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Host functional traits as the nexus for multilevel infection patterns

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Understanding pathogen transmission and infection patterns at multiple biological scales is a central issue in disease ecology and evolution. Here, we suggest that functional traits of host species readily affect infection patterns of species, communities, and landscapes, and thus serve as a linkage for multilevel studies of infectious disease.

Infection patterns at multiple biological scales

Infectious diseases pose a great challenge to public health, economies, and conservation [1]. Understanding the spatiotemporal patterns and driving factors of pathogen transmission and infection outcomes is central to tackling these challenges [2]. Such patterns and drivers may vary among different biological scales, where various ecological processes can shape host–pathogen interactions [2]. However, few studies have compared infection patterns and drivers across multiple scales. We propose that host **functional traits** (see Glossary) may serve as a key link for elucidating infection patterns across multiple organizational scales (from species to communities to landscapes), as such traits can ultimately affect pathogen transmission within and among these scales.

Functional traits as predictors for host exposure and competence at the species level

Pathogen transmission from a donor to a recipient host involves a series of ecological processes (Figure 1), including interindividual contact, pathogen invasion and establishment of infection, pathogen development within the host, pathogen excretion from the host, and then pathogen survival until recipient host **exposure** (e.g., direct contact, environmental uptake, and vector contact) [3,4]. These processes could be determined, at least partly, by several epidemiological characteristics of hosts, including exposure, **susceptibility**, and **suitability** [4]; the latter two within-host characteristics make up **host competence** (i.e., a host's ability to acquire and transmit pathogens [3]).

For any specific pathogen, host exposure and competence vary substantially between species [3], and this variation can be attributed in part to host functional traits [5,6]. Host exposure to any specific pathogen can vary across species due to interspecific differences in hosts' foraging ecology (e.g., diet guild and foraging strata) and behavior (e.g., migration strategy, sociality, and territoriality) [5]. Additionally, the relationship between species' life-history traits and host competence has been documented across a variety of host–pathogen systems (see an example in [2] and reviews [4,6]). This relationship may stem from life-history theory, which predicts greater competence of fast-lived species as they usually invest less in immune defense than growth [7]. However, greater evidence is needed to link established life history–immunity relationships to those between life history and competence [8].

Functional structure affects community-level infection risk

Considering the potential associations of functional traits with both host exposure and competence, it would not be difficult to conclude that the functional structure of a host community can influence infection patterns at the community scale. The effect of a specific functional trait on host exposure or competence can readily be scaled up to a correlation between **community-level infection risk**, which we define as all newly infected hosts in a community (Box 1), and the **community-weighted mean (CWM)**. When the target trait is linearly correlated with host exposure or competence, CWM could serve as an ideal predictor for the strength of intraspecific transmission (Box 1).

Glossary

Community assembly: the process by which species dispersal and local extirpations shape the composition of species in a local community.
Community functional structure: the distribution (i.e., the value, range, and relative abundance) of the functional trait measured in a given community. It is also termed by some ecologists as community functional diversity.
Community-level infection risk: the strength of pathogen transmission within the community. It could be measured with different metrics (e.g., infection prevalence and infection abundance) and in different ways (e.g., for a focal species or all host species across the community). Herein, we define this measure of risk as the number of newly infected individuals in the community given a specific time (i.e., *dI/dt* in Box 1 in the main text).
Community-weighted mean (CWM): the mean trait values weighted by the relative abundance of the species in the different communities.
Density-dependent transmission: pathogen transmission in which the number of new infections per unit time is proportional to the product of the density of infected hosts and the density of susceptible hosts.
Environmental filtering: the process that an organism must tolerate under particular environmental conditions.
Exposure: the process of encountering a pathogen (or infected vector), or the rate at which this happens.
Frequency-dependent transmission: pathogen transmission in which the number of new infections per unit time is proportional to the product of the proportion (or frequency) of infected hosts and the density of susceptible hosts.
Functional divergence: the unevenness in the distribution of trait values in the volume of the functional space occupied by the species/individuals of a community.
Functional trait: a species' morphological, physiological, or phonological characteristic that influences the fitness of an individual via its effects on growth, reproduction, or survival.
Host competence: the host's ability to acquire and transmit pathogens to a recipient.
Suitability: the host's ability to allow the pathogen to reproduce within it.
Susceptibility: the probability of successful infection of a host, given the exposure to a specific pathogen.

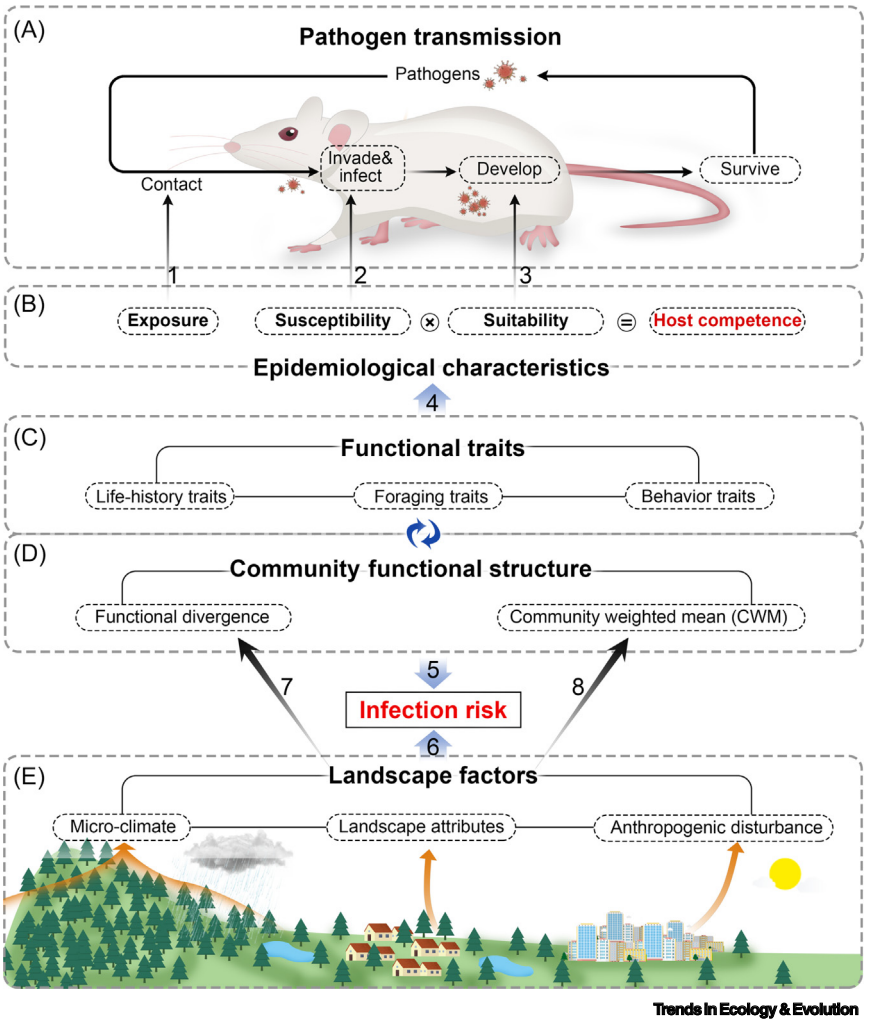

Figure 1. Functional traits as the nexus for pathogen transmission at different scales. (A) Pathogen transmission can involve several ecological processes, such as interindividual contact, pathogen invasion and establishment of infection, pathogen development within the host, pathogen excretion from the host, and then pathogen survival until a recipient host exposure. (B) Some of these processes could be affected by host epidemiological characteristics, such as exposure (1), susceptibility (2), and suitability (3). The latter two make up host competence. (C) Host functional traits can, at least partially, influence these species-level characteristics (4). (D) Community functional structure is defined as the distribution of the functional trait measured in a given community. Both of the two components of community functional structure, functional divergence, and community weighted mean (CWM), can affect community-level infection risk (5). (E) Landscape factors – such as landscape attributes, microclimate, and anthropogenic disturbance – can directly influence pathogen transmission processes (6), such as pathogen survival in the environment. Landscape factors can also indirectly affect infection risk through their effects on functional divergence (7) and CWM (8).

Aside from CWMs, **community functional structure** may also affect infection risk through its influence on interspecific transmission. Although interspecific transmission is usually weaker than intraspecific transmission because of the strong limiting effects of phylogenetic distance [9], interspecific transmission can sometimes considerably contribute to community-level infection risk, particularly for pathogens transmitted through the environment [10]. Theoretically, interspecific transmission could be largely determined by exposure alone (Box 1). Species with similar functional traits could have higher interspecific contact, as they are more likely to aggregate at similar habitats due to shared physiological or behavioral requirements [9]. Therefore, **functional divergence** metrics representing cross-species dissimilarity in habitat use, sociality, or foraging strata could be leveraged to quantify the likely relative contribution of interspecific contacts in driving cross-species transmission, even when the target traits are not correlated with host exposure or competence.

Community functional structure can mediate the effects of landscape factors

As functional traits affect a species' fitness in a given environment, community functional structure (including both CWMs and functional divergence) can be strongly associated with landscape factors, including but not limited to microclimate, landscape attributes, and anthropogenic disturbance [11]. Consequently, community functional structure could serve as a mediator for the indirect effects of landscape factors on infection risk at the community and landscape scales (Figure 1).

If a functional trait facilitates relatively higher fitness in a specific environment, its CWM should be higher. This would lead to a relatively higher or lower infection risk, assuming the trait is monotonically associated with host exposure or competence. For example, as fast-lived species have been predicted to have greater host competence for certain zoonotic pathogens, the CWMs of traits representing both fast life-histories and zoonotic risk could be higher in urban environments, since species with these traits can better adapt to and thrive in such habitats [8]. However, when the effect of functional traits on host exposure or competence varies across an environmental gradient, landscape factors can modify how CWMs relate to infection risk (i.e., an interaction effect). For example, along a 1101-meter elevation gradient, the CWM of pace-of-life became a stronger

Box 1. Intraspecific and interspecific transmissions contribute to community-level infection

Based on a multihost susceptible–infected model, the dynamics of newly infected hosts could be described as:

$$\frac{dI}{dt} = \overbrace{\sum\left(c_{ii} * b_{ii} * I_i * \frac{S_i}{N}\right)}^{\Delta I_{intra}} + \overbrace{\sum\left(c_{ij} * b_{ij} * I_j * \frac{S_i}{N}\right)}^{\Delta I_{inter}} \quad [I]$$

where i and j are different host species, c_{ii} and c_{ij} are the intraspecific and interspecific contact rates, b is the transmission rate (i.e., probability of infection given a contact). N is the density of all hosts in the community, and can be divided into susceptible (S) and infected (I) hosts. $c*b/N$ could be merged as β (i.e., transmission coefficient) in **density-dependent transmission** or as β/N in **frequency-dependent transmission** [10].

In Equation I, ΔI_{intra} represents newly infected hosts generated by intraspecific transmission, while ΔI_{inte} represents the contribution of interspecific transmission.

Given a fully susceptible community, ΔI_{intra} should be the abundance-weighted mean of $c_{ii} * b_{ii}$:

$$\Delta I_{intra} = \sum_i c_{ii} * b_{ii} * \frac{N_i}{N} \quad [II]$$

Here, b_{ii} and c_{ii} could respectively be considered as host competence and host exposure. Considering its definition, the CWM of a trait could be an ideal predictor for ΔI_{intra}, when the trait is linearly correlated with host competence or exposure.

As c_{ij} could be correlated with the similarity of involved species in their habitat use and/or foraging traits, interspecific transmission (ΔI_{inter}), largely determined by c_{ij}, could be further affected by the functional divergence metrics based on these traits.

predictor of plant foliar disease severity in southeastern Switzerland as temperature increased [12].

In addition to altering CWMs, landscape factors can also determine functional divergence via the dominant processes (e.g., **environmental filtering** versus competition) in **community assembly** [11]. When the employed functional divergence metrics are also related to interspecific contact rates, this coupling can lead to an indirect relationship between landscape factors and cross-species transmission risk (Figure 1).

Current gaps and future directions

We propose that functional traits of host species can link pathogen transmission across multiple scales, although considerable knowledge gaps remain. Foremost, we here consider host traits, representing only one side of any host–pathogen interaction. Functional traits of pathogens can also shape pathogen transmission and dispersal [13], such that future studies should also explore their role in determining infection patterns across scales. While many comparative studies have explored relationships between functional traits and host competence or its component parts (i.e., susceptibility or suitability) at the species level [3,14], future studies should also investigate these relationships at the within-species level (i.e., intraspecific trait variability). This will generate more realistic insights and predictions about pathogen transmission across scales.

Bringing the ingredient of functional traits into community-level studies is also promising for addressing some essential questions such as the 'diversity–disease relationship' [1,3]. Extensive studies have focused on species diversity or phylogenetic diversity, whereas the role of community functional diversity has been rarely considered, particularly in empirical studies. We thus invite systematic studies on the effect of functional diversity on community-level infection risk, as well as on the relative importance of functional diversity versus species and phylogenetic diversity. Such analyses will only become more and more tractable due to increasingly available standardized databases of species' functional traits across a wide range of host taxa [15].

Particularly poorly studied are effects of community functional structure on infection risk at the landscape scale. A clear merit of incorporating this approach at the landscape scale will be to better understand how ongoing habitat changes and biodiversity declines may impact disease risk in the Anthropocene, as well as separating the indirect and interaction effects of landscape factors from their direct effects (e.g., on pathogen survival) (Figure 1). Addressing these key questions will improve our understanding and predictive ability of infection patterns in this rapidly changing world.

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Declaration of interests

No interests are declared.

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