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Opinion

Deconstructing host quality offers insight into disease ecology

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Disease risk varies among ecological communities because species differ in their host quality, that is, their contribution to parasite fitness. We propose a four-component framework of host quality that harmonizes terminology across plant and animal domains. Using this framework, we demonstrate how the host defense strategies of resistance and tolerance relate to distinct components of host quality. Easily extendable to multi-parasite systems, the framework also helps to identify new ways of examining the continuum between specialist and generalist parasites. Ultimately, breaking down and formalizing the components of host quality helps with synthesizing disease ecology across domains and unlocking relationships between biodiversity and disease risk.

Highlights

Synthesizing disease-ecology insights across host–parasite systems is essential for understanding how anthropogenic pressures, including those that threaten biodiversity and reshape host communities, alter the risk of disease for plants, animals, and humans.

Tackling major questions in disease ecology requires a conceptual framework for exploring the spectrum of host quality for diverse parasites in both plant and animal systems.

We propose a framework that deconstructs host quality into exposure, establishment, growth, and infectivity, to facilitate new approaches to methodological challenges, fundamental questions, and applied concerns related to biodiversity and human health.

The framework readily extends to multi-host × multi-parasite systems, enabling comparative insights across complex disease networks in animals and plants.

Variation in host quality as a key feature of parasite dynamics

Anthropogenic pressures are threatening global biodiversity and reshaping the composition of plant and animal communities across ecosystems, with profound consequences for disease risk in plants, animals, and humans [1–3]. Understanding the relationships and mediating mechanisms between altered community structure and the amplification or dampening of disease risk remains a central challenge in disease ecology [4–6]. Key to making progress in this realm is understanding how host species vary in their contribution to parasite fitness, a concept we define as variation in **host quality** (see Glossary) [5,7,8].

Research in disease ecology of plants and animals spans a wide range of host–parasite systems. Yet despite progress, integration across plant and animal domains has been hampered by the use of domain-specific terminologies shaped by disciplinary traditions. To accelerate the integrative, cross-domain research needed to better address pressing questions in disease ecology, a shared language is needed, particularly around the concept of host quality [5,9,10]. Moving beyond the binary categories of hosts and non-hosts – an overly simple distinction often used for pragmatic reasons (e.g., [11–15]) – will enable a richer understanding of the role of variation in host quality for parasite dynamics. To facilitate collaboration across plant and animal disease systems, we present a framework deconstructing host quality into four components: **exposure**, **establishment**, **growth**, and **infectivity**. We then examine how various proxies relate to the different components of host quality. Furthermore, we illustrate how synthesizing host quality components across systems can (i) improve understanding of different aspects of host defense, and (ii) advance ecological theory when evaluating multiple parasites in diverse communities of plants and animals, all with the aim of better understanding how disease risk is influenced by anthropogenic pressures and their impacts on biodiversity.

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An integrated framework for host quality

We define host quality as a property of a host species from the perspective of the parasite, leading to its successful transmission [5,16]. Our definition captures the host's contribution to parasite fitness through four components: exposure, establishment, growth, and infectivity (Figure 1). While it appears similar to the term **competence** [10,17,18], host quality is a broader concept that also encompasses the host-specific aspect of exposure to a parasite. Earlier characterizations of components of host quality (and competence) were presented mainly with animal systems in mind (e.g., [8,10]), but similar concepts exist in the plant literature, albeit with differences in terminology [19,20]. Here, we present a framework (Figure 1A) applicable to both domains.

Exposure: the probability of contact between a host and infectious parasite propagules

Variation in exposure can result from differences in contact rates among species due to habitat use (animals [21]; plants [11,22]), behavior (animals [23,24]), morphological characteristics (animals [25]; plants [26]), or some combination of these.

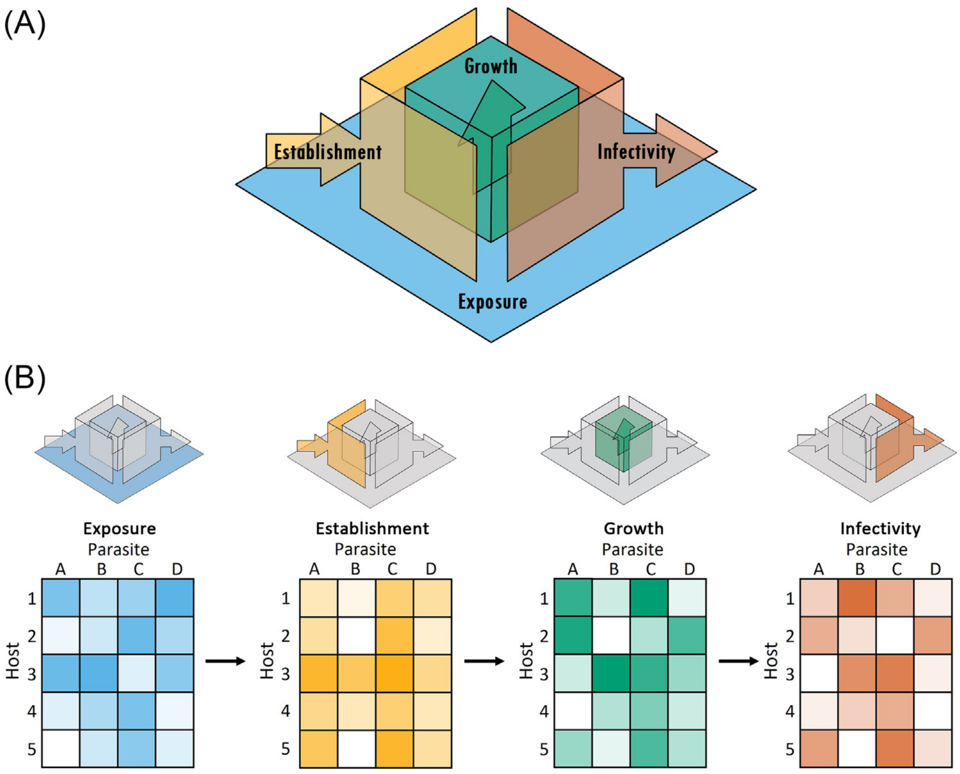

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Figure 1. Host quality in host × parasite systems. (A) Framework of host quality: traits of the host species that influence parasite fitness. The figure depicts host quality as a nested structure of four components that together determine variation in host quality. The size of the base area (blue) represents aspects of host quality related to exposure, such as territory size or the extent of the horizontal root system. The probability of establishment of the parasite within the host (yellow) depends on mechanisms of resistance. Development within the host corresponds to parasite growth (green), which leads to the release of infectious propagules corresponding to infectivity (red). (B) Conceptual visualization of variation in host quality in multi-host ×multi-parasite systems. Matrices indicate variation among host species (rows) and parasite species (columns) across the four components of host quality. Color intensity scales with the relative value of each component (white = 0, darkest = highest).

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Establishment: the probability that the parasite infects a host, given that contact has occurred

Variation in establishment can be driven by variation among species in immunological or physiological defenses (animals [27]; plants [28]), morphological traits (animals [29]; plants [30]), population structure (animals [23,31]; plants [11]), or some combination of these. In field studies, exposure and establishment can be difficult to disentangle and are therefore sometimes combined as susceptibility [32–34] (Table 1).

Growth: parasite growth and persistence in an infected host

Variation among species in the growth of parasites involves host traits that influence the development of individual parasites and the expansion and persistence of parasite populations within infected hosts (i.e., post-establishment; animals: [35]; plants [36,37]). In animal systems, this component is sometimes split into two elements: parasite development and pre-transmission survival of the host [8,10,38]. The duration of establishment and growth together comprise the **incubation period** [39].

Infectivity: the release of infectious propagules from an infected host that are accessible to susceptible hosts

Variation among species in infectivity can depend on variation in morphology, physiology, behavior, contact structure, or some combination of these (animals [40,41]; plants [11,18,19]).

Understanding components of host quality informs defense mechanisms

Host quality is central to critical questions in disease ecology. For example, to what extent is host quality inversely related to extinction risk and, thus, relationships between biodiversity loss and disease risk [17]? What intrinsic host traits (e.g., immune defenses, life history, behavior) drive variation in host quality? Is intraspecific host quality relatively constant, or does it vary with context? If it varies, how does the magnitude of intraspecific variation in host quality compare to interspecific variation [8]?

One research domain where an integrated framework of host quality might be particularly useful is in cross-system comparisons of host defense strategies (e.g., **resistance** versus **tolerance**). Host quality is ultimately constrained by the component that contributes least to parasite fitness, thereby acting as a bottleneck [4]. This is best illustrated by so-called 'dead-end hosts'. For example, in the case of West Nile Virus (*Orthoflavivirus nilense*), exposure, establishment, and growth may occur in humans and horses. Yet viral loads in the blood remain too low for mosquitoes to acquire the virus from these hosts, thereby preventing the release of infectious propagules and resulting in zero infectivity [42]. Host defense strategies dictate the contribution of each component to host quality, and strategies that limit even one component can therefore constrain parasite fitness. For instance, physical barriers (e.g., cuticle thickness) may limit exposure whereas innate defenses (e.g., antimicrobial peptides) may limit establishment [17,43]. A better understanding of how host defense strategies restrict the contribution of each component of host quality may help to explain why some species are more resistant or tolerant to certain parasites than others.

Resistance refers to a host's ability to prevent or reduce parasite colonization and is therefore closely associated with two key components of host quality: establishment and growth [18,44,45]. For example, an experimental study involving six tree species – one chestnut (*Castanea*) and five oaks (*Quercus*) – inoculated with the causative agent of chestnut ink disease, *Phytophthora cinnamomi*, found that the most resistant tree species were those in which parasite inoculation was often unsuccessful (i.e., poor establishment) and parasite load remained low (i.e., poor growth) [18]. However, tolerance of the host is concerned primarily with limiting the

Glossary

Competence: the ability of a host species to support an infection, given exposure.

Establishment: the probability that the parasite infects a host, given that contact has occurred. Second component of host quality framework.

Exposure: the probability of contact between a host and infectious parasite propagules. First component of host quality framework.

Growth: parasite growth and persistence in an infected host. Third component of host quality framework.

Host quality: a host's contribution to parasite fitness, including the four components of exposure, establishment, growth and infectivity. It differs from competence because host-specific variation in exposure is included.

Incubation period: the time interval between exposure and infectivity, that is, the duration of establishment and growth. In medical literature, it is the time interval between exposure and the onset of clinical symptoms.

Infectivity: the release of infectious propagules from an infected host that are accessible to susceptible hosts. Fourth component of host quality framework.

Prevalence: depending on the type of study, the percentage of individuals in a population that has been exposed to a parasite and/or in which the parasite has been established.

Resistance: a host's ability to reduce parasite burden by limiting the parasite's establishment and growth. Thus, resistance determines the degree to which a pathogen is able to establish and grow within a host.

Tolerance: a host's ability to limit the damage (negative health or fitness consequences) by a certain parasite, without necessarily reducing the parasite load itself.

Table 1. Examples of proxies for the four components of host quality across animal and plant host–parasite systems[a]

	Proxy	System	Study Laboratory/field	Exposure	Establishment	Growth	Infectivity	Refs
Parasite–host contact	Home range, body size, contact rate	Animal		×[b]	–[c]	–	–	[25,78,79]
	Hairiness, leaf size, leaf angle, root system extent	Plant		×	–	–	–	[26]
Infection response	Clinical symptoms of disease	Animal	Lab	–	×	×	×	[80]
			Field	×	×	×	×	[81]
		Plant	Lab	–	×	×	×	[82]
			Field	×	×	×	×	[83–90]
	Antibody titer – individual animal observations	Animal	Lab	–	×	×	–	[91]
			Field	×	×	×	–	[92,93]
	Seroprevalence – population-based observations	Animal	Field	×	×	–	–	[92,94–96]
	Innate immune indices	Animal	Lab	–	×	×	–	[27,97]
			Field	×	×	×	–	[27,98]
	Proportion of leaves infected	Plant	Lab	–	×	–	–	[99]
			Field	×	×	–	–	[84]
	Lesion growth of infection	Plant	Lab	–	–	×	–	[36,37,87]
	Hypersensitive response (to prevent spread of infection)	Plant	Lab	–	–	×	–	[100]
	Mortality	Both	Field	×	×	×	×	[101–103]
			Lab	–	–	×	×	[7,11]
Parasite detection	Propagule release (e.g., sporulation, eggs in feces)	Both	Lab	–	–	–	×	[36,104–106]
	Parasite detected in/on host (i.e., incidence)	Animal	Lab	–	×	–	–	[105]
			Field	×	×	×	–	[92]
		Plant	Lab	–	×	–	–	[49,107]
			Field	–	×	–	×	[57,70,84,103,108–111]
	Parasite abundance in/on host (i.e., quantity)	Animal	Lab	–	×	×	×	[91]
			Field	×	×	×	×	[25,92,105,112]
		Plant	Lab	–	×	×	×	[100,113]
			Field	×	×	×	×	[114]
Transmission estimates	Secondary cases, new vectors infected	Animal	Lab & field	–	–	–	×	[80]
		Plant	Lab	–	–	–	×	[11]

[a] Each row lists a proxy trait or measurement used to assess components of host quality, the system (plant of animal), the research setting (laboratory versus field), and examples of references.

[b] A cross (x) indicates that the trait is used as a proxy for the respective host quality component.

[c] A dash (–) indicates that the trait is not associated to the host quality component.

impact of infection on host fitness and does not necessarily affect components of host quality [18]. Thus, the mechanisms underlying host tolerance may operate independently of, or in parallel with, those affecting host quality [43,46].

Nevertheless, growth and infectivity – two components of host quality – can still correlate with tolerance. For example, hosts exhibiting a high degree of tolerance might live in an infected state longer than other hosts (e.g., due to reduced parasite clearance or reduced parasite-related mortality). This longer infection period could effectively increase the number of parasite generations (growth) and provide more opportunities for transmission (infectivity) [47]. In the experiment with *P. cinnamomi*, the chestnut *Castanea sativa* had moderate tolerance, as measured by plant performance relative to parasite load, and the highest infectivity [18]. Alternatively, if transmission of a parasite (e.g., *Bacillus anthracis* [48]) depends on increased mortality or reduced mobility of the host, then tolerance could limit infectivity, reducing parasite fitness and host quality [41]. Understanding how individual components of host quality contribute to parasite success is thus important to be able to make cross-system comparisons of defense strategies, including resistance and tolerance.

Answering big questions in disease ecology requires consistent use of proxies for host quality

Host quality is key for cross-system synthesis in disease ecology, but directly quantifying host quality (via one or more of its four components) presents a challenge, which only few studies have overcome (e.g., [8,49,50]). For example, five amphibian species were exposed to realistic doses of four trematode parasites, with exposure and establishment determined at day 1.5, and growth and infectivity at day 20 [8]. In many systems, thoroughly quantifying host quality in this way, particularly in studies involving multiple hosts, multiple parasites, or both, is impractical or impossible, so researchers often rely on proxies to capture variation in host quality (Table 1). While practical reasons justify the use of proxies, their interpretation can be challenging. For example, **prevalence** often conflates host competence with exposure [17]. These issues can be clarified by placing such measures within the broader context of host quality. Our framework provides a means to standardize terminology, highlight areas of ambiguity, and promote consistent use of proxies across systems and studies. Two examples illustrate our point.

The first example relates to the term prevalence, that is, cross-sectional screening of individuals in a population via serological or other techniques to determine the percentage diseased, infected, or, at least, exposed (Table 1). However, prevalence relates to components of host quality in ways specific to a study's setting [51]. For example, in field studies, parasite prevalence based on serological indicators (i.e., antibody titers) reflects a combination of exposure and establishment. By contrast, in laboratory studies involving experimental infection, all hosts are typically exposed, so that prevalence serves as a proxy only for establishment (Table 1).

The second example highlights ambiguity associated with proxies of infectivity. These proxies can vary from counting secondary infections in controlled environments [11,19,52] to measuring released infectious propagules (e.g., counting parasite eggs in feces [53], or spore production on leaves [54,55]). Quantification of parasite titers [56] or sequence reads (e.g., [57]) can be interpreted as proxies for growth, but both can also correlate with infectivity, making their interpretation less straightforward. With examples like these in mind, we challenge the field to carefully consider how laboratory-based approaches complement ecologically meaningful assessments of host–parasite interactions.

Although breaking down pathogen fitness into multiple traits is not unprecedented (e.g., [4,10,17], formalizing the components of host quality can strengthen efforts to generalize across systems. Measuring components of host quality using consistent, less ambiguous proxies, combined with direct quantification when feasible (sensu [8]) would support both conceptual [4] and mathematical modeling (e.g., efforts rooted in Susceptible-Infected-Recovered frameworks [58]). For example, the shedding rate of infectious propagules, which relates to what we term 'infectivity', is a parameter in equations describing the basic reproduction number of a pathogen in individual host species (R_0) and in host communities (community R_0 or R_0^C) [58].

Embracing the natural complexity of multi-parasite communities

While numerous studies fall into the category of multi-host × single parasite [1,59–61], far fewer can be described as multi-host × multi-parasite. Notable examples of the latter include the amphibian–trematode system [8] and the plant–soilborne fungal system [49], both of which employ integrative, experimental approaches. By definition, certain questions can be addressed only via a multi-host × multi-parasite system (Figure 1B). For example, are some host species generally more susceptible to a range of parasites than others [59]? Applying the host quality framework to multi-parasite systems allows questions about whether host quality components align (i.e., correlate positively) or trade-off (i.e., correlate negatively) across hosts species and parasites. Addressing such larger questions among studies utilizing diverse study systems (e.g., wild plants, greenhouse plants, wild animals, and captive animals) will advance the field of disease ecology.

Studies of plant–soil feedback offer valuable opportunities to explore multi-host × multi-parasite dynamics, despite not being originally designed to characterize host quality. Historically, the identity of soil biota, including parasites, often remained unknown due to limited molecular tools [62,63]. Advances in sequencing technologies now allow researchers to characterize a broad spectrum of plant-associated parasites (e.g., [57,64,65]). By coupling parasite abundance to the roots of co-occurring host species, plant–soil feedback studies can generate 'heatmaps' that reflect patterns of infectivity in multi-host × multi-parasite systems (Figure 1B), assuming that higher parasite abundance on roots scales with parasite success in the next generation. Such patterns can be interpreted as evidence that some species are better hosts than others. This approach has recently inspired similar steps in animal host–parasite systems [66,67].

Breaking down host quality into its four components within the plant–soil feedback approach, outlined earlier, remains a future challenge. The plant–soil feedback approach assumes exposure in proportion to relative abundance of host species [68], but that may not be applicable to all systems. In animal hosts, exposure may be a function of herding behavior [67]. In plants, exposure might be inferred from root system size or specific root traits such as root length [69]. Distinguishing establishment from growth will require temporal sampling designs to capture changes in pathogen abundance through time. Sampling pathogen abundance in complex communities repeatedly would provide the temporal resolution needed to disentangle these components, in both plant and animal systems. Developing such a timeseries approach across systems could promote coordination and ultimately lead to a synthetic understanding of how interspecific variation in host-quality components creates synergies and trade-offs that structure parasite communities and shape disease dynamics across changing communities.

Another key inspiration from plant–soil feedback research lies in its ability to couple measurements of parasite fitness (reflecting host quality) with impacts on host fitness [9,68] and impacts on host coexistence [68]. Such a dual focus allows us to capture the reciprocal interactions that constitute the feedback loop between hosts and their associated parasites. For example, Wang *et al*. [57] combined amplicon sequencing of soil-borne fungal communities with greenhouse bioassays measuring plant biomass across multiple host species. This integrated approach revealed that host community composition shaped parasite assemblages and, in turn, parasite communities influenced host performance. Pathogens suppressed productivity in single host communities, and dilution of these pathogens permitted increased productivity in multi-host communities [57,70]. Such integration of both directions of host–parasite feedback, also in animal systems, will be essential for understanding the multi-host × multi-parasite dynamics and coexistence in changing ecosystems [67].

The lens of host quality can help to clarify the concept of host specificity

The multi-host × multi-parasite context offers a powerful new perspective on the concept of host specificity. Patterns of apparent specificity may be more accurately revealed when multiple parasites interact with diverse host communities. Although true specialist parasites appear to be less common than once assumed [71–73], the multi-host × multi-parasite framework may help to clarify the conditions under which host specificity becomes functionally meaningful [74–76]. We illustrate this idea schematically as two ends of a gradient (Figure 2). In the classical view, each parasite infects only one or a few hosts with equal probability, and host specificity is treated as a fixed trait of the parasite. In contrast, when all parasites can infect all host species but differ in their success among hosts, emergent patterns of apparent specificity can still arise. For example, even if four parasites can infect five host species, one host (e.g., Host iii in Figure 2) may consistently provide higher parasite fitness. Thus, host-specific outcomes can emerge from variation in host quality rather than strict specialization. Host specificity patterns may thus also depend on the composition of host and parasite communities as well as environmental context. A key future challenge will be to determine how variation among host communities feeds back to shape the success of parasite communities, both ecologically and evolutionarily.

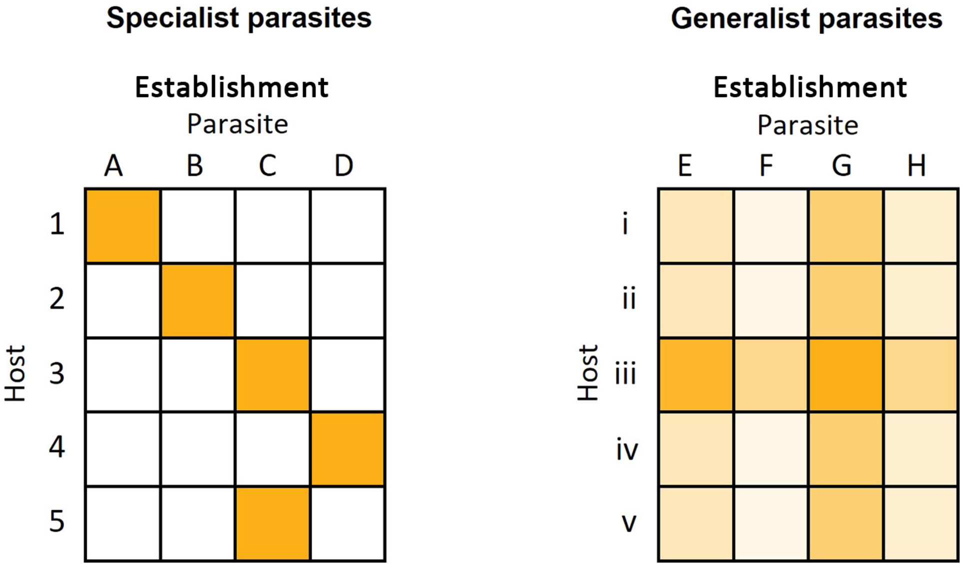

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Figure 2. Patterns of host specificity can be driven by parasite traits and by interactions among hosts and parasites. The matrices represent two ends of a gradient from specialist to generalist parasite strategies. In the specialist case (left), four specialist parasites (A–D) establish in only one (A, B, D) or a few (C) of the five host species (1–5) with uniform establishment success (indicated by equal color intensity). Here, host specificity is assumed to be an intrinsic trait of the parasite, largely independent of host community context. In the generalist case (right), all parasites are generalists capable of infecting all five host species (i–v) but vary in establishment success across hosts. Host species iii supports consistently higher establishment across all parasites (darker coloring), suggesting higher overall host quality. This scenario demonstrates how generalist parasites, interacting with a community of heterogeneous hosts, can produce emergent patterns of apparent specialization. In reality, both parasites and hosts vary along gradients of specificity. This illustration suggests no overlap between the identities of the specialist and generalist parasites (A, B, C, D versus E, F, G, H) and the host species (1–5 versus i–v), but in reality, the situation can certainly be more complicated. Furthermore, while this illustration focuses on establishment, the concept is equally applicable to other host quality components and host quality overall.

Concluding remarks

Here, we have proposed a framework of host quality that consists of four components – comprising exposure, establishment, growth, and infectivity – that is a biologically interpretable model for parasite fitness (Figure 1A). This tool should enable comparisons across taxa and foster conceptual crosstalk between the research fields of plants and animals (see Outstanding questions). A major challenge ahead is identifying robust proxies for these components, particularly in natural systems, to promote methodological convergence. Clarifying how plant and animal defense strategies map onto these components, whether via resistance, tolerance, or other mechanisms, could anchor host quality in measurable functional life history traits.

Taking multi-host × multi parasite interactions explicitly into account using this framework may be another leap forward (Figure 1B). For example, interactions with other (invasive) host species (e.g., competition, facilitation), coinfections with multiple parasites, and local environmental conditions such as pollution and climate may affect each of the four components of host quality, determining parasite dynamics [77]. In addition, the multi-host × multi-parasite perspective can help to explain patterns of host specificity as emergent properties of host and parasite community composition, rather than as intrinsic parasite traits alone.

Our host quality framework offers a scalable framework to synthesize how biodiversity loss, species turnover, and environmental change will affect host–parasite dynamics. Advancing the field of disease ecology will require interdisciplinary collaboration and methodological innovation to bring us closer to a unified and predictive theory of disease ecology.

Outstanding questions

What are robust proxies for the components of host quality that enable cross-system comparisons? Host quality and its components are difficult to measure directly, particularly in natural systems. Identifying reliable proxies for exposure, establishment, growth, and infectivity is essential for enabling comparisons across host–parasite systems.

How do host defense strategies map onto the components of host quality across taxa? Can exposure, establishment, growth, and infectivity be interpreted as functional indices of host defense, and thus host quality in both plant and animal systems? Understanding how defense traits like resistance or tolerance operate across these components can reveal general principles of host–parasite interactions.

To what extent is host quality dependent on ecological complexity? How much of the variation in species-specific host quality is shaped by the presence of other host species, diversity of the parasite community, or environmental conditions? Identifying when and why host quality is emergent, rather than intrinsic, is crucial for scaling predictions.

Can we better forecast how biodiversity loss, species turnover, or global change will alter parasite dynamics and disease risk across scales, by disaggregating host quality into biologically interpretable parts?

Author contributions

L.M. and K.D.M. conceived the idea for the work, organized the funding and the workshops. All authors contributed significantly to the workshops, the development of the conceptual framework, and the writing of the manuscript.

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Declaration of interests

The authors declare no competing interests.

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