Opinion

The Coevolution Effect as a Driver of Spillover

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Global habitat fragmentation is associated with the emergence of infectious diseases of wildlife origins in human populations. Despite this well-accepted narrative, the underlying mechanisms driving this association remain unclear. We introduce a nuanced hypothesis, the 'coevolution effect'. The central concept is that the subdivision of host populations which occurs with habitat fragmentation causes localized coevolution of hosts, obligate parasites, and pathogens which act as 'coevolutionary engines' within each fragment, accelerating pathogen diversification, and increasing pathogen diversity across the landscape. When combined with a mechanism to exit a fragment (e.g., mosquitoes), pathogen variants will spill over into human communities. Through this combined ecoevolutionary approach we may be able to understand the fine-scale mechanisms that drive disease emergence in the Anthropocene.

Ecology and Emerging Infectious Diseases

Global habitat fragmentation is positively correlated with **emerging infectious diseases** (**EIDs**) (see Glossary) of wildlife origins moving into human populations [1–4]. Human land alteration continues to shift existing landscapes into island-like habitats with reduced area, increased isolation, and increased ratios of edge habitat to total area [5]. The linkages between habitat fragmentation and alterations in community structure have established the basis for most research on pathogen **spillover** and the ecology of infectious diseases in the past few decades through two key hypotheses: the **dilution effect** and the **amplification effect** (described in Box 1). These hypotheses use community ecology to explain this pattern, and contemporary reviews and meta-analyses [3,4,6] have provided great insight on the topic; however, the evolutionary mechanisms driving the association between habitat degradation and **zoonotic** disease emergence remain unclear. Importantly, to date it is unclear how pathogens with zoonotic potential arise in a degraded landscape.

To bridge this gap, here we describe a **coevolution effect** hypothesis that integrates theory from disease ecology, **landscape genomics**, and evolutionary theory, to explain an underlying mechanism that could drive the emergence of zoonotic infectious diseases in fragmented landscapes. Below, we describe our new hypothesis in detail and provide a comparison to major existing hypotheses in Table 1. We then describe ecological systems that are well suited for testing the predictions of the coevolution effect.

The Coevolution Effect

We propose the coevolution effect as a mechanistic hypothesis for understanding the interplay between hosts, **obligate parasites**, and pathogens to explain why the frequency of EIDs increases with habitat fragmentation. Pulling from theories of island biogeography [7], we

Highlights

Habitat loss is associated with emerging infectious diseases.

Most hypotheses to explain this association focus on ecological drivers and community structure; however, none have been developed to consider coevolutionary selective pressures across a fragmented landscape.

The 'coevolution effect' proposes that, within habitat fragments, shifts in population structure among hosts, obligate parasites, and pathogens function in parallel and act as 'coevolutionary engines', accelerating pathogen diversification, and therefore increasing pathogen diversity across a degraded landscape. When combined with bridge vectors (e.g., mosquitoes and ticks) this increases the probability that pathogens with zoonotic potential may spill over into human communities.

Multiple parasite–host systems such as non-human primates and lice, rodents and fleas, and bats and batflies, are amenable to testing this hypothesis, and can be integrated into existing studies of disease ecology at the community level.

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Box 1. Leading Ecological Hypotheses Describing Zoonotic Pathogen Spillover

Dilution Effect

The dilution effect hypothesis has been integral to the field as it introduced clear linkages between biodiversity and human health [50,54]. The hypothesis suggests that increased biodiversity reduces opportunities for zoonotic pathogens to leave forested landscapes, therefore reducing disease emergence. The hypothesis was first tested in northeastern USA, where the emergence of Lyme disease was found to be associated with shrinking habitats and a loss of biodiversity. The loss of competition and predation allowed the white-footed mouse (*Peromyscus leucopus*), wildlife reservoir of the causative agent, *Borrelia burgdorferi*, to proliferate and increase in population density relative to less-competent reservoir species. This, in turn, provides increased opportunities for arbovectors like ticks, which feed on *P. leucopus*, to become infected [55]. Global evidence for the dilution effect is prevalent, and the hypothesis has been tested in species ranging from white-footed mice to avian communities. Despite supportive evidence, this hypothesis has been heavily debated in the literature [56]. While the 'dilution effect' often holds true on small, spatial scales, on broad spatial scales, vertebrate biodiversity may actually amplify disease risk [52] through the 'amplification effect'.

Amplification Effect

Similar to studies on the dilution effect, studies of the amplification effect benefit from a community ecology approach [57] by examining the role of biodiversity which may positively correlate with disease risk. The amplification effect assumes that species-rich communities are associated with parasite-rich communities, and that diverse upstream host communities can drive the development of diverse parasite communities in downstream host populations [58].

Despite the controversy, associations between biodiversity and infectious diseases have been heavily explored and stimulated discourse. Recent Sin Nombre hantavirus findings suggest support for both the dilution and amplification effects simultaneously [59].

One key criticism of existing hypotheses is what happens at extreme ends of the spectrum (continuous and absent forest). A clarified model framework, 'optimal land-use theory', links habitat loss and pathogen emergence with community ecology, movement ecology, biodiversity, interspecies contacts, and host spatial use to describe a critical threshold of fragment size that is most likely to facilitate pathogen spillover [6], highlighting the role of habitat heterogeneity in disease emergence.

Table 1. Comparisons of Predictions for Factors Affecting the Emergence of Human Disease Based on the Dilution Effect, the Amplification Effect, and Our Proposed Coevolution Effect

Effect on emergence of human diseases	Dilution effect	Amplification effect	Coevolution effect
Habitat fragmentation	Increases risk	Decreases risk	Increases risk
Biodiversity	Loss of biodiversity increases risk	Loss of biodiversity decreases risk	Increase in pathogen biodiversity increases risk
Host population structure	NA[a]	NA	Increases risk
Coevolution of host and obligate vector	NA	NA	Increases risk
Lack of coevolution of host and bridge vector (mosquito)	NA	NA	Increases risk
Loss of predators and hosts (dilution of vectors)	Increases risk	NA	NA
Higher host–pathogen encounter rates (more edge)	Increases risk	Decreases risk	Increases risk if true for bridge vectors
Enhanced abiotic conditions for bridge vectors (higher population size for mosquitoes)	Increases risk	Increases risk	Increases risk

[a]NA, not applicable.

Glossary

Amplification effect hypothesis: where an increase in species diversity leads to increased disease risk. This is the opposite of the dilution effect [52].

Bridge vector: an arthropod vector (such as ticks and mosquitoes) that moves a disease-causing agent from a wild animal to a human.

Coevolution effect hypothesis: where pathogens, obligate parasites, and hosts have shifts in population structures in response to habitat fragmentation, and together they act as coevolutionary engines, increasing pathogen diversity across a fragmented landscape thereby increasing the likelihood of spillover from fragments into human communities through a mobilization mechanism (e.g., bridge vectors).

Coevolutionary engines: the process by which obligate parasites and hosts coevolve in a way that they accelerate the evolutionary trajectories of pathogens.

Coevolve: when two or more species evolve in response to selection imposed by one another.

Dilution effect hypothesis: where increased species diversity in an ecosystem leads to reduced disease risk [53]. The inverse of this hypothesis is the amplification effect hypothesis.

Emerging infectious diseases (EIDs): diseases caused by new infectious agents that have appeared in a population, or known infectious agents that are introduced into new populations or new geographic regions. Diseases that have been known, but have a newly recognized causative agent, or diseases which historically declined and now are re-emergent, may also be considered EIDs.

Enteric: pathogens that impact the gastrointestinal tract resulting in diarrhea and vomiting or other symptoms of disease.

Genetic drift: random change in the frequencies of alleles in a population.

Landscape genomics: a field of research integrating data on population structure with landscape features, to gain an understanding of the spatiotemporal flow of genes in a population.

Obligate parasite: an organism that lives on (ecto-) or in (endo-) a

emphasize that, while forest fragments may be considered to be islands, hosts may also be considered to be islands for their obligate parasites, and those parasites (in the case of obligate ectoparasites) are islands for vector-borne pathogens. The central concept is that, as the habitat becomes fragmented, the hosts, obligate parasites, and pathogens within each fragment become isolated and coevolve along different trajectories. We refer to the host, obligate parasite, and pathogens within each fragment as a coevolutionary engine. These diverging **coevolutionary engines** accelerate the rate at which pathogen genetic diversity increases in aggregate (i.e., across the landscape). The result is an increase in the overall probability that a pathogen variant evolves that can avoid host immune defenses and therefore have zoonotic potential.

This hypothesis is developed in the context of habitat fragmentation affecting vertebrate hosts (as these are the most likely to be reservoirs of human disease [3,4]), and their obligate parasites that circulate pathogens within the host population. In this context, the hypothesis depends on the following conditions.

  (i) A decrease in habitat connectivity increases population genetic structure in hosts and their obligate parasites in a non-sink population (or cold spot), resulting in genetic divergence among the forest fragments due to mutation and **genetic drift**.

 (ii) This increased isolation allows the host, obligate parasites, and pathogens within each fragment to **coevolve** along separate trajectories; we call these fragment-level units coevolutionary engines (Figure 1, Key Figure). These coevolutionary engines will accelerate genetic divergence among the pathogen populations within each fragment, resulting in greater overall genetic diversity of pathogens across the landscape, relative to a continuous habitat.

(iii) Mechanisms, such as **bridge vectors** (e.g., mosquitoes, ticks) that thrive in edge habitats [8], mobilize the diverse pathogen variants from wildlife in fragments to human communities, increasing the probability of disease emergence (Figure 1).

Below, we discuss in detail each of the conditions that would need to be tested and supported for the coevolution effect to be validated.

Habitat Fragmentation Increases Population Genetic Structure across Trophic Levels

Recent arguments suggest that anthropogenic habitat loss for wildlife will be the dominant evolutionary driving force of the Anthropocene [9]. Loss of habitat connectivity can increase the genetic structure of host species and alter the spatial distribution of their genetic diversity (as reviewed in [10]). The island-effect of fragmentation on the genetics of vertebrate host species has been demonstrated all over the globe, with examples in bats and owls [11], nonhuman primates [12], birds [13], and reptiles [14]. Habitat fragmentation has varied outcomes for genetic structure in the parasites within the hosts, depending upon the parasite and host life cycles, life histories, and vagility [15–17]. Thereby parasites that generally have more obligate lifestyles and higher host specificity will typically have increased genetic structure that mirrors that of their hosts, or even stronger genetic structure relative to their hosts due to their faster generation times. For example, a study of rock pigeons (*Columba livia*) found that their obligate wing lice evolve 11 times faster than their hosts [18]. Some recent examples of concordant genetic structure in hosts and obligate parasites include lice on pocket gophers [19], non-phoretic lice on birds [18], and mites on bats [20]. These types of coevolving host-obligate parasite systems with persistent populations that become increasingly isolated with habitat fragmentation (rather than becoming population sinks) are a fundamental requirement for the coevolution effect to play out as described in the next section.

Coevolutionary Engines Increase Pathogen Diversity across a Fragmented Landscape

Obligate parasites and their hosts have, in many cases, coevolved for millennia. Obligate ectoparasites like sucking lice, bat flies, and some fleas are species-specific blood feeders,

specific host and completes part or all of its life cycle and cannot survive as a species without the host.
Pathogen: a microorganism that triggers an infection in a host which may cause disease: specifically viruses, bacteria, and parasites – which, in this text, refer to protozoan or helminth species which are not obligate parasites (as defined above).
**Pathogen diversity:** the genetic variants of disease-causing agents found in an ecosystem.
Spillover: the movement of disease-causing agents (pathogens) from one species to another, which may (but does not always) result in disease.
Zoonotic: a term used to describe a class of pathogens or diseases which are transmitted from one species to another (typically described from animals to humans).

Key Figure

The Coevolution Effect Describes the Mechanisms Underlying Increased Zoonotic Pathogen Spillover with Habitat Loss
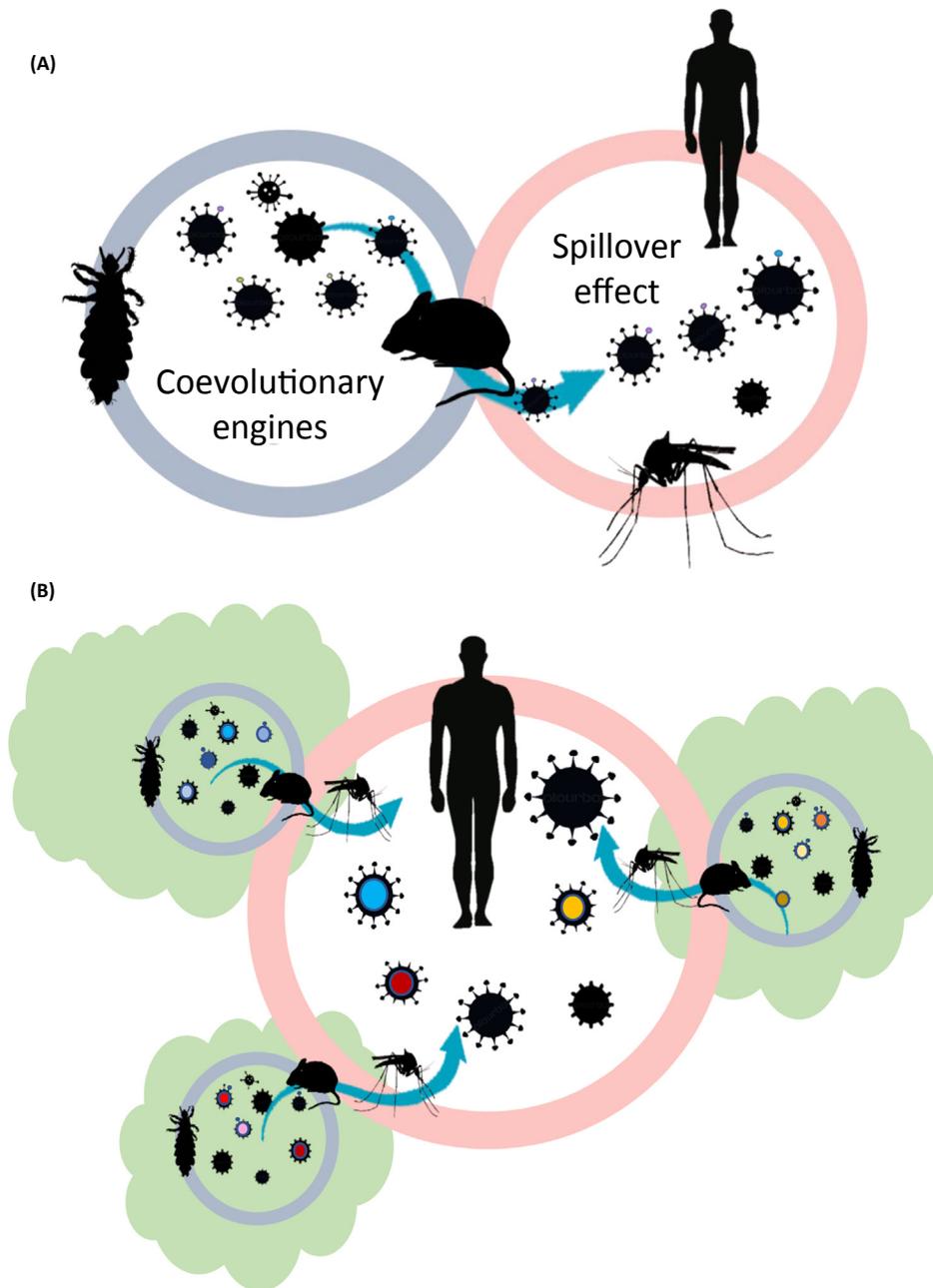
Figure 1. (A) The left (purple) transmission cycle of pathogens (indicated by black viral particles) between wildlife host (e.g., rodent) and obligate parasite (e.g., louse). Right (red) transmission cycle of pathogens through a bridge vector (e.g.,
(Figure legend continued on the bottom of the next page.)

meaning that they imbibe a blood meal at every feeding bout and have the potential to vector blood-borne pathogens among individuals of the host species. For example, in human populations, body lice have been responsible for outbreaks of typhus [21], trench fever [22], epidemic relapsing fever [23], and plague [24,25]. Therefore, while spillover of these pathogens occurs, something has to keep them circulating in host populations, which we propose is heavily facilitated by obligate parasites. These parasites are often ignored in disease ecology because they are host-specific and therefore unable to mediate a vector-borne host switch. However, we propose that obligate hematophagous parasites play a critical role in the circulation and evolution of pathogens in host populations. Within a fragmented landscape, the obligate parasites would be maintaining the circulation of pathogens within a host population restricted to a forest fragment.

If we look at the pathogen, obligate parasite, and host species in isolation, the null (i.e., neutral) expectation is that allele frequencies within fragmented populations will begin to diverge due to mutation and genetic drift as they become more isolated. While genetic diversity may decrease within each subpopulation, the divergence among them will cause the overall diversity across fragments to increase (see Box 2 for more details). Thus, even assuming neutrality, it is clear that taking an evolutionary genetic perspective to the issue of EIDs is important. When we layer on top of this neutral expectation that the host, parasite, and pathogens are engaging in an increasingly independent coevolutionary arms race within each fragment, the rate of divergence among fragments increases over time, especially for pathogens with fast mutation rates and effective population sizes that are likely not affected by the habitat fragmentation. Thereby, forest fragmentation creates a scenario where each persistent forest fragment harbors a coevolutionary engine comprising the host, obligate parasite, and pathogens circulating between them. The divergence among these engines will increase the genetic diversity of pathogens across a fragmented landscape (Figure 1). This increased pathogen genetic diversity across the landscape is a requirement of the coevolution effect hypothesis as it would increase the probability that a genetic variant arises that has the capacity to avoid host immune defenses and be pathogenic in another species, such as humans.

Mobilization Mechanisms Facilitate Spillover of Pathogens Generated in Coevolutionary Engines

The coevolutionary engines described above are interesting from an evolutionary perspective, but are not relevant to human health on their own. For the coevolution effect to contribute to the emergence of infectious diseases, there must be evidence of the pathogens moving from the fragments to human populations at least at the same rate as from contiguous forests. Many zoonotic pathogens found in obligate ectoparasites and their hosts are also transmitted by arthropod vectors [26–30]. Edge habitats surrounding forests are known for their increased abundance in mosquitoes due to ideal breeding habitat conditions [8,31]. Therefore, in a situation where **pathogen diversity** is increased, and arthropod vector abundance is maintained or even increased, there is an opportunity for arthropods to act as bridge vectors, mobilizing the pool of diverse pathogens from fragmented habitats to unique human and animal

mosquito) among hosts causing the spillover of the pathogens that diversified in a species-specific cycle (purple). (B) Loss of habitat connectivity leads to small forest fragments (green shapes) that act as separate coevolutionary engines, with genetic drift and selection causing divergence in pathogens among fragments indicated by the colored viral particles in each fragment. Across a landscape, an accelerated rate of pathogen diversification and increased probability that a new variant would have zoonotic potential in other species (e.g., humans). With transmission through the bridge vector maintaining exposure to the pathogens (spillover), this process would lead to an increased probability of disease emergence in a fragmented habitat.

Box 2. Habitat Fragmentation Can Lead to Increased Genetic Diversity

Assuming neutral evolution (i.e., ignoring natural selection), a loss of subpopulation connectivity due to habitat fragmentation will cause increased genetic diversity within hosts, obligate parasites, and **pathogens** (parasites, bacteria, and viruses) across the fragmented landscape. When coevolutionary selective pressures among these species are occurring within each fragment, the trend of increasing diversity will likely be accelerated. We can quantify the neutral expectations of increased genetic diversity using population genetic and phylogenetic theory.

Following the loss of connectivity, the overall genetic diversity (π) of a species can be partitioned into the diversity within (π_W) and among the sites of low connectivity (π_F). Assuming no migration among fragments, the null expectation for the latter can be represented by

$$E[\pi_F] = 2N_e\mu + 2\frac{t}{g}\mu \qquad [\mathrm{I}]$$

where N_e is the effective haploid population size prior to fragmentation, t is the time since fragmentation in years, g is the generation time (years per generation), and μ is the rate of mutation per base per generation. Thus, following fragmentation, the diversity among fragments will increase independently of the fragment population sizes at a rate of $2(t/g)\mu$. When there are multiple fragments, the diversity among fragments explains most of the overall diversity, despite the loss of diversity within fragments due to smaller population size. Thus, the null expectation is an increase in overall diversity across the fragmented sites following the loss of connectivity, the rate of which is greater with increasing mutation rate and decreasing generation time (Figure I). While we do not expect dramatic divergence among host subpopulations, even subtle shifts in allele frequencies across their genome could create different selective environments for the obligate parasite and pathogen. The coevolutionary selective pressures among hosts, obligate ectoparasites, and pathogens will further accelerate the rate at which the subpopulations diverge, and, as a result, the rate at which diversity increases [60] across the landscape. In other words, within each fragment coevolutionary engines generate pathogen diversity.
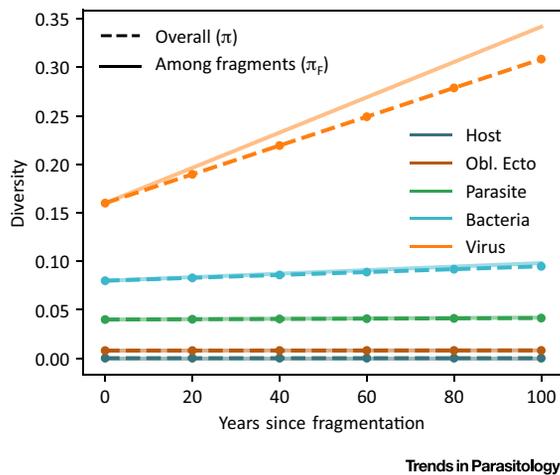

Figure I. An Increase in Diversification in Host–Parasite–Pathogen Systems with Fragmentation.

communities, assuming the mobilization mechanism is not pervasive enough to homogenize pathogen diversity across the fragments. Therefore, the third condition of the coevolution effect hypothesis requires that the new pathogen variants generated through coevolutionary engines can be mobilized out of forest fragments and increase the probability that a new variant of pathogen will emerge as the causative agent of an infectious disease in humans. Additionally, bridge vectors can expose humans to the increased pathogen diversity as they move through a fragmented landscape (Figure 1B). We highlight the role of arthropod bridge vectors because it is predicted that the majority of emerging infectious diseases in humans in the future will be vector-borne [32,33]. However, the coevolution effect hypothesis was developed such that any obligate parasite–host system distributed across a fragmented landscape may act as a coevolutionary engine for pathogen diversity; therefore, pathogens with other transmission

routes may also move out of forest fragments mechanically or environmentally into locations where humans can be infected.

Model Systems for Testing the Coevolution Effect

We have presented the coevolution effect hypothesis to describe a mechanism driving zoonotic pathogen spillover from wildlife to human communities. This hypothesis links the known association between habitat loss and disease emergence with fine-scale patterns of host–parasite population genetics. In the previous section we define the conditions that must be met to validate this hypothesis. These predictions could be tested using new or existing datasets through modeling, experimental approaches, or field research that quantifies spatial and/or temporal genetic diversity across the host, obligate parasite, and pathogens. While the coevolution effect can be applied to disease systems with any transmission route, for example obligate **enteric** parasites that circulate through populations and then have opportunities to exit coevolutionary engines and spillover through the environment (water, soil, etc.), we propose that it may be best to test this hypothesis using vector-borne diseases. Of all documented EIDs, 29% have been vector-borne [1,4], and recent literature has highlighted that arthropod vectors, like ticks and mosquitoes, are increasing in abundance and distribution due to climate change and human movement, as is the number of emerging RNA arboviruses [33,34]. Therefore, the coevolution effect hypothesis may be best tested in model systems to explain the mechanisms underlying the boom in emergence of zoonotic RNA arboviruses. RNA viruses frequently spill over from one species to another, likely because of their ability to avoid host immune defenses, and evolve rapidly due to extremely high mutation rates [35,36]. This allows RNA viruses to adapt to new hosts and environmental conditions at very rapid rates, which makes them excellent model systems for investigating pathogen evolution in vectors and hosts using real-time sampling. Zika virus (ZIKV), Chikungunya virus (CHKV), West Nile virus (WNV), and Rift Valley fever virus (RVFV) are just a few contemporary examples of mosquito-borne RNA viruses that have spilled over from animal hosts to humans and now present public health threats. Here we propose obligate parasite and host taxonomic systems in which to evaluate the coevolution effect.

Primates, Bats, and Rodents

In 2017, Olival *et al.* [4] developed a database of every known virus of mammals, and the 754 species infected by the viruses, to better identify the mammalian species that are most likely to host zoonotic viruses, the geographical location where the most undescribed zoonotic viruses are likely to emerge, and viral traits that correlate with whether a virus will be zoonotic or not. The results identified primates, bats, and rodents to be the organisms most likely to host zoonotic viruses. Primates, due to their phylogenetic proximity to humans, rodents, due to their resilience to human disturbances, and bats in particular were found to host higher numbers of zoonotic viruses than other mammals, due to their metabolic protection against febrile illness, high density populations, and migratory behaviors.

Non-human Primates (NHPs)

Human infections, including yellow fever and infections caused by Chikungunya fever virus and Zika fever virus, are likely to have originated in NHPs [4,37]. Given the physiological similarity among primates it is hypothesized that spillover is likely to occur from NHPs to humans [3]. Sucking lice (Phthiraptera: Anoplura) are hematophagous, host-species-specific, obligate ectoparasites which show widespread movement between host individuals in wildlife populations [38,39]. There is evidence of high species specificity in NHP sucking lice [40–42], and genetic evidence of host-switching between primate species [40,42]; however, not much work has been done on the characterization of pathogens in lice from wildlife. Pathogens such as

species of *Anaplasma* and *Rickettsia* have been detected in sucking lice from domestic animals and livestock [26,43]; therefore, we hypothesize that the lice of wild animals will harbor blood-borne pathogens, especially given the vector potential of human sucking lice. We recommend pathogen screening of sucking lice as a first step in the approach to test the coevolution effect hypothesis in NHPs. There is plenty of evidence of NHP shifts in population genetic structure with habitat fragmentation from Africa, South America, and Madagascar [5,12,44,45], and sucking lice have been identified in many of these species, making NHPs and sucking lice ideal systems to examine the concept of coevolutionary engines.

Bats

Bats are recognized as natural reservoirs of a large number of disease-causing agents that lead to emerging infectious diseases in humans [27,46]; therefore, understanding the obligate and bridge vector communities on bat populations can provide significant insight into pathogen evolution and the mechanisms that drive emerging infections. Specifically, bat flies (Diptera: Hippoboscidae), are obligate blood-sucking parasites, and adults have vestigial wings that restrict them to their host species. By feeding on host blood they also encounter any blood-borne pathogens the host may have, and they move between individuals via direct contact, similar to sucking lice. These flies are viviparous, and larvae are fed by the female until they pupate. It has been proposed that this life history strategy may facilitate vertical pathogen transmission, amplifying their role as disease vectors. Recent evidence has shown that the bacteriome of bat flies captured from wild Malagasy bats contains bacterial genera with well-known zoonotic agents, like *Bartonella* and *Rickettsia* [47], and new viruses (in the family Rhabdoviridae) have been discovered in bat flies infesting a new species of pteropodid bat in Uganda [48]. There is also evidence of bat flies infected with bat malaria parasites (*Polychromophilus* spp.), and evidence that they may play a role in the transmission of the disease in caves in Central Africa [49]. Due to their host specificity and the mounting evidence that they vector infectious pathogens, bat flies and their host species are excellent models with which to test the coevolution effect.

Rodents

Rodents are recognized as competent reservoirs in the emergence of zoonotic human infectious diseases such as Lyme disease, bubonic plague, and disease caused by viruses of the Hantaviridae, etc. [50]. There is a wealth of literature on fleas (Insecta: Siphonaptera) and their vector competence. Similar to bat flies and sucking lice, fleas are obligate hematophagous ectoparasites that have the ability to transmit zoonotic disease-causing agents [29], and over 2500 species have been described from mammals and birds, with the majority parasitizing rodents. However, unlike bat flies and lice with high host specificity, fleas are not always species specific, but fleas likely emerged with mammals and speciated with rodents [51], so they exhibit specificity to groups of mammals. Similar to bat flies and sucking lice, fleas can transmit pathogens through contaminated fecal pellets near the feeding site; however, they can also regurgitate blood meals, making them highly competent vectors. There are numerous examples of zoonotic pathogens circulated by fleas in rodent populations, including murine typhus (*Rickettsia typhi*), plague (*Yersinia pestis*), and cat scratch disease (*Bartonella henselae*). We propose that fleas and rodents could also be interesting models for testing the coevolution effect hypothesis. There are also large existing datasets on the evolution of RNA viruses that are not vector-borne (such as members of the Hantaviridae) which may be used to test this hypothesis.

Not Just Ectoparasites and Vector-borne Disease

While the proposed hypothesis may best be tested by examining vector-borne disease dynamics, the concept may be applied to any system where hosts and obligate parasites

are isolated in an island-like fragmented setting where they may act as coevolutionary engines. Rather than the increased pathogen diversity being mobilized to human communities through bridge vectors, in this scenario environmental conditions may facilitate that role. For example, parasites transmitted by the fecal–oral route may cycle through a population of hosts in a forest fragment more rapidly due to density- and frequency-dependent transmission than in a continuous forest environment. If host populations are isolated, we would expect to see an increase in parasite diversity across the fragmented landscape. With a growing human population living in close proximity to fragmented environments our hypothesis describes a potential reason for why we may see a global increase in pathogen diversity in a time when over 70% of the world's forests are near fragments [5].

Concluding Remarks

Here, we introduce a new hypothesis rooted in evolutionary theory to describe the underlying mechanisms of zoonotic pathogen spillover associated with habitat loss. With strong evidence in the literature supporting the idea that ecological communities can facilitate the emergence of zoonotic infectious diseases, we aim to fill a knowledge gap describing the underlying evolutionary processes that drive this phenomenon (see Outstanding Questions). Our hypothesis was developed such that it can be integrated into existing empirical and theoretical studies examining the dilution and amplification effects, and as such can provide additional information on populations and coevolutionary trajectories. Through these combined ecological and evolutionary approaches, we may be able to understand the fine-scale mechanisms that drive disease emergence in the Anthropocene.

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Outstanding Questions

Can forest connectivity predict parasite diversity (is an increase in parasite diversity seen across a fragmented versus a continuous landscape)?

Are emerging infectious diseases in human populations associated with an increase in pathogen diversity on the landscape?

Should host reservoir studies include data on associated obligate parasites and parasite population genetics on hosts?

Can the dilution, amplification, and coevolutionary effects be examined simultaneously in research on the coevolution effect?

What methods can be used to alleviate coevolutionary engines of pathogen diversity and prevent zoonotic spillover events? Can high pathogen diversity be detected in bridge vectors or the environment in between fragments?

Within forest fragments, does parallel coevolution between hosts, parasites, and vector-borne pathogens occur – thus, serving as 'coevolutionary engines' driving rapid genetic divergence among pathogen populations?

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