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Null expectations for disease dynamics in shrinking habitat: dilution or amplification?

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As biodiversity declines with anthropogenic land-use change, it is increasingly important to understand how changing biodiversity affects infectious disease risk. The dilution effect hypothesis, which points to decreases in biodiversity as critical to an increase in infection risk, has received considerable attention due to the allure of a win–win scenario for conservation and human well-being. Yet some empirical data suggest that the dilution effect is not a generalizable phenomenon. We explore the response of pathogen transmission dynamics to changes in biodiversity that are driven by habitat loss using an allometrically scaled multi-host model. With this model, we show that declining habitat, and thus declining biodiversity, can lead to either increasing or decreasing infectious-disease risk, measured as endemic prevalence. Whether larger habitats, and thus greater biodiversity, lead to a decrease (dilution effect) or increase (amplification effect) in infection prevalence depends upon the pathogen transmission mode and how host competence scales with body size. Dilution effects were detected for most frequency-transmitted pathogens and amplification effects were detected for density-dependent pathogens. Amplification effects were also observed over a particular range of habitat loss in frequency-dependent pathogens when we assumed that host competence was greatest in large-bodied species. By contrast, only amplification effects were observed for density-dependent pathogens; host competency only affected the magnitude of the effect. These models can be used to guide future empirical studies of biodiversity–disease relationships across gradients of habitat loss. The type of transmission, the relationship between host competence and community assembly, the identity of hosts contributing to transmission, and how transmission scales with area are essential factors to consider when elucidating the mechanisms driving disease risk in shrinking habitat.

1. Introduction

Understanding how habitat loss affects pathogen prevalence and infection risk is a critical challenge in the face of unprecedented rates of anthropogenic landscape transformation [1]. It is clear that habitat loss leads to biodiversity loss [2–4], but it

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remains uncertain how declining habitat and diversity affect the transmission of infectious diseases [5]. A dominant hypothesis, known as the dilution effect, states that declining biodiversity leads to increased infectious-disease transmission [6,7]. The rationale is that greater host diversity provides a higher proportion of low competent hosts and therefore ‘dilutes’ the transmission chain. This creates a strong incentive to conserve intact ecological communities from the perspective of human health.

Despite the appeal of applying the dilution effect to guide win–win management strategies for conservation and public health, the empirical evidence has been inconsistent [8]. Some studies suggest that increasing host diversity decreases infectious-disease risk (a ‘dilution effect’) [7,9–13], while others suggest increasing diversity increases infectious-disease risk (an ‘amplification effect’) [10,14]. Large-scale surveys of West Nile virus and Lyme disease suggested that communities with higher diversity (measured as species richness) have a lower prevalence of disease [15,16]. By contrast, surveys of trophically transmitted (pathogens transmitted through food webs) and host-specific pathogens have suggested that intact ecosystems have higher pathogen prevalence than less-diverse systems. For example, losses of host populations due to fragmentation in Californian estuarine ecosystems led to a reduction in observed pathogen prevalence [17–20]. These conflicting patterns in empirical data have resulted in considerable academic debate about the generalizability of the dilution effect [10,21–25].

An increasing number of studies have investigated relationships between biodiversity and disease risk, and subsequent meta-analyses have attempted to identify consistent patterns. The majority of empirical studies use prevalence as a proxy for infection risk. By comparing communities with one host species to communities with at least two host species, Civitello and co-workers [26] found support for lower disease risk in multi-species communities compared with single-species populations across a variety of pathogen systems. By contrast, Salkeld and co-workers [24] found minimal support for the dilution effect among zoonotic pathogens. Salkeld et al. also highlighted a potential publication bias for studies supporting the dilution effect [24]—small studies that do not support the dilution effect have not been published. The allure of the dilution effect may be influencing the publication rates and overselling it as a generalizable phenomenon.

In parallel to empirical studies, models of disease transmission in multi-host parasite systems have been developed to understand when species diversity and community structure impact parasite transmission [27–30]. Theory suggests that the type of pathogen transmission determines the outcome of diversity–disease relationships. Multi-host pathogens with frequency-dependent transmission are expected to decrease in prevalence as biodiversity increases, i.e. the dilution effect [29,30]. Vector-borne pathogen transmission is typically frequency-dependent and dilution effects are predicted [29,30]; however, vector preference and variation in host competence can complicate the outcome of these diversity–disease relationships [31–33]. Pathogens that follow density-dependent transmission are expected to increase as host diversity increases [29]. While amplification effects are predicted for most density-dependent pathogens, when total abundance saturates well before species richness, both amplification and dilution effects can be observed [34].

Most theoretical studies of disease and diversity rely on measuring changes in community \( R_0 \). Community \( R_0 \) describes the number of secondary cases caused by the introduction of a single infectious individual into a completely naive community of multiple-host species. Community \( R_0 \) is a proxy for infection risk—it indicates whether or not an epidemic will occur (\( R_0 \geq 1 \)) and is linked to peak prevalence of an epidemic. In simple cases, \( R_0 \) can be used to infer endemic equilibrium, but numerical simulations are required to understand more complex multi-host pathogens [35]. Methods have been developed to use equilibrium prevalence of a disease to infer community \( R_0 \) (see [36]), but oftentimes there is a disconnect between model predictions and available empirical data.

While habitat loss is a major driver of declining biodiversity [37], this process has not yet been linked explicitly to models of disease. Landscapes undergoing habitat loss often have lower biodiversity, both in number of species and evenness of communities. Empirical studies of mammalian disease burdens often report higher parasite prevalence in fragmented landscapes [38–46]. By contrast, a positive relationship between parasite prevalence and patch size has been observed in several wild-bird populations [47–49]. Relationships between habitat patch size, host diversity and pathogen transmission within a habitat remain relatively unexplored (although see [5]). While most studies of parasites across a gradient of habitat loss do not directly measure biodiversity, they offer the opportunity to understand how real-world processes affecting community composition impact disease transmission.

Here, we develop null expectations for disease transmission in a single habitat patch undergoing loss of area and host diversity. We explore how a directly transmitted pathogen responds to changing habitat size in a multi-host community. Specifically, we ask: (i) How does habitat patch size affect the dynamics and prevalence of a generalist pathogen with density-dependent transmission? (ii) How does patch size affect a generalist pathogen with frequency-dependent transmission? (iii) How does the order of community disassembly affect these results? (iv) What assumptions about host competence are necessary to observe dilution versus amplification effects in an area undergoing habitat loss? and (v) How does changing assumptions of host range affect transmission of density-dependent pathogens?

2. Material and methods

(a) Definitions of biodiversity and disease risk

An array of terminology and measurements are used in the dilution and amplification effect literature to describe the relationships between host biodiversity and disease (reviewed in [10,25]). To keep the results of simulations consistent with empirical observations, we used species richness and equilibrium disease prevalence to measure biodiversity and disease, respectively. Unless indicated otherwise, we used the terminology and metrics interchangeably.

(b) Model assumptions and formulation

(i) Disease-free equilibrium

We examined community composition and resultant disease dynamics across a gradient of habitat size, from 0.1 km\(^2\) (10 hectares) to 100 km\(^2\) (10,000 hectares). We assumed there were up to 10 host species that contributed to pathogen transmission in these simulations. The average body mass \( m \) of individuals...
from each of the 10 host species was randomly selected from a skewed normal distribution fit to observed distributions of terrestrial mammalian body sizes [50,51] (electronic supplementary material, figure S1).

The carrying capacity \( K_i \) for each species \( i \) was determined by its body-mass–determined density \((16.2 m^{0.70} \text{ per km}^2)\) and a given habitat size \(a\). These assumptions gave us reasonable species accumulation curves at disease-free equilibrium: increasing habitat size increased the number of individuals in a given patch and the number of species, realistically mimicking patterns observed in natural systems (electronic supplementary material, figures S2–S4). We did not take into account interspecies interactions such as competition, as other authors have explored the impacts of these interactions on disease dynamics [52].

The community of hosts at a given habitat size was determined following one of three community assembly rules: (i) a species was resident in a patch if the carrying capacity was above a fixed threshold of 10 individuals, (ii) a species was considered resident if the carrying capacity was above the species-specific threshold, which was randomly selected from a uniform distribution (1–100), or (iii) a unique habitat density modifier \((e_i)\) was randomly selected from a uniform distribution (0,2) for each species \(i\), and thus density of each host species varies at each habitat area (figure 1, column 1). In the first two scenarios, species are perfectly nested—i.e. once they are extinct in a habitat patch, they never recolonize as the habitat continues to shrink. This constant density across habitat size is supported by empirical data [27]. The second community assembly rule is akin to some species being more tolerant to habitat loss (low population threshold) compared with other species that may be highly sensitive (high population threshold). The third assembly rule allows variation in species responses to habitat size—this can be due to variations in resource requirements, sensitivity to edge effects or another confounding factor.

(ii) Disease model and assumptions

The deterministic multi-host model was modified from an allometrically scaled S-I-R model for microparasites [53]. For each species \(i\), susceptible \((S_i)\), infected \((I_i)\) and recovered \((R_i)\) numbers of hosts were modelled (density-dependent transmission shown in equations (2.1)–(2.3)), frequency-dependent transmission in electronic supplementary material, equations S1–S3):

\[
\frac{dS_i}{dt} = b_i N_i \left(1 - \frac{N_i}{K_i} \right) - \frac{S_i(b_iI_i + \psi \sum_{j \neq i} B_{ij}I_j)}{a} - d_iS_i, \quad (2.1)
\]

\[
\frac{dI_i}{dt} = \frac{S_i(b_iI_i + \psi \sum_{j \neq i} B_{ij}I_j)}{a} - I_i (a_i + \gamma_i + d_i) \quad \text{and} \quad \frac{dR_i}{dt} = \gamma_i I_i - d_iR_i. \quad (2.2)
\]

In these models, we are assuming microparasites are transmitted through direct contact with conspecifics and heterospecifics. For density-dependent pathogens, transmission rates are dependent on the baseline force of infection and the density of infected and uninfected hosts \((S_i/N, \text{where } N \text{ is the total area of individuals in a patch})\). By contrast, frequency-dependent pathogen transmission is related to the proportion of infected individuals in the population \((S_i/N, \text{where } N \text{ is the total number of individuals in a patch})\). Conceptualizing transmission in this way focuses on two ends of what is probably a continuum [54], but it is a convenient way to understand the spectrum of transmission modes.

Demographic and disease parameters were determined by allometric scaling (following [53] detailed in electronic supplementary material, table S1). For baseline models, it was assumed that \(R_0\) within each species was constant (electronic supplementary material, equations S4 and S5)—an isolated, closed, non-breeding population of each species would experience similar disease epidemics and host competence did not vary between species. \(R_0\) is often thought of as a static parameter, but it is dependent on the population into which it is introduced. \(R_0\) for a single species is different than the community \(R_0\). For multi-host pathogens, the number of secondary cases depends not only on characteristics of a single-host species, but also of all species capable of transmission in the community. In the methods, we will use \(R_0\) to talk mainly about the species-specific parameter, but will specify when we are referring to \(R_0\) for the whole community.

Within-species transmission rates \((\beta_j)\) were calculated using species-specific recovery, mortality and disease-induced mortality rates (electronic supplementary material, table S1). We assumed that between-species transmission rates were the product of the between-species transmission rate \((\beta_{ij})\), which was the geometric mean of the donor \((i)\) and recipient \((j)\) within-species transmission rates \((\frac{\beta_i}{\beta_j} \beta_{ij})\), and a between-species contact rate, which was a proportion of within-species contact \((\psi)\). The between-species contact allowed us to vary interactions between species from zero (non-overlapping contact patterns; \(\psi = 0\)), to one (between-species contact was nearly equivalent to within-species contacts; \(\psi = 1\)). Simulations were run to equilibrium at a given patch size \((t = 150 \text{ years})\) and resultant prevalences in each species and across the community were calculated. In the above model set-up, we assumed that host competence, defined as within-species \(R_0\) (equations S4 and S5), was the same for each species.

(c) Variation in host competence

An alternative scenario is that host competence varies between species, and therefore within-species \(R_0\) is not constant across body size for a given pathogen. Some authors argue that life-history trade-offs dictate a negative relationship between \(R_0\) and body size [55], meaning smaller species are more competent hosts. Species with faster life histories, indicating smaller body sizes, have been shown to acquire and transmit infections better than slow-lived species [56,57]. This variation in competence could be related to lower investment in adaptive immunity in fast-living species [58,59]. We scaled \(\beta_i\) as a function of body size, ensuring that the smallest-bodied species’ \(R_0\) was the largest (electronic supplementary material, figure S5).

Conversely, if behavioural allometry is taken into account, \(R_0\) is expected to increase with body size [60]. This increase in \(R_0\) is mediated by increasing group size and social contacts in larger-bodied animals and is supported by prevalence data for ungulates and primates from the Global Mammalian Database [60]. In some simulations, we included conditions of behavioural allometry, scaling \(\beta_i\) so that the largest \(R_0\) was found in the largest host species (electronic supplementary material, figure S5).

Lastly, we examined the effect of variation in host competence by assembling a community that included incompetent hosts unrelated to body size (and thus unrelated to community assembly in this model). Incompetent hosts could get infected but could not transmit the pathogen onwards \((\beta_i = \beta_j = 0)\; \text{within–and between–incompetent-host transmission rates were set to zero}.

(d) Variation in evenly mixed populations

Finally, we considered a null model of community pathogen dynamics with allometric scaling of home ranges. In the baseline model, we considered infection risk of a density-dependent pathogen to scale with individuals per square kilometre. However, individual species may not come into contact with all individuals in a square kilometre. To account for heterogeneity in between-species contact, we used approximate home ranges as the area in which contacts occur. The minimum within-species transmission rate, \(\beta_i = 0.25/(md + (\gamma_i/d_i))m^{-0.69}\), is similar to density-dependent transmission calculated previously [53] but includes the assumption that home range scales with body mass.
In addition to a different transmission parameter, the system of ODEs was adapted so that the force of infection was dependent on the number of individual species infected in the home range of the recipient species, rather than the density of hosts in a square kilometre.

3. Results

(a) Null disease model results ($R_0$ constant across all species)

Assuming that host competence does not vary between species, we simulated how changing the size of a habitat patch, thus changing the diversity of hosts, would affect pathogen prevalence. For frequency-dependent transmitted pathogens, ($\beta SI/N$), additional species decreased prevalence because contacts were divided between all hosts (figure 1, column 2) and the addition of larger-bodied species meant that contacts were likely to be with individuals that had a lower force of infection in the community (electronic supplementary material, figure S5c). By contrast, for pathogens with density-dependent transmission ($\beta SI/A$), additional species increased prevalence within each species and across the entire population, irrespective of the new species' body size relative to the existing community (figure 1, column 3). The largest changes in equilibrium prevalence of a disease occurred when subsequent species were lost in small and intermediate habitat patches, mainly because these species changed the density and number of individuals in the habitat patch to a greater degree than large-bodied species. This was especially true when...
between-species transmission was very small (i.e. 10%) compared with within-species transmission (electronic supplementary material, figure S6). These outcomes assumed that $R_0$ and therefore host competence, did not depend on host body size. In these simulations, we assumed that the disease does not cause mortality ($\alpha = 0$).

Changing assumptions regarding the order of community disassembly did not affect the general relationship between diversity and disease—as long as species abundance and presence were nested. Although the general trend did not change for nested community assemblies, the rate at which prevalence within each species and in the overall community increased (DD) or decreased (FD) with habitat size and as host density increased (figure 1, row 2). When the densities were not constant across habitat size, the relationships between prevalence, species richness and habitat size were less predictable (figure 1, row 3). Although on average the relationships between diversity and disease were consistent among pathogen transmission types, there is more variation in prevalence (electronic supplementary material, figure S7).

(b) When host competence was dependent on body size ($R_0$ varies among species)

If $R_0$ within populations of smaller species was larger than $R_0$ within populations of larger-bodied species [55], then the difference in prevalence of a frequency-dependent pathogen between the smallest and largest habitat was even greater than observed for null simulations (electronic supplementary material, figure S8). This means when smaller hosts are more competent at transmitting frequency-dependent pathogens, the dilution effect should be easier to observe. Yet when the same assumptions are applied to density-dependent pathogens (figure 2a), there is still an amplification effect. Even though a higher proportion of competent hosts occupy smaller habitats, larger habitats have a higher host density, and thus drive higher pathogen prevalence, at least within the range of $R_0$ used in these scenarios.

Under the assumption that behavioural traits vary with body size and influence $R_0$ for a given pathogen [60], biodiversity can increase disease risk for frequency-dependent pathogens at larger more diverse patch sizes, despite a dilution effect for small to intermediate patches (figure 2b). The assembly of species and a significant change in $R_0$ by body size (electronic supplementary material, figure S5) can reverse the relationship between biodiversity and prevalence observed for null models.

When we assumed that host competence was unrelated to body size and dead-end hosts were randomly assigned to species, there were communities that produced non-monotonic changes in prevalence across habitat loss. For each community simulation, 50% of species in the global community were assigned dead-end host status. These dead-end hosts could become infected but were not infectious. When the smallest host was incompetent, the pathogen invaded only when competent hosts were present in habitat patches. Incompetent hosts always decreased the prevalence of disease when they were added to a community with frequency-dependent pathogens (figure 2c). For frequency-dependent pathogens, the distribution of body sizes of the incompetent species determined the degree of this decline, but decreases could be ‘rescued’ by the addition of competent host species (electronic supplementary material, figure S9).

(c) Heterogeneous mixing of populations

If we assumed host contacts scaled with home range size, then the direction of diversity–disease relationships remained unchanged. However, when new host species entered habitat patches, the within-species prevalence increased more than in the baseline assumptions (figure 3b versus figure 3a). The greater infection prevalence in these hosts was driven by a larger home range, and thus more potential infectious contacts over the lifetime of an individual. While individual species had greater infection prevalence, the community infection prevalence was lower than the baseline model (figure 3a). As body size increased, transmission rate declined for a density-dependent model including home ranges of species. Prevalence increased because the overall density of hosts was greater in species-rich patches, but the larger-bodied species were less efficient at

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**Figure 2.** Variation in host competence and underlying assumptions impact diversity–disease relationships. (a) Even if the smallest hosts are the most competent [61] and there are extreme differences in $R_0$ between body sizes (electronic supplementary material, figure S5), only the amplification effect is observed for density-dependent pathogens. (b) If behavioural allometry leads to an increase in $R_0$ across body size, this can lead to an amplification effect for frequency-dependent pathogens at larger patch sizes, but a dilution effect for small to intermediate patches. (c) When species that can become infected but are in turn not infectious (incompetent hosts, denoted by x) are randomly assembled along the distribution of body sizes, then dilution effects can be exacerbated for frequency-dependent pathogens, but this depends on the order of community introduction of these incompetent hosts.
transmitting pathogens and thus the increase in prevalence was less steep than in the baseline simulations.

4. Discussion

We simulated null expectations of changing disease prevalence in a host community in shrinking habitat. Using an allometric multi-host model, we showed that declining biodiversity can either amplify or dilute pathogen prevalence. The direction of the effect depended upon the transmission mode and how relative host competence scaled with body size. Amplification effects occurred when we increased host diversity for pathogens with density-dependent transmission and in some scenarios involving changes of host competence with frequency-dependent pathogens. Dilution effects were observed for simulations when host diversity increased for pathogens with frequency-dependent transmission. By contrast, dilution effects were never observed for density-dependent pathogens when host competence varied among species. Incorporating assumptions of home ranges and community composition did not affect diversity–disease relationships for a given pathogen transmission mode, but did change the magnitude of increases in prevalence observed over the community. This framework we have developed can be tailored to specific systems and host assemblages to predict disease prevalence in habitats of varying size. It can also be used to determine the assumptions necessary for observing specific diversity–disease relationships.

Allometric scaling provides an appropriate tool for understanding null expectations for diversity and pathogen transmission across a fragmenting landscape. Allometric disease models can recreate epidemic cycles observed in nature [62,63], and empirical evidence supports scaling of disease parameters by body size [64]. Although another study also suggested host

![Figure 3](https://example.com/figure3.png)

**Figure 3.** Impact of heterogeneous contacts on density-dependent pathogen transmission. Using identical disease parameters, endemic prevalence was observed for a host system that assumed contacts were determined by density of hosts (a) and compared with a system in which home range determined the average contacts of an individual from a given species (b). When home range is not taken into account, overall prevalence is higher, but when home range is considered, larger-bodied species that have larger home ranges have higher within-species infection prevalence.

Confirming results of other theoretical work, we found that transmission mode was consistently the best predictor of diversity–disease relationships. We recognize that these transmission processes are an over simplification of actual dynamics [54], but they provide an important benchmark and models with these frameworks can be used to explain empirical data. Density-dependent multi-host pathogens may best capture dynamics of foot-and-mouth disease ([66]), frequency-dependent transmission has better represented dynamics of Lyme disease (Borellia burgdorferi), sarcoptic mange and Mycoplasma ovipnuemonae ([15,67,68]). By contrast, work on other wildlife pathogens shows that empirical data
are best represented by models that incorporate transmission that lies in between frequency- and density-dependent transmission [69,70]). The framework presented here can be adapted to incorporate the complexities of transmission observed in natural systems.

The patterns of community assembly and disassembly are important for disentangling diversity–disease relationships. Previous work assembled communities from a global species pool [34,71,72], using Fisher’s Law and Preston’s Law for determining the number of vector and host species, respectively. In our models, we are considering a community within shrinking habitat with predictable community disassembly patterns. Although fragmentation can have complicated effects on local species abundance and persistence, overall habitat loss consistently excludes larger-bodied species from landscapes. Our model is consistent with empirical observations: larger habitats support a greater number of species than smaller habitats; larger habitats support increasing numbers of larger-bodied species [73,74], all at higher net density. Nestledness of host communities has been recorded in several systems, suggesting that the hosts found in the least-biodiverse communities are predictable [61,75].

A key research focus should be how to quantify host competence across body size and determine if predictable changes occur across multiple taxonomic groups [61]. If we are able to quantify how host competence varies across the habitat range, then we will improve predictions of how diseases respond to changes in habitat. Changes in host competence may be even more important than variations in host density [61], which are often highlighted as important factors driving disease dynamics. Additionally, we show that these differences in competence can lead to dilution effects and amplification effects, depending on the details of habitat loss and community assembly.

Host competence can be described as the product of the probability of getting infected (susceptibility), the duration of infectious period and the probability of infecting another host. Host competence varies in natural systems [76–78], and this variation has been highlighted as an important mechanism driving the dilution effect in natural systems [55,56,61,75,79,80]. Our simulations confirm the importance of host competence—although we did not explicitly include each component of host competence; we varied host competence by changing species-specific $R_0$, resulting in a change in transmission rate ($R_0$) but not infectious period (1/$\gamma$). By linking host competence to body size, we demonstrated that the amplification effect could be observed for frequency-dependent pathogens—reversing the effect observed when host competence is assumed to be constant across species. Fast-lived species (i.e. smaller body size) are often thought to have higher host competence than larger-bodied hosts. For host competence to affect transmission of density-dependent pathogens, additional incompetent hosts must also be competitive and suppress or indirectly compete with the competent hosts. Unless the density of competent hosts changes, the addition of species will not affect dynamics even if they are incompetent. Competition may explain why the dilution effect has been observed in density-dependent pathogens such as Sin Nombre virus [11,81]. Although the dilution effect was observed for frequency-dependent pathogens using null assumptions, decreasing host competence with larger body size increased the dilution effect observed. Therefore, incompetent hosts (also called incidental hosts), facilitated a dilution effect (i.e. [82]), but their inclusion was a sufficient, but not necessary, condition to explain the dilution effect for frequency-dependent pathogens.

Alternatively, it has also been proposed that $R_0$ is greater in larger-bodied species. This general trend is driven by increases in average group size as hosts get larger. If larger hosts are actually more competent, this exacerbates the amplification effect observed for null expectations of density-dependent pathogens. It can also flip the null observations of frequency-dependent pathogens, so that in extreme cases the amplification effect can be observed. Although we do not know of any empirical examples to support this, the lack of published results may be due to publication biases [24] rather than the absence of this phenomena in natural systems. Variation in host competence may arise from vector preference for particular hosts [83], rather than innate host biology [79], which in turn may be related to allometry. Understanding how host species contribute to infection dynamics [84] and how this changes with body size, and thus community assembly, should be a priority for future empirical studies.

Most of our simulations indicated a monotonic change in prevalence and diversity—either increasing (amplification) or decreasing (dilution) prevalence as patch size and diversity increased. Yet some field studies show a concave relationship between prevalence and diversity, so that the highest observed prevalence is found in intermediate fragments and the lowest prevalence is found in the smallest and largest habitat fragments [39,43]. These situations may arise when the community abundance peaks prior to biodiversity as habitats get larger—these conditions were simulated using similar sets of differential equations [35]. These patterns could also be observed in our scenarios when larger hosts are more competent (figure 2b), mirroring trait-based amplification effects observed for frequency-dependent pathogens by O’Regan and colleagues [52]. Competent hosts that enter intermediate habitat patches could bolster infection prevalence, and subsequent addition of incompetent hosts in larger habitats could decrease prevalence, but this is complicated by the size distribution of hosts and their relative densities. Host and pathogen life history could also play a role in individual parasite species’ responses to habitat fragmentation [45,46], particularly for pathogens with complex life cycles. These complexities underscore the importance of understanding how species assemble and how host competence changes with increases in diversity.

We did not include explicit models of vector populations, which are expected to undergo frequency-dependent pathogen transmission. Including non-competent hosts into vector-borne disease models reduces disease prevalence as biodiversity increases [25], yet observed dilution effects in populations that undergo frequency-dependent transmission do not necessarily require the addition of incompetent hosts, as it is the null expectation for this type of transmission. Temporal and spatial heterogeneity within a habitat patch [85], host distributions [86] and immunological responses to prior infection [87] will also impact transmission dynamics of vector-borne pathogens. Stage-specific transmission dynamics has also been shown to impact disease dynamics [88]. Incorporating vector populations into these null models and how these respond to changes in habitat area and host availability in these models will be an essential next step [89,90].

Although habitat loss often results in a reduction in overall diversity, effects on individual species abundance and
distribution often vary [91]. Patterns of infection in wildlife populations will be influenced by host ranging patterns, density, predation, intraspecific and interspecific contact rates and diet [92], and these characteristics can be affected by changes in habitat structure [93,94], edge effects [95] and synergistic effects of habitat loss and disturbances such as fire [96]. The presence of competitors and/or predators has been suggested as a mechanism for reducing infection prevalence in communities with high levels of biodiversity [25,97], and is supported by a model with varying interspecific contact rates [52]. These variations in species-specific responses to habitat loss will be important to consider for certain disease systems and will probably influence changes in prevalence depending on the host competence and sensitivity to habitat loss. Here, we highlight that host communities decline in a predictable manner, emphasizing a nested host species community that is dominated by smaller-bodied species in low-diversity, small habitats. While this produces the dilution effect for frequency-dependent pathogens and the amplification effect for density-dependent pathogens, changes in how host competence scales with body size can alter these expectations.

Our multi-host model focuses on a single habitat patch and describes infection dynamics that vary with patch size and community composition. In natural populations, habitat patches are rarely isolated to the extent that immigration and emigration are non-existent. Habitat patches exist in meta-populations with varying levels of connectivity and models emphasize that connectivity and host dispersal are important characteristics determining system dynamics [5,98–100]. Larger habitat patches may support higher incidences of density-dependent pathogens and serve as sources of pathogen pollution for small habitat fragments in the landscape. By contrast, if movement only occurs towards larger, higher-quality habitat, the average prevalence in large patches could decline if uninfected individuals emigrate, or increase if the density of susceptible hosts increases. The framework we present here could be extended to incorporate spatial structure so that the influence of allometric home ranges and dispersal on prevalence can be explored in each sub-population within a meta-population/meta-community context [60].

Critically, the temporal and spatial scale at which observations are made will affect the infectious-disease dynamics [27]. Moving forward, some of the biggest challenges will be to determine at which spatial and temporal scale empirical measurements would be useful for testing these null predictions. Disease prevalence should be measured in fragmented long-term ecological study sites, such as Stability of Altered Forest Ecosystems (S.A.F.E.) in Borneo [101] or Biological Dynamics of Forest Fragments Project (B.D.F.F.P.) in Brazil [102]. These systems have existing data on host demographics, population densities, community compositions and environmental changes, which would complement disease-dynamic investigations. Changes in pathogen prevalence can be observed with respect to transmission mode, pathogen characteristics and host competence in order to disentangle mechanisms driving transmission dynamics.

5. Conclusion

Using realistic allometric models of a multi-host pathogen, we showed that amplification and dilution effects can be observed in a shrinking habitat. The observed change in equilibrium prevalence with declining habitat and decreasing biodiversity depends on how communities disassemble, how competence scales with body size, the likelihood of a given species’ residence in a patch and the transmission mode. The infectious-disease–habitat loss model developed in this study provides a useful template for the design of longitudinal empirical studies of multi-host pathogens in shrinking habitat. It can be adapted to study-specific host species, transmission mode and habitat loss responses to direct sampling effort across a landscape.

Understanding how habitat loss, biodiversity and disease are related is an essential challenge in natural area management. In cases where habitat loss correlates with emerging diseases of humans [103–105], management of disease systems will benefit from evaluating whether biodiversity is the underlying mechanism of pathogen emergence, or if it is a combination of changing contact patterns, environmental conditions and complex species interactions driving emergence. While we often focus on human health, habitat loss and subsequent changes in disease incidence also affect disease risk in animal and plant populations [5,106].

Data accessibility. The data-gathered model parametrization was gathered from the primary literature. All data have been included as tables or figures in the electronic supplementary material.


Competing interests. We have no competing interests.

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