Trends in Parasitology

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REVIEWS

Opinion

Scaling of Host Competence

Cynthia J. Downs,[1,*,@] Laura A. Schoenle,[2] Barbara A. Han,[3] Jon F. Harrison,[4] and Lynn B. Martin[5]

Body size influences many traits including those that affect host competence, the propensity to cause new infections. Here, we employ a new framework to reveal that, for at least two infections, West Nile virus and Lyme disease, large hosts should be more competent than small ones, but their lower abundance could mitigate their impacts on local risk. By contrast, for rabies, small hosts will be disproportionately more competent than large ones, an effect amplified by the higher densities of small species. These outcomes differ quite a bit from previous approaches that incorporate allometries into epidemiological models. Subsequently, we advocate for future integrative work to resolve how interspecific variation in body size influences the emergence and spread of infections.

Highlights

Competence, defined as the rate at which an individual is exposed to parasites and transmits a resultant infection to a new host or vector, is affected by body size because body size strongly affects behavioral and physiological responses to disease risk.We developed a framework for integrating scaling into models of competence for multihost parasite systems and highlight what data exist for populating such a model.

We apply this framework to West Nile virus, Lyme disease, and rabies to demonstrate its utility and reveal important gaps in knowledge.

We found that competence scaled hypermetrically for two infections, but also found that the hypometric relationship between host size and population density might mollify the effect of host body size on disease risk. The third infection we modeled exhibited hypermetric scaling that was amplified by including population density.

Using Body Size to Predict Complex Phenomena

When parasites have multiple host species for a particular life stage, those host species can vary substantially in **competence** (see Glossary), or propensity to cause new infections [1]. Understanding the drivers of competence is critical to predicting disease dynamics, and **scaling** research presents a potentially insightful directive for discovering broad patterns. Scaling patterns are typically described by power functions $Y_{[trait]} = aX^b$, where $Y_{[trait]}$ is the trait of interest, X is body size, b is the scaling exponent, and a is the y-intercept. A logarithmic transformation provides a linear form of these equations: $log(Y_{[trait]}) = log(a) + b*log(X)$. **Isometric scaling** occurs when larger animals are geometrically equivalent to smaller animals; for masses and rate processes $b = 1$, and for concentrations and densities $b = 0$. **Hypermetric scaling** occurs when b is greater than the isometric values, and **hypometric scaling** occurs with b is less than predicted for isometry. Multiple traits that could contribute to competence exhibit **allometric scaling** [2]. For example, many behaviors related to **exposure** and **transmissibility** [3], multiple physiological mediators of defense [4,5], parasite-carrying capacity [6], and symbiont species richness [7] all scale allometrically. Larger species also tend to have longer lifespans, which also impinges on various facets of competence [8,9].

Scaling relationships have been studied for years in disease ecology, typically by adjusting elements of SIR (susceptible-infected-recovered) models by body mass. Many of these models also incorporate concepts arising from the Metabolic Theory of Ecology, which emphasizes that physical constraints govern biological processes [2,4], including those affecting parasitism [10,11]. Importantly though, many studies invoking MTE lack empirically derived scaling relationships for host competence or they fail to scale important factors. Although host traits that are known to scale allometrically with body mass (e.g., demographic traits, disease-induced mortality) affect the basic reproductive number for a parasite (R_0) [3,12–14], many other facets of competence are, or could be, affected by body mass, but they have not yet been incorporated in modeling efforts. In order to understand fully how host size influences disease dynamics, we need a framework that accounts for the complexities of host–parasite interactions. Our goal here is to offer such a framework (Figure 1, Key Figure), which differs from existing frameworks because (i) it focuses on the organismal rather than population level traits,

[1]Department of Biology, Hamilton College, Clinton, NY 13425, USA
[2]Office of Undergraduate Biology, Cornell University, Ithaca, NY 14853, USA
[3]Cary Institute of Ecosystem Studies, Millbrook, NY 12545, USA
[4]School of Life Sciences, Arizona State University, Tempe, AZ 85287-4501, USA
[5]Global and Planetary Health, University of South Florida, Tampa, FL 33620, USA
[@]Twitter: @CynthiaJDowns

*Correspondence: cdowns@hamilton.edu (C.J. Downs).

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and (ii) it is based on observed allometries rather than first principles. The scant data yet available indicate that competence often scales allometrically and in a direction opposite to that which has been predicted from other approaches (Table 1 and Box 1).

Scaling Host Competence

Competence can be partitioned into four main components (Figure 1), each comprised of simpler traits [1,15]. For an individual to transmit a parasite successfully, it must first encounter a parasite and that parasite must successfully infect the host. Then, the parasite must use host resources but also successfully cope with host defenses such that it is able to reproduce and/or transmit [1,15]. Ultimately, transmission will occur through infection of another host or vector, or passage of parasite(s) into the environment. The scaling exponent for competence can thus be calculated by multiplying the scaling exponents for each stage of a host–parasite interaction (Figure 1B), revealing which body size classes present the greatest disease risk in a community (Figure 1C). Scaled estimates of competence can then be multiplied by the population density of various host species to discern the relative contribution of a given size class of hosts to disease risk in an area (Box 1). In the following section, we discuss how the four components of competence are known (Table 1) or expected to scale with body size. The scaling of competence probably varies among parasite–host systems, so in Box 1 we apply our framework to three very different parasites: a vectored virus (West Nile virus, WNV), a contact-transmitted (rabies) virus, and a vectored bacterium (*Borrelia burgdorferi*), the causative agent of Lyme disease.

Exposure to Parasites

Host behavior and life history govern contacts with infected individuals or vectors. Behavior underpins patterns of parasite exposure and is likely under selection to mitigate exposure risk [16,17]. Behaviors, such as territoriality and sociality, and life history traits, such as longevity and population density, often scale allometrically [3,18,19]. By integrating such scaling relationships into mathematical models, we can predict what kinds of communities are vulnerable to parasite invasion [3,20]. For example, host longevity, a key parameter driving host population dynamics, correlates positively with body mass in many homeotherms [21], and it is also inversely related to parasite species richness in hoofed mammals [22].

The consequences of scaling relationships will often depend on the mode of parasite transmission. For example, large-bodied host species with a correspondingly large geographic range may be exposed to and accumulate a greater diversity of soil-transmitted parasites. All else being equal, they might be disproportionately more competent than other species. Conversely, small territorial species might experience large risks of infection, and impose high risk on other resident conspecifics, if defended ranges are contaminated [23]. In contrast, for vectored infections, any allometric scaling for host home range size will likely be irrelevant. In those cases, exposure risk is more likely related to time spent inactive because vectors more easily bite hosts that are immobile (Box 1). Any allometries for locomotion would thus be expected to be important in vectored systems [3].

Host Susceptibility

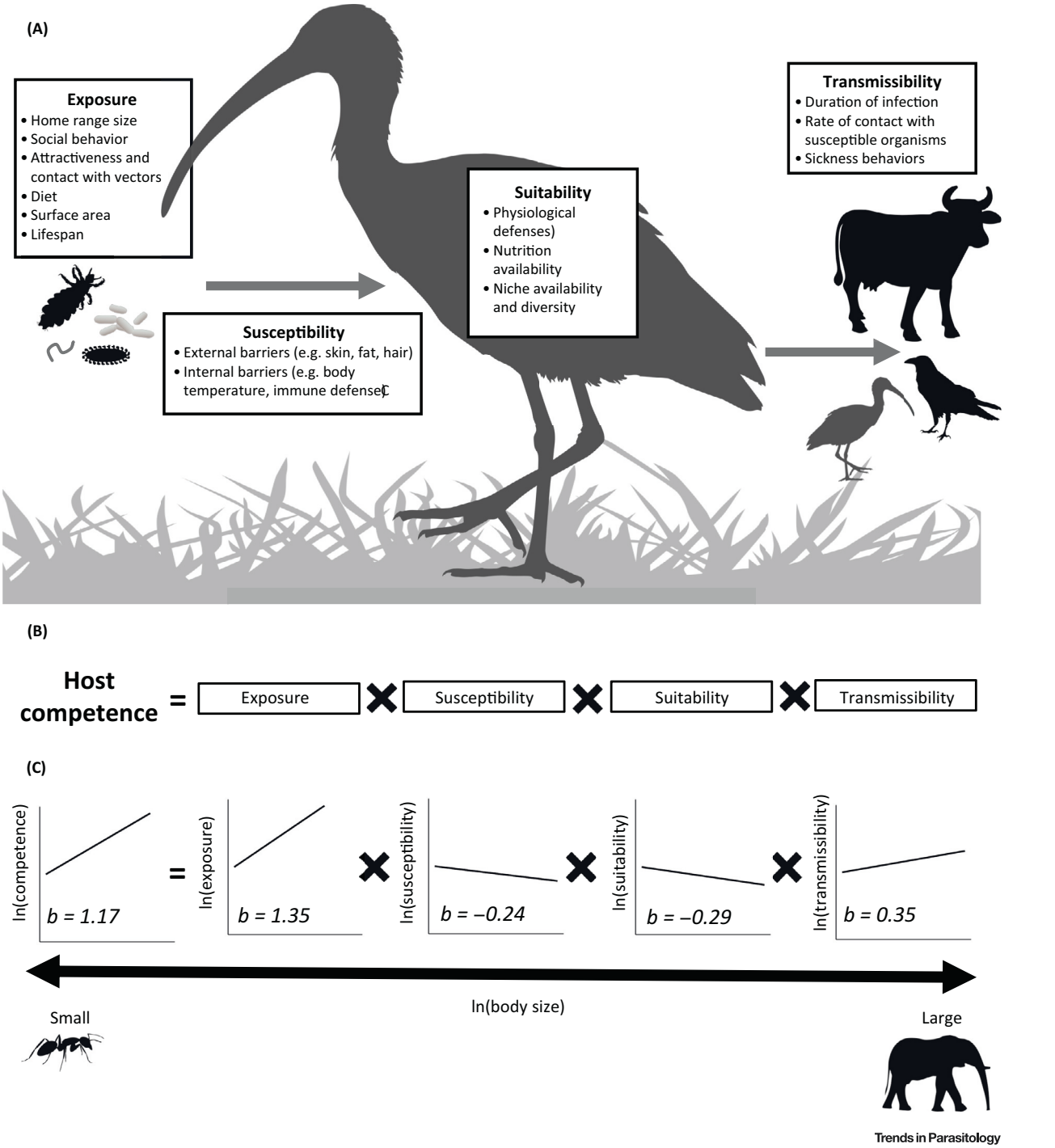
The likelihood that parasites successfully invade hosts depend on antiparasitic defenses and the extent to which hosts provide hospitable environments. Many traits that contribute to host **susceptibility** scale with body mass [9]. For example, the thickness and surface area of external barriers, such as skin and subcutaneous fat, scale allometrically. Skin mass scales hypometrically in birds and mammals (b = 0.84–0.94, [24–26]), but with a greater exponent than surface area (b = 0.67, [27]). Larger animals, thus, might be more difficult to infect by

Glossary

Allometric scaling: any departure from isometric scaling such that animals of different sizes are not geometrically identical.
Competence: the ability of a host to acquire and transmit parasites to another host or a vector either directly or through shedding into the environment.
Exposure: the likelihood that a host encounters a parasite through contact with other infected individuals, an infected vector, or free-living parasites in the environment.
Hypermetric scaling: an allometric scaling pattern in which larger animals have proportionally more of the focal trait.
Hypometric scaling: an allometric scaling pattern in which larger animals have proportionally less of the focal trait.
Isometric scaling: a scaling pattern in which large animals are geometrically identical to small organisms. Isometric scaling predicts that volumes, masses, rates, and proportions increase with mass^{1}, concentrations and densities scale with mass^{0}, areas scale with mass$^{2/3}$, and lengths scale with mass$^{1/3}$.
Resistance: the ability of a host to reduce parasite burden by killing parasites and/or limiting their reproduction.
Scaling: the study of the effect of body size and organismal characteristics among otherwise similar organisms. This concept is rooted in the concept of geometrical similarity, which states that two bodies are geometrically similar if they are in constant proportion.
Suitability: the likelihood that a parasite reproduces within its host to the minimum threshold number required for transmission.
Susceptibility: the likelihood of successful infection of a host, given that the host is exposed to a specific parasite.
Tolerance: the ability of a host to minimize the costs of infection for a given parasite burden by limiting or repairing damage.
Transmissibility: the propensity of a host to transfer parasites to another susceptible host or vector, including the sensitivity of said host to manipulative effects of parasites.

Key Figure

Scaling of Host Competence



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biting vectors or burrowing external parasite stages. Similarly, the ease with which animals can remove ectoparasites and defend against vectors is likely to vary with hair density and plumage weight [70]. Hair density is disproportionately lower in larger mammals [71], suggesting that protection conferred by hair decreases with increasing size. Similarly, plumage is disproportionately lighter in larger birds [26], suggesting that protection conferred by feathers decreases with size in birds. For some infections, behavioral defenses are important for susceptibility. For example, flapping behavior effectively reduces the odds that mosquito vectors will successfully bite; larger birds flap more slowly than expected for their body size (b = –0.24, [28]). Finally, from the perspective of the host as a new patch of habitat [29], large animals can store more resources on their bodies and they have more cell types (i.e., niches) for parasites to infect [27]. Subsequently, nutritional resources available to support both parasite growth and host immune defenses should be disproportionately higher in large animals. Larger hosts also harbor more gut microbial diversity than do small organisms [7], and as communities with higher diversity are harder to invade (e.g., [30]), hypermetric scaling of commensal microflora might protect large hosts.

Host Suitability

Suitability of hosts depends upon the interplay of parasite population growth and host defenses, including immune responses [31,32]. Host body size determines the carrying capacity of hosts for parasites. The space available for habitation scales isometrically such that $b = 1$ for volume-filling parasites and $b = {}^{2}/_{3}$ for surface-dwelling parasites [6]. Body size also influences energy metabolism and protein synthesis rates, but as both traits scale hypometrically, large hosts should exhibit comparatively slow immune responses, perhaps benefiting parasites [4,27]. On the other hand, the lower mass-specific metabolic rates of large hosts also mean that resources become available to parasites at slower rates [6]; this difference coupled with overall more resources for defense makes the exact scaling exponent of suitability hard to predict.

The immune system is arguably the most important mediator of host suitability, and yet we know fairly little about how it scales with body size [6]. A review of all described relationships is beyond the scope of this paper and may be premature anyway, as many measures are hard to interpret (Table 1). For instance, some immune cell concentrations, including neutrophils, scale hypermetrically [33], but what cell concentrations mean for protection is obscure. Functional immune traits, including bactericidal capacity of blood or blood components, appear invariant with host size (b = 0) among 24 species of bats [34], up to 70 species of birds [35,36], and six species of carnivores [37]. Still, we expect that more insight with regard to scaling of suitability will be revealed when functional defenses receive attention.

Indeed, the two main drivers of host suitability, **resistance** and **tolerance,** are functional traits. Resistance quantifies how hosts control parasite burden [31] whereas tolerance assesses how hosts minimize the costs of infection [38]. There have been a few efforts to describe scaling of

Figure 1. (A) Body size can influence the extent to which an organism is a competent host for parasites through its effects on the likelihood that the organism is exposed to parasites, the host is susceptible to the parasites, the parasites remain viable and/or amplify within or on the host, and the host successfully transmits the parasites. (B) Host competence can be quantitatively estimated as the product of all four traits. (C) Each of these traits is likely to scale allometrically with body size, and here we portray relationships between host body size and aspects of competence for West Nile Virus (see Box 1 for details). The product of the four scaling functions will be another function that captures how host competence scales with body size. In our example, competence is depicted as a rate (infections transmitted (host × day)$^{-1}$. (Image credits: Ibis and grass supercoloring Bob Comix Creative Commons Attribution-Share Alike 4.0 License. Microbes have no copyright. https://openclipart.org/detail/66655/bacteria. All other animal images are public domain.)

Table 1. Examples of Scaling of Components of Competence

Route of transmission	Infecting agent	Competence step(s)[a]	Measure of competence component(s)	Scaling pattern	Hosts	n[b]	Body mass range (g)	Refs
Vectored	*Borrelia burgdorferi*[c]	S2, T	Spirochete counts in infected nymphs[d]	Hypometric	Mammals	6	$10^{1.3}$–$10^{3.79}$	[65]
		S1, S2	Host suscepibility × host infectivity	Hypometric	Mammals	9	$10^{0.1}$–10^{1}	[66]
		S2, T	Ability to infect standard # of ticks	Hypometric	Mammals	9	$10^{0.1}$–10^{1}	[67]
		T	Prevalence in nymphal ticks[e]	Hypometric	Mammals	6	$10^{1.3}$–$10^{3.79}$	[65]
	West Nile virus	S1, S2	Susceptibility × mean daily infectiousness × mean duration of infectiousness	Hypometric	Birds	15	$10^{1.4}$–$10^{3.4}$	[66]
		S1, S2, T	The sum (over the viremic period) of the daily probabilities that a mosquito biting a bird will become infectious	Hypometric	Birds	24	$10^{1.3}$–$10^{3.4}$	[66]
		S1, S2, T	Susceptibility × mean daily infectiousness × mean duration of infectiousness	Isometric	Birds	25	$10^{1.3}$–$10^{3.6}$	[44]
		S2, T	Number of infectious mosquitoes that would be derived from feeding on these hosts	Hypometric	Birds	25	$10^{-1.75}$–$10^{0.5}$	[68]
		S2	Time from viral inoculation to death[i]	Hypometric	Birds	10	$10^{1.3}$–10^{4}	[41]
		S2	Viremia	Hypometric	Birds	25	$10^{-1.75}$–$10^{0.5}$	[68]
		S2	Neutralizing antibody production	Isometric	Birds	14	$10^{-1.75}$–$10^{0.5}$	[68]
	Anaplasma phagocytophylum	T	Ability to infect standard # of ticks	Hypometric (NS[f])	Mammals	9	$10^{0.1}$–10^{1}	[67]
	Babesia microti	T	Ability to infect standard # of ticks	Hypometric (NS)	Mammals	9	$10^{0.1}$–10^{1}	[67]
	Plasmodium brasilianum	E, T	Prevalence of infected hosts	Hypermetric	Primates	29	$10^{2.1}$–10^{4}	[69]
	Eastern Equine Encephalitis virus	S2, T	Proportion of mosquitoes that become infected after feeding during the period of infectious viremia times the number of days duration of infectious viremia	Hypometric	Birds	10	$10^{1.3}$–$10^{2.5}$	[66]
Direct	Mammalian microparasites	E, T	Contact mediated by social group size	Hypometric	Primates Ungulates[g]	166 92	~10^{-2}–10^{7}	[3]
	Pseudorabies virus	S2	Time from virus inoculation to death[h]	Hypometric	Mammals	13	$10^{1.3}$–$10^{5.7}$	[41]
	Rabies virus	S2	Time from virus inoculation to death[h]	Hypometric	Mammals	12	$10^{1.3}$–$10^{6.7}$	[41]

Table 1. (continued)

Route of transmission	Infecting agent	Competence step(s)[a]	Measure of competence component(s)	Scaling pattern	Hosts	*n*[b]	Body mass range (g)	Refs
Direct and environment	*Bacillus anthracis*	S2	Time from virus inoculation to death[h]	Hypometric	Mammals	8	$10^{1.3}$–$10^{5.7}$	[41]
Environment	Mammalian macroparasites	E, T	Contact mediated by intensity of home range use	Hypometric	Primates Carnivora Ungulates[g]	114 39 26	~10^{4}–10^{14}	[3]
	Escherichia coli	S1, R	Plasma-mediated killing (microbiocidal assay)	Isometric	Birds	12	10^{1}–10^{2}	[35]
		S1, R	Plasma-mediated killing (microbiocidal assay)	Isometric	Birds	70	$10^{0.8}$–$10^{2.3}$	[36]

[a]E = exposure; S1 = host susceptibility, S2 = host suitability; T = transmission. All stages that are encompassed by the measure based on the methods of the original paper or that the measure might apply to are included.
[b]Nn: number of species included in the analysis.
[c]Focused on Lyme disease caused by *Borrelia burgdorferi*.
[d]Counts are based on experimentally controlled host species fed to nymphs.
[e]Prevalence is based on experimentally controlled host species fed to nymphs.
[f]NS: trend; not significant at $\alpha = 0.05$.
[g]Perissodactyla and Artiodactyla.
[h]Considered a measure of rate of damage.

Box 1. Using Scaling to Predict Host Competence: Three Examples

In Table I, below, we list the results of applications of our scaling framework to three very different infections: a vectored virus (WNV, [56]), a contact-transmitted virus (rabies, [57]), and a vectored bacterium (*Borrelia burgdorferi*, the causative agent of Lyme disease, [58]). We first estimated scaling exponents for facet of competence, trying to rely on published examples from the literature (bolded terms). When a published scaling exponent was not available, we assumed isometry or estimated it from related literature. We then extrapolated how scaling of individual-level competence impinged on risk by accounting for scaling of population-level traits, namely density. Details of calculations are available in Tables S1–S3 in the supplemental information online. At the level of individuals, we found that larger species should be disproportionately more competent than smaller species for both vectored infections (Table I). However, when we accounted for allometric effects on host population density, small species were predicted to impose proportionally greater transmission risk than large hosts. In contrast, for rabies, small species were disproportionately more competent than large ones, and this effect was amplified by the inclusion of the allometry for host population density (Table I).

Our motivation is similar to other efforts seeking to incorporate scaling into disease ecology [3,12,59]. For instance, De Leo and Dobson scaled natural and disease-related mortality, maximum birth rate, and carrying capacity of host species to query how body size might affect density and frequency-dependent disease dynamics [12]. They found that B_{min}, the threshold value of the transmission exponent required for a sustainable infection, scaled hypermetrically for infections transmitted in a density-dependent manner ($b = 0.44$) but hypometrically for frequency-dependent infections ($b = -0.26$). Subsequent efforts, specific to rabies, integrated scaling for latency to become infected and the infectiousness period [13,14,41]. Interestingly, those approaches led to different scaling exponents with signs opposite to those of ours (e.g., B_{min} for rabies scaled at 0.45). Although our methods are too distinct for direct comparisons, it is possible that exclusion of some facets of host competence could misrepresent the influence of host body size on disease risk. On the other hand, we too lacked data for several aspects of competence, and assumed isometry for the sake of implementing our approach. We also have still not yet resolved exactly how to combine facets/exponents within each of the four stages so as to avoid artificially inflating the impact of any one stage on competence. For instance, sustainability at least is comprised of resistance, tolerance, and the propensity of a parasite to exploit host resources; how one should combine scaling of all three factors is nontrivial. As a consequence, we caution against overinterpretation of our examples, but we encourage bolstering of the data underlying our framework. Hopefully, via integration with classic epidemiological efforts and modern ideas associated with the Metabolic Theory of Ecology, we can eventually implicate accurately the body size classes of hosts that enhance and dilute infection risk.

Table I. Summary of Scaling Exponents for Each Component of Competence, Individual-level Competence, and Population-level Risk Proxy for West Nile Virus, Lyme disease, and Rabies

Exponent type	West Nile virus	Lyme disease	Rabies
Components of competence			
Exposure	1.35	1	**0.25**
Susceptibility	**−0.24**	**0**	0
Suitability	−0.29	0	−0.25
Transmission	0.35	0	**−0.75**
Individual competence (rate per day)	**1.17**	**1**	**−0.75**
Duration of infection	0.25	0.25	0.26
Lifetime individual competence	**1.45**	**1.25**	**−0.49**
Population density	−0.49	−0.54	−0.54
Population competence	**0.93**	**0.71**	**−1.03**

resistance, and the direction and magnitude appears to depend on the host–parasite system (Table 1). For tolerance, the effects of body size are as yet unknown, and predictions are difficult to make because large size and long lifespan often co-occur [8]. On one hand, proportional damage caused by a single infection will be lower for a larger individual than a smaller one; larger individuals have more cells to sacrifice in the interest of whole-host viability. In this case, tolerance should scale hypometrically because large hosts could lose substantially more tissue before suffering from infection. This type of tolerance could be termed passive tolerance

because it is simply a consequence of host morphology. On the other hand, large-bodied hosts are likely to encounter more parasites throughout their lifetimes, both because of higher exposure risk associated with traversing more risk space (through movement and feeding) and because they tend to live longer [9,39]. In this light, such *active* tolerance, induced only in the presence of infections, might scale hypermetrically, as large hosts seek to minimize the cumulative costs of resistance as they experience greater lifetime selection on their defenses. Presently, two studies support hypermetric scaling of tolerance. A recent meta-analysis found that long-lived but small organisms experience the greatest costs of immune responses [40]; the authors argued that this pattern might manifest because large, long-lived animals evolved robust tolerance mechanisms to mitigate repeated costs of resistance. A second study found that time to death, once infected, scaled hypermetrically for four different host–parasite combinations [41]. We advocate that scaling of tolerance receive particular attention in the future because hosts that are more tolerant might also be more capable of transmitting infections if their resistance and sickness behaviors are also modest [42,43].

Parasite Transmissibility

Several host traits could scale with body size to influence transmissibility. In many host–parasite systems, the likelihood of transmission will depend on surpassing some threshold of parasite burden and/or how host behavior is altered by infections. For these reasons, parasite burden or even duration of infectiousness may be poor proxies of competence (Box 1). The durations of West Nile virus infections are unrelated to body size among many species of passerines [44], and vectors must bite hosts when viremia in blood exceeds a particular level for transmission to occur [45]. These results might suggest that body size does not dipose greater risk among species; however, sickness behaviors can have strong effects on transmission, yet there is little known about whether they scale with body size [46,47]. Variation in sickness behaviors, including anhedonia (inability to feel pleasure), adipsia (absence of thirst), anorexia, and libido, is thought to maximize fitness of infected hosts [46,47]. Nevertheless, small-bodied species should be less able than large species to use anorexia to mitigate bacterial infections due to their high metabolic rates and low resource stores [48]. For similar reasons, adipsia should scale hypermetrically, as small-bodied species would likely be unable to endure low water intake given their relatively high water loss rates via respiration [25,49]. Because sickness behaviors include reductions in activity [46,47], they could also alter parasite dispersal by reducing the number of potential hosts or environments encountered while infected. Scaling of sickness behaviors clearly warrant future study.

From Individual to Population-level Competence

Parasites spread by interactions among hosts, vectors, and environments, so both population- and individual-level host variables will affect disease dynamics. Subsequently, local host density, population age structure, degree of sociality, social network architecture, and other population-level factors will be important and all scale allometrically too. In general, larger species exist at lower densities, which will tend to reduce the impact of individual-level competence on infection risk (Box 1). For WNV, competence scales hypermetrically ($b = 1.18$, Box 1), but when individual competence (infections host^{-1} day^{-1}) is multiplied by host population density (hosts km^{-2}), the greater risk imposed by larger hosts seems to disappear. Specifically, in passerines, because population density scales hypometrically ($b = -0.49$, [50]), per unit area WNV risk is more likely to scale as $b = 0.44$, much shallower that individual-level competence scaling suggests. Scaling of population density also varies with trophic level and the spatiotemporal distribution of resources [51], so population-level impacts on the scaling of competence will probably vary with habitat and the feeding strategies of hosts. Moreover, all the above scenarios estimate competences as steady-states. To

Box 2. Evolutionary Perspectives on the Scaling of Host Competence

Our approach implicitly minimizes the ongoing coevolutionary arms race among hosts and parasites, yet host–parasite interactions are the product of reciprocal evolution over generations during which adversaries face quite distinct challenges. Parasites are often many orders of magnitude smaller than their hosts, which leads to extreme differences in reproductive rates and generation times, and subsequently more opportunity for evolution for parasites. For example, the average generation time for *Escherichia coli* is 12 min [60], whereas the generation time for an African forest elephant (*Loxodonta cyclotis*) is 31 years [61]. Parasites also have higher absolute and per generation mutation rates than their hosts, and many even have access to nucleic acids from other parasite species (via horizontal gene transfer) [62]. Long-lived organisms may mitigate these inequities with extraordinarily specific immune responses via somatic recombination of B and T cells as well as fast-acting, broadly defensive mechanisms (e.g., reactive oxygen species) [63,64]. Altogether, it might seem that parasites would hold the evolutionary upper hand over the largest hosts. However, as host cellular replication and metabolic rates, population density, and other life history traits tend to scale hypometrically, transmission opportunities could be low enough from large hosts that parasites might be favored to infect the smallest host species available. Presently, it is premature to predict exactly which body size extreme is the best evolutionary refuge for hosts, if indeed there is a universal optimum given the disparities discussed in the main text and elsewhere [12,14,41].

Outstanding Questions

Are there generalities about the scaling of competence across disease systems or is each disease system unique? Is there any consistency if mode of transmission is considered?

How might we use competence allometries to predict and control disease emergence and spread?

How does variation in scaling relationships among aspects of competence (e.g., exposure risk and susceptibility, resistance and tolerance) ultimately affect overall competence? Due to coevolution between hosts and parasites, greater threats could reduce or even eliminate body mass effects.

When do behavioral defenses against infectious parasites scale allometrically?

At what level of biological organization are field data most appropriate to quantifying host competence (individual, population, community)?

In natural systems, do allometric effects on host population density eliminate scaling effects on competence?

discern how competence and population-level traits combine to drive infection risk, it will be important to consider seasonality in competence as well as other factors that change on the short time scales of some epidemics.

The scaling of group size and social network architecture will also be important in some systems [52]. In general, small species tend to live at higher densities than large hosts, but large hosts use their home ranges less extensively [19,53] and tend to have more complex social interactions than small ones [54]. Although data are scant [55], the relationship between group size and parasite risk seems to vary according to parasite transmission mode. For example, the larger social group sizes of large-bodied mammal species increase the risk of group detection by host-seeking vectors; however, the per capita exposure risk of being bitten is reduced in large groups. Conversely, larger group sizes should support higher transmission rates than small groups for directly transmitted parasites [17], but risk of transmission and exposure should be disproportionately greater for small species given that they also maintain high densities. In general, we require much more data to resolve how scaling among competence, group size, social network architectures and other population-level factors combine to affect local risk, especially because all are strongly shaped by host–parasite coevolution [8] (Box 2).

Concluding Remarks

We implemented a novel framework for investigating how body mass affects disease risk, finding that individual-level competence scales allometrically for three distinct infections: WNV, Lyme disease, and rabies (Box 1). If we are to apply this framework to disease modeling and management, however, we must first obtain more data on key facets of competence and integrate those with population-level factors also influenced by body size (see Outstanding Questions). For instance, our modeling exercise suggests that the lower density of large hosts might offset hypermetric scaling for competence for some infections. Also, although we have focused primarily upon microparasites here (because most data were available for microparasites), our framework should also be suitable for many macroparasites. Finally, we must emphasize that our host-centric expertise and space constraints prevented us from considering scaling in parasites themselves; the small sizes of most parasites expose them to distinct constraints and opportunities than their much larger hosts (Box 2). Although scaling studies have long been a cornerstone of biology, their relative absence in regard to host–parasite ecology and evolution should be rectified. Such an approach will entail challenges, some of which are described in Outstanding Questions, but such work could provide new insight and new opportunities to manage infectious diseases.

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Supplemental Information

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