evolution. But what are the relevant evolutionary mechanisms and how are they modulated by landscape structure? Previous work on virulence evolution highlights the central role of kin selection in driving parasite virulence evolution in spatially structured host-parasite systems (O'Keefe and Antonovics, 2002; Wild et al., 2009; Lion and Boots, 2010). In models of fecundity virulence like ours, it is expected that virulence reaches its maximum possible value in the absence of spatial structure ($v = 1$; O'Keefe and Antonovics 2002). This is because increased virulence leads to increased transmission. However, increased fecundity virulence also reduces the number of hosts available locally since fewer hosts are born. Thus, if locally parasites are related to each other (that is, they share identical alleles), this should lead to the evolution of reduced parasite virulence as an altruistic strategy in spatially structured systems. Since landscape structure and host dispersal can modulate both patterns of relatedness and availability of host, we predict that landscape topology drives patterns of relatedness and host availability which, in turn, define the evolution of virulence.

Patterns of virulence evolution are consistent with within-patch relatedness in terrestrial vs. aquatic landscapes

In order to test this hypothesis, we measure parasite relatedness within a patch by tracking a neutral marker locus in our individual-based model (details can be found in Methods and SI Appendix), and we measure host population densities through time for different landscapes (geometric mean host density as a long term measure of host availability). Since individual-based models are intrinsically eco-evolutionary and may exhibit complex feedbacks loops, we generate these expectations using additional simulations in which we fix virulence and do not allow for any evolutionary change (Fig. S7–S9).

In Fig. 1 D ($v = 0.94$, all other virulence values are found in Fig. S7) we show that patterns of within-patch relatedness are consistent with virulence evolution (Fig. 1 C) as follows: 1) The initial decrease in relatedness (increase in virulence) as a function of dispersal is lower in the riverine landscapes compared to terrestrial landscapes, 2) riverine aquatic landscapes attain a lower minimum relatedness (and maximum virulence) compared to terrestrial landscapes and 3) Relatedness increases (virulence decreases) in riverine aquatic landscapes at high dispersal rates. Further, in all the control landscapes (Fig. S8–S9), relatedness patterns are also consistent with virulence evolution. Thus, we show that differences in virulence evolution in terrestrial and aquatic landscapes are explained by differing patterns of neutral genetic diversity in the two landscapes, as captured by within-patch relatedness.

What are the network properties that explain differences in within-patch relatedness in terrestrial vs. aquatic landscapes?

We now seek to understand the network properties that drive differences in within-patch relatedness between terrestrial and riverine aquatic landscapes. As shown above, the shape of the degree distribution is relevant, particularly the large amount of degree 1 patches in OCNs (Fig. 2 A). For both landscapes parasite relatedness is low in patches with high connectivity and high in patches with low connectivity (Fig. 2 B). Further, at lower dispersal rates (up to $d = 0.1$), local connectivity dominates and all landscapes have similar distributions of parasite relatedness given a patch degree. But since there are many more degree-1 patches in the riverine aquatic landscapes (Fig. S10), overall relatedness is greater. As dispersal increases, due to the heterogeneity in degree distribution in riverine aquatic networks, there is greater dispersal out of low-connectivity patches and lower dispersal into them (Fig. S11; Fronhofer and Altermatt 2017; Altermatt and Fronhofer 2018), which leads to relatively lower infected densities in low degree patches and larger infected densities in high degree patches relative to terrestrial networks (Fig. 2 C). This implies that the parasite rarely goes extinct in high degree patches and recolonises low degree patches of aquatic landscapes, reducing the effect of genetic drift in the landscape and leading to lower relatedness relative to terrestrial landscapes (Fig. 2 D). Finally, as dispersal increases further, infected densities are further reduced in low degree patches, which leads to increasing extinction in high degree patches, and overall greater relatedness.

Closing the eco-evolutionary feedback loop: Ecological consequences of virulence evolution in aquatic and terrestrial systems

As shown above, evolutionary optima differ between the two landscape types (Fig. 1). Further, except for very low dispersal rates (see Supplementary Information Fig. S12 and Fig. S13), virulence as a function of patch connectivity (degree) is homogeneous (Fig 3 A–C). In the following, we ask how evolved parasite virulence feeds back on spatial host-parasite dynamics?

As an illustrative example, in Fig 3 D–F, we focus on the differences in spatial distribution between the two landscape types for one key epidemiological variable: local parasite extinction, or the fraction of time there is no parasite in a given patch, as a function of patch connectivity (degree). As seen earlier in Fig. 2, at relatively low dispersal rates ($d = 0.1$; Fig 3 D), from a purely ecological perspective, low connectivity patches have similar high local parasite extinction for a fixed equal virulence in terrestrial and aquatic landscapes. But evolution of lower virulence in riverine aquatic landscapes leads to reduced local parasite extinction irrespective of connectivity relative to terrestrial landscapes. At intermediate dispersal ($d = 0.5$; Fig 3 E), without accounting for differences in virulence evolution, one would predict lower

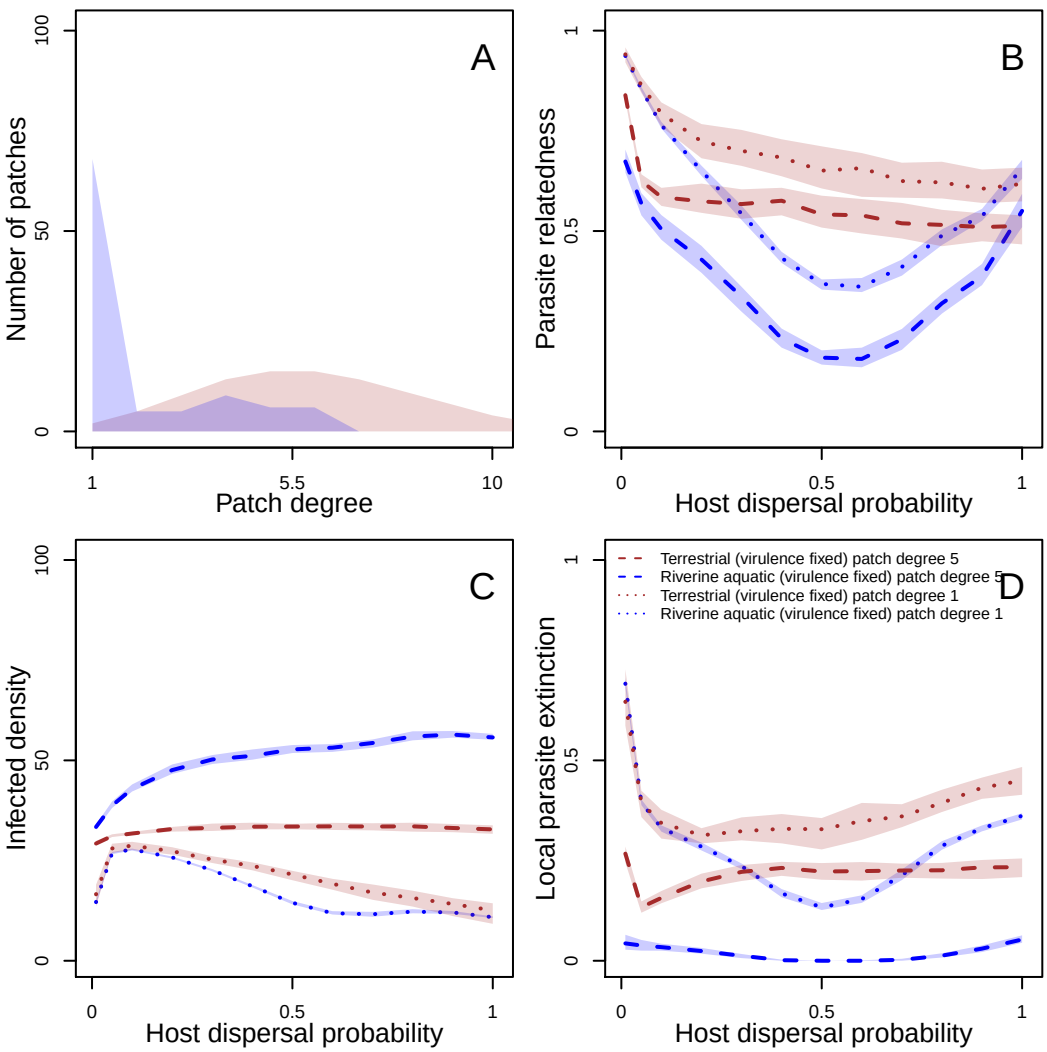

Figure 2: Degree distribution (A) and parasite relatedness (B), infected densities (C) and local parasite extinctions (D) as a function of dispersal for low (degree-1) and high (degree-5) connectivity patches for riverine aquatic and terrestrial landscapes. A: Degree distribution of terrestrial vs. riverine aquatic landscapes. The x-axis represents patch degree and the shaded region shows the median frequency of a patch of a given degree over 1000 landscape realisations for both landscape types. B: Parasite relatedness as function of dispersal rate for low and high degree patches. C: Average density of infected individuals for low and high connectivity patches in the landscape as a function of host dispersal over the last 100 time steps. D: Local parasite extinction calculated as the fraction of time over the last 100 time steps for which the parasite is extinct for low and high connectivity patches. For all plots, medians are taken over for 100 and 1000 realisations for ecological and evolutionary simulations respectively. Model parameters of the focal scenario: intrinsic growth rate of the host $\lambda_0 = 4$, intraspecific competition coefficient of the host $\alpha = 0.01$, maximum parasite transmission $\beta_{max} = 8$, shape of trade-off curve $s = 0.5$, searching efficiency of parasite $a = 0.01$.

parasite extinction as a function of connectivity throughout the landscape in riverine aquatic relative to terrestrial landscapes due to the heterogeneity in degree distribution (Fig. 2 A) maintaining high infected densities (Fig. 2 C) such that all patches in the landscape are always recolonised. However, the evolution

of greater virulence in riverine aquatic landscapes means that local parasite extinction is higher in low connectivity patches, and lower in high connectivity patches in riverine aquatic landscapes relative to terrestrial landscapes. Finally at high dispersal ($d = 1$; Fig 3 F), since virulence evolves to similar values in both landscape types, assuming similar virulence, one can predict accurately spatial distribution. This example shows how knowing evolutionary optima for parasite virulence can allow us to make general predictions about differences in spatial parasite distribution across landscape types. Thus overall, by accounting for evolutionary stable virulence, we show that we should find greater local parasite extinction in riverine aquatic landscapes(Fig. S14). Interestingly, since most of these extinctions are located in degree-1 patches (Fig 3 D–F), this decreases the likelihood that the parasite goes extinct globally, relative to terrestrial landscapes in which local extinctions are more homogeneous across connectivity. Other examples, namely parasite prevalence and host population densities (Fig. S15) show that differences in virulence evolution between the two landscape types do not change predictions relative to what is expected for a fixed virulence.

Thus, with these examples, we highlight the complex interplay between landscape structure, evolutionarily stable virulence and predicting epidemiological variables.

General discussion

In summary, we find that virulence evolves differently in terrestrial and riverine aquatic landscapes and that these evolutionary dynamics are driven by differing patterns in parasite relatedness. Parasite relatedness is the direct consequence of network topology impacting demography, hence the distribution of neutral variation. Ultimately, landscape types together with their specific evolutionarily stable virulence defines the distribution of hosts and parasites, and more broadly host-parasite metapopulation dynamics.

Our comparison of trait evolution across landscape structures allows us to highlight a mechanistic eco-evolutionary feedback loop (Fig. 4; Govaert et al. 2019; Fronhofer et al. 2023). Ecologically, landscape structure impacts dispersal patterns of individual hosts and their parasites, leading collectively to characteristic host-parasite spatial dynamics. These dynamics further define patterns of parasite relatedness in the landscape, which impacts the evolution of parasite virulence (eco-to-evo). Parasite virulence evolution in turn impacts the demographic rates of the hosts and parasites driving ecological patterns at the level of the patch (e.g., patterns of local parasite extinction), but also the distribution of hosts and parasites at the landscape scale (evo-to-eco).

At a coarse scale, this feedback loop allows us to derive the empirically testable prediction that parasite virulence as a function of host dispersal should differ generally between terrestrial and aquatic landscapes (Fig. 1). This prediction remains untested despite recent efforts to quantify impacts of parasites on host

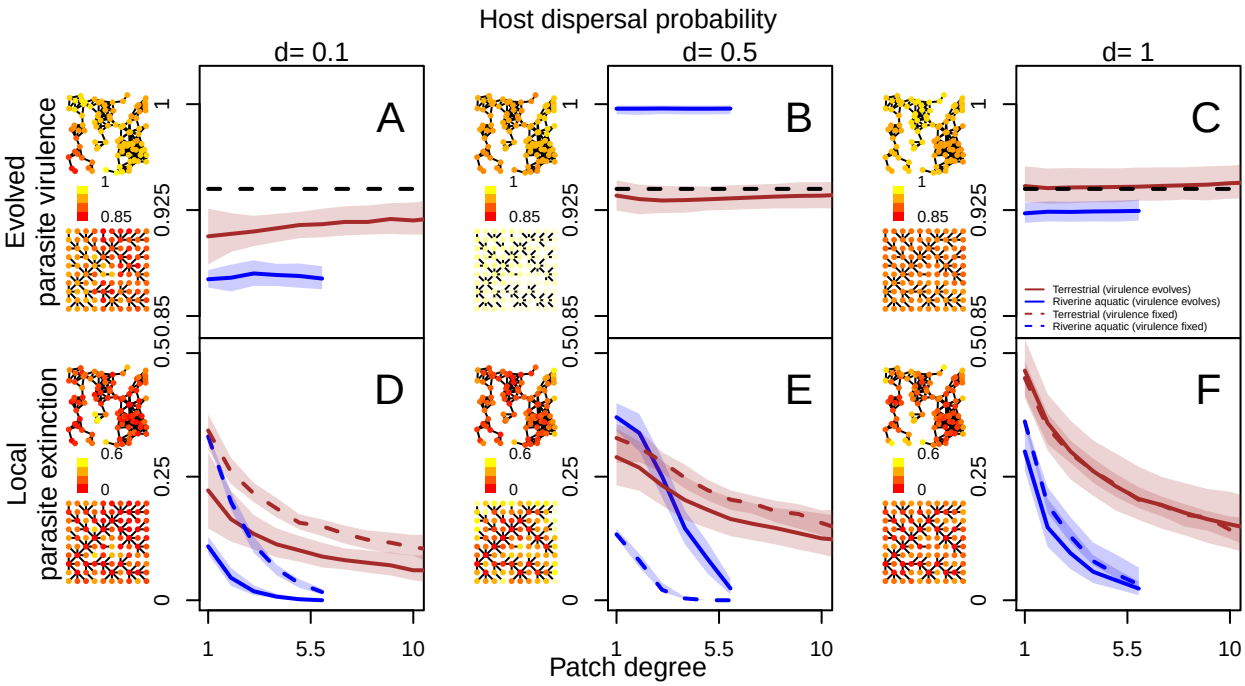

Figure 3: Spatial distribution of evolved parasite virulence (A–C) and local parasite extinction (D–F) as a function of patch degree for terrestrial (brown) and aquatic (blue) landscapes for different host dispersal probabilities ($d = 0.1, 0.5, 1$) in ecological (virulence $v = 0.94$, dashed line) and evolutionary (solid lines) simulations. From left to right, dispersal probability increases. A–C: Evolved parasite virulence in a patch, averaged over the last 100 time steps is is plotted as a function of patch degree. On the right side of each plot, the distribution of evolved virulence averaged over the last 100 time steps for one representative terrestrial and aquatic landscape is shown with more yellow colours indicating higher virulence. D–F: Local parasite extinction calculated as the average fraction of time in which parasite is absent from a patch is plotted as a function of patch degree. On the right side of each plot, the distribution of local parasite extinction over the last 100 time steps for one representative terrestrial and aquatic landscape is shown with more yellow colours indicating higher local parasite extinction. For all plots, medians are taken over for 100 and 1000 realisations for ecological and evolutionary simulations respectively. Model parameters of the focal scenario: intrinsic growth rate of the host $\lambda_0 = 4$, intraspecific competition coefficient of the host $\alpha = 0.01$, maximum parasite transmission $\beta_{max} = 8$, shape of trade-off curve $s = 0.5$, searching efficiency of parasite $a = 0.01$.

reproduction (Hasik and Siepielski, 2022) in a meta-analysis since data are mostly available on terrestrial systems. Of course, natural host-parasite systems likely also have other drivers of virulence than landscape structure. Additionally, riverine aquatic and terrestrial parasites could differ generally in their life cycles. While we recognise these limitations, our model shows that, all else being equal, the landscape context alone can modify eco-evolutionary outcomes. Beyond predicting how virulence evolution should differ between realistic landscapes, Fig. 4 allows us to put our work in the context of previous studies focusing on virulence evolution, landscape structure impacts on ecological dynamics and finally eco-evolutionary feedbacks on host-parasite dynamics.

Eco-to-evo: Strength of kin selection results from network topology

Focusing the eco-to-evo component of Fig. 4, we emphasise the role of kin selection for virulence evolution in spatially structured systems. It has previously been shown in infinite island models (Wild et al., 2009) and using pair approximations in lattice models (Lion and Boots, 2010) that virulence evolution in spatially structured systems should be driven by kin selection. In Wild et al. (2009), increasing parasite dispersal leads to greater virulence due to reduced competition with kin locally (kin shading). Thus, evolution of reduced virulence can be thought of as an altruistic strategy (Lion and Boots, 2010). While these studies assume virulence acting on host mortality which behaves differently from our assumption of fecundity virulence (Abbate et al., 2015), how spatial structure impacts both these kinds of virulence is similar (O'Keefe and Antonovics, 2002). Generally, the simplifying assumptions on spatial structure allow for an analytical expression for selection gradients to be obtained. Thus, the mechanisms driving evolution of parasite virulence going from well mixed to simplified systems are well worked out. While modelling more complex spatial structure does not allow for analytical treatment, we show that the strength of kin selection itself results from spatial network topology, which determine local parasite relatedness, further driving the evolution of parasite virulence.

While it is now recognised that terrestrial and aquatic landscapes have different properties (Rinaldo et al., 2014; Carraro et al., 2020; Gilarranz, 2020), we show that these properties lead to different evolutionary dynamics. Previous work has investigated the impact of modularity, which is a feature of terrestrial landscapes, and how it slows the spread of spatial perturbations (Gilarranz et al., 2017). Studies on riverine networks have focused theoretically and experimentally on metapopulation (Fronhofer and Altermatt, 2017) and metacommunity (Carrara et al., 2012) dynamics. Abstract representations of riverine landscapes have also been used in a more applied perspective to show differences in parasite spread upstream and downstream such landscapes (Carraro et al., 2018). While these studies focus only on one landscape type, Saade et al. (2023) have recently studied bistabilty and hysterisis comparing terrestrial and aquatic metapopulations. Going beyond this, we show that not only should ecological dynamics differ between terrestrial and riverine aquatic landscapes, but also patterns of selection and gene flow, leading to differences in trait evolution. Therefore, the patterns of kin selection reported here are only one example of how landscape structure impacts eco-evolutionary feedbacks.

Our result that it is the heterogeneity in connectivity of riverine networks that leads to characteristic patterns of parasite relatedness can be placed in context of previous work on patterns of neutral genetic and species diversity in spatial networks. Boussange and Pellissier (2022) study this effect of network structure on neutral genetic diversity and its consequences on evolution of local adaptation to an environment. They show that neutral genetic differentiation in spatial networks depends on both

connectivity and variation in population densities due to heterogeneous degree distributions. Similarly, it has been shown theoretically that dendritic networks maintain greater genetic diversity compared to grid landscapes (Paz-Vinas and Blanchet, 2015; Thomaz et al., 2016). Parallels can also be drawn to characteristic patterns of species diversity in spatial networks (Economo and Keitt, 2008) and particularly riverine networks (Muneepeerakul et al., 2007; Carrara et al., 2012). Finally, spatial network structure has also been shown to drive the evolution of dispersal (Henriques-Silva et al., 2015; Fronhofer and Altermatt, 2017).

Importantly, while going from a simplified spatial context (such as infinite island models), to more complex spatial structures, the basic mechanisms driving trait evolution do not change. However, landscape structure acts to modulates them (Fig. 4). In a host-parasite context we here study an illustrative example of virulence evolution, however, patterns of local adaptation as a result of co-evolution also critically depend on gene flow (Gandon and Michalakis, 2002) within networks (Gibert et al., 2013; Jousimo et al., 2014; Höckerstedt et al., 2022). Beyond host-parasite systems, linking our results to kin selection also implies that landscape structure should modulate the evolution of social behaviour, in general.

Evo-to-eco: Differences in virulence evolution may amplify, override or reduce differences between terrestrial and aquatic landscapes at different spatial scales

Finally, taking into account how virulence evolution differs between terrestrial and riverine aquatic landscapes, we can predict the spatial distribution of hosts and parasites. As discussed above, even without differences in virulence evolution, terrestrial and aquatic landscapes are generally expected to differ in their ecological properties (see for example, Saade et al. 2023). Fig. 3 re-emphasises this point, where the fixed virulence scenario shows differences in the spatial distribution of parasite prevalence and host density between terrestrial and aquatic landscapes. First, this shows that ecological variables as a function of patch degree can only be understood in the context of the entire landscape, since for the same connectivity they differ between two landscape types. Second, taking an evolutionary perspective allows us to understand how differences in trait evolution can increase, decrease or not impact ecological variables given similar connectivity in different landscapes. Thus, accurate predictions of disease distributions and spread may require information on landscape topology.

Our work demonstrates how virulence evolution can in turn impact host-parasite ecological dynamics at different spatial scales (evo-to-eco). Considering virulence evolution in terrestrial and aquatic landscape allows us to show how differences in the spatial distribution of hosts and parasites between such landscapes can be reduced (in the case of local parasite extinction, and intermediate dispersal), amplified (in the case

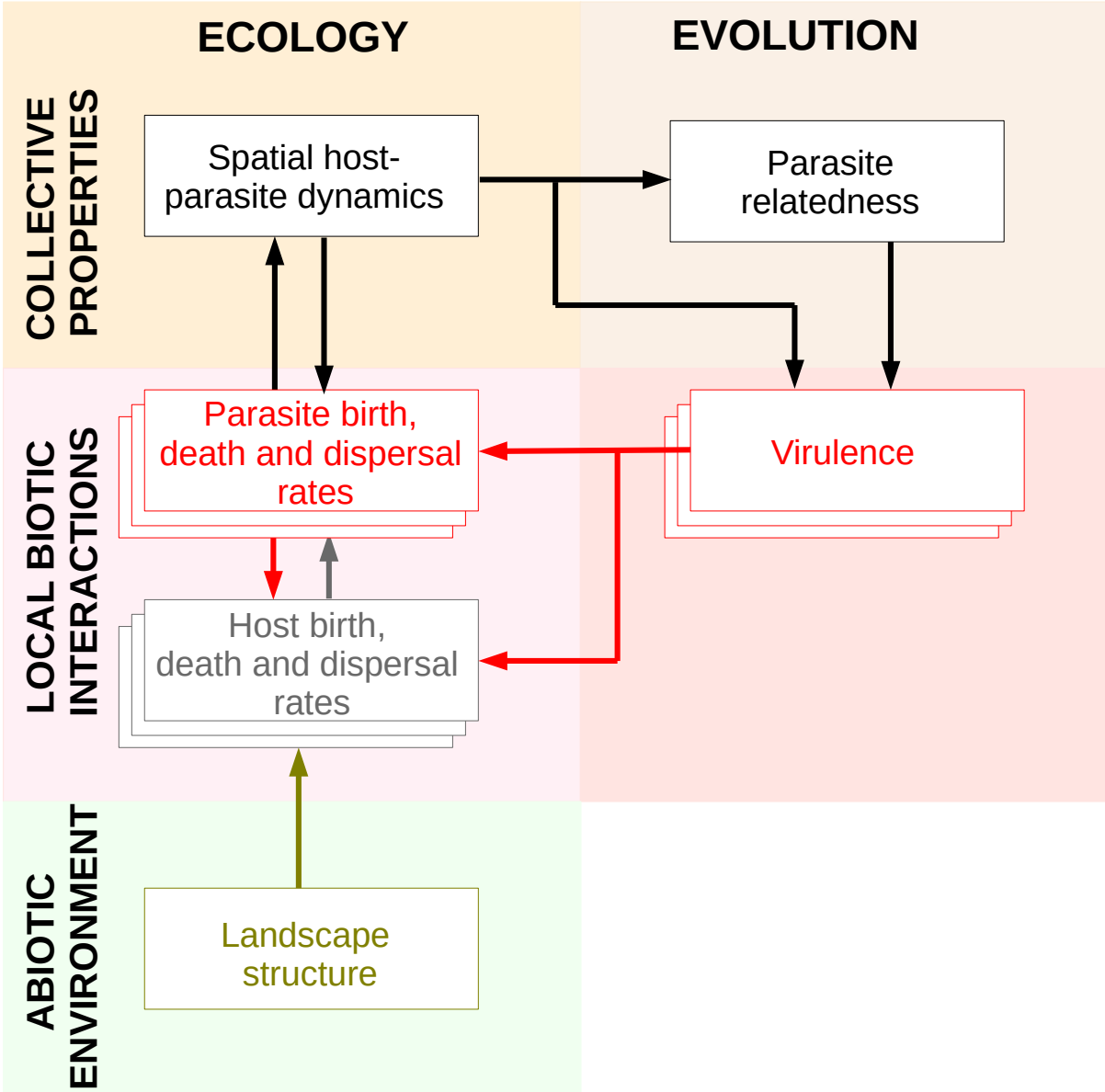

Figure 4: An eco-evolutionary feedback between host-parasite demography, landscape structure and virulence evolution. Clearly, landscape structure represents the abiotic environment, the biotic host-parasite interactions are shown by stacked boxes of hosts and parasites, ecological interactions and evolutionary dynamics represented by the virulence genotypes. Together, the abiotic environment, demography and evolution lead to collective properties at a higher level of organisation that can further feed back onto demographic rates. In our study, the landscape context is the abiotic environment that impacts host and parasite dispersal, since it impacts host (hence, parasite) dispersal patterns, and also their demography by modifying their spatial distribution. Virulence is genetically encoded, and it directly impacts parasite demography, via the virulence-transmission trade-off, and host demography by reducing the number of offspring the host produces (evo-to-eco). Finally, patterns of host-availability and parasite relatedness that result from host-parasite demography feed back onto parasite demographic rates, which, via selection and gene flow, impact trait evolution (eco-to-evo).

of local parasite extinction, and low dispersal) or stay the same (host population density and parasite prevalence for all dispersal rates), as a consequence of virulence evolution. This complex interaction

can be explained based on Fig. 4, since landscape structure directly impacts the spatial distribution of hosts and parasites. Because this in turn impacts how virulence evolves, the consequences of virulence evolution are indirect, and differences in landscape structure alone may dominate. At the level of the metapopulation, landscape structure clearly dominates over differences in evolved virulence for parasite persistence (global parasite extinction), with global extinctions being greater for terrestrial landscapes even though evolved virulence is lower. Thus, the interaction between landscape type and evolution must be considered when trying to predict spatial epidemiology. Recent work by Jousimo et al. (2014) and Höckerstedt et al. (2022) support this finding, since the authors show that increased host-gene flow in highly connectivity patches leads to increased resistance, reducing harm to the host, and lower parasite occupancy.

Perspectives

In conclusion, by studying how parasite virulence evolves in terrestrial and riverine aquatic landscapes, we highlight an eco-evolutionary feedback between landscape structure, host-parasite demography and parasite virulence (Fig. 4). It is now increasingly recognised that eco-evolutionary dynamics of host-parasite systems (Rogalski et al., 2017) are greatly modified by anthropogenic change leading to phenomena such as emerging infectious diseases (Daszak et al., 2000). Particularly, landscapes are vulnerable to changes such as habitat fragmentation and increased barriers to dispersal, generally, rewiring of dispersal networks (Bullock et al., 2018). Such disturbances maybe intrinsically (due to landscape structure) or extrinsically (due to the nature of human activities), differ between terrestrial and riverine aquatic systems (Johnson and Paull, 2011), and thus might require differing management effort. Our work highlights that an eco-evolutionary perspective may be needed for a complete understanding and an efficient management of such systems.

Methods

The model

We developed an individual-based susceptible-infected (SI) metapopulation model in which parasite virulence is genetically encoded and can evolve. We explore virulence evolution in terrestrial (represented by random-geometric graphs; RGGs Fig. 1 A) and in riverine aquatic landscapes (represented by optimal-channel networks; OCNs Fig. 1 B). A summarised model description is given below and details are found in the SI Appendix.

Life cycle

We model a population of asexual haploid hosts that may be infected by asexual haploid parasites or remain susceptible. Host generations are discrete and non-overlapping. After individuals are born, they disperse with probability d, to one of the patches connected to the natal patch with equal probability. Thereafter, individuals can reproduce. Local population growth follows Beverton-Holt dynamics (Eq. S1; Beverton and Holt 1957). Virulence (v) acts on the fecundity of the host (Jaenike, 1996; O'Keefe and Antonovics, 2002; Abbate et al., 2015), so infected individuals produce $1-v$ times the number of offspring. After reproduction, the parental generation dies and is replaced by the offspring generation. Parasite spores are then released from the dead infected individuals, which then find susceptible hosts according to a modified Nicholson-Bailey model (Eq. S2) with a type II functional response (Chaianunporn and Hovestadt, 2012; Deshpande et al., 2021). The transmission rate ($\beta(v)$) of parasites is assumed to increase with virulence (Eq. S3; Alizon et al. 2009). Multiple parasite strains can find a host, but eventually only one can infect it. Virulence undergoes mutation with a probability m_v and mutation effects on logit transformed virulence are drawn from a normal distribution with standard deviation σ_v. Parasites also inherit a neutral locus which is used to track relatedness between parasite strains.

Landscapes

We model terrestrial and aquatic landscapes using random-geometric graphs (RGGs) and optimal channel networks (OCNs), respectively. For each landscape type we generate 1000 realisations of $n = 100$ patches that are encoded in adjacency matrices. RGGs are generated using R package igraph (Csardi and Nepusz, 2006), version 3.6.3 by drawing n points from a $U[0,1] \times [0,1]$ distribution, and points that are within a radius r are neighbours (radius parameters of $r = 0.15$; average degree 6.7 after discarding networks that are not connected Saade et al. 2023). OCNs are generated on un-aggregated 10×10 grid (Carraro et al., 2020) with a fixed outlet position. The average degree of OCNs is 1.98.

Model analysis

Our main response variable is evolved parasite virulence which we measure at the the end of simulation (at $t = 2500$, see Fig. S1) where evolutionary dynamics have reached an equilibrium. We run simulations for all parameter combinations in Table S1, and the main text results are presented for one focal scenario that is described in all figure captions. We run additional ecological controls in which virulence is fixed ($v = 0.8, 0.82, ..., 1$) to show how relatedness and host availability should change when virulence does not evolve. We calculate parasite relatedness within a patch according to Eq. S4.

Author contributions

JND and EAF conceived the study. JND developed and analysed the models in collaboration with EAF. JND wrote the manuscript in collaboration with EAF and all authors commented on the draft.

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Data availability

Model code is available at via GitHub and Zenodo DOI (https://zenodo.org/doi/10.5281/zenodo.4043667).

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