

## Is biodiversity bad for your health?

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**Abstract.** Natural ecosystems provide services that support human well-being, but ecosystems may also contain elements that can endanger humans. Some researchers have argued that ecosystems that support high vertebrate diversity pose a danger to human health because they are likely to support a high diversity of zoonotic pathogens, leading to the emergence of infectious diseases. We evaluated the evidence for the three necessary links in the hypothesized causal chain linking high vertebrate diversity to a high probability of emergence of infectious diseases. We found no support for one critical link—that high total diversity of vertebrate pathogens correlates with high diversity of actual or potential zoonotic pathogens. In contrast, there is now substantial evidence that high diversity *protects* humans against the transmission of many existing diseases. These results have substantial relevance for environmental policy.

**Key words:** biodiversity; dilution effect; ecosystem disservices; ecosystem services; emerging infectious disease; One Health; spillover.

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### INTRODUCTION

Natural ecosystems absorb and recycle nutrients; produce biomass, food, and water; modulate the impacts of physical forces on living organisms; and support the life cycles of myriad species of plants, animals, and microbes. When these functions are considered useful for humans, we term them “ecosystem services” (Millennium Ecosystem Assessment Program 2005, Daily and Matson 2008, Guerry et al. 2015). Natural ecosystems can also endanger human well-being, for example, when fires and floods destroy lives and property or when dangerous animals and plants compromise human health. These negative effects of ecosystems on humans, sometimes termed “ecosystem disservices,” often arise when humans have altered natural ecosystems, inadvertently

converting their positive effects into detriments (Dunn 2010). For example, the eutrophication of coastal waters can undermine the ability of these waters to support fisheries and recreation—an ecosystem service—and compromise human and animal health through harmful algal blooms—an ecosystem disservice.

Recently, some researchers have emphasized the potential for *undisturbed* ecosystems to be sources of disservices and outright harm to humans (Burgin et al. 2013, von Doehren and Haase 2015). Natural ecosystems certainly contain dangerous elements—venomous snakes, allergens, and natural fire regimes—that can, even when undisturbed, compromise human well-being. Recently, it has been suggested that native biodiversity may be dangerous because it increases the probability that a new zoonotic

disease will emerge, constituting an important ecosystem disservice (Jones et al. 2008, Dunn 2010, Dunn et al. 2010, Johnson et al. 2015a). If biodiversity poses a real health risk to humans, then enthusiasm for the conservation of biodiversity might be dampened by concerns about disease emergence. Here, we explore the evidence that high native biodiversity increases the likelihood of emergence of human infectious diseases. We compare the evidence concerning the effects of native biodiversity to that concerning anthropogenic changes in biodiversity affecting disease emergence and dynamics, and we reflect on the policy and management consequences of this evidence.

## MECHANISMS OF DISEASE EMERGENCE

Infectious diseases of humans can emerge through a variety of pathways, only some of which are related to biodiversity. For example, Rosenthal et al. (2015) identified seven mechanisms, or pathways, that have been used to identify emerging diseases or pathogens in humans: (1) when a disease increases in incidence, (2) when a disease increases in impact, (3) when a disease increases in geographic range, (4) when a pathogen has undergone recent evolutionary change, (5) when a pathogen is detected in the human population for the first time, (6) when a pathogen significantly changes its pathology or clinical presentation, or (7) when a pathogen is discovered for the first time. Despite the diversity of these mechanisms, most attention has been devoted to the “spillover” process, whereby a pathogen that is typically restricted to non-human vertebrate hosts is transmitted to humans. Spillover to humans seems most compatible with pathways 4 or 5 above. However, these two pathways rank third and fifth of the seven in terms of frequency of occurrence in human emerging infectious disease (EID) events (Rosenthal et al. 2015). Despite the dominance by pathways that do not necessarily involve spillover, many conceptual models of human disease emergence emphasize the spillover pathways (e.g., Wolfe et al. 2007, Jones et al. 2008, Hatcher et al. 2012, Gortazar et al. 2014).

Jeschke et al. (2013) described how a spillover event, or species jump from a non-human vertebrate host to humans, is the first of several sequential steps required for emergence. Following spillover, establishment, spread, and impact

on hosts are required to constitute an EID. For the latter three steps, evidence is accumulating that high biodiversity is strongly inhibitory (Keesing et al. 2010, Ostfeld and Keesing 2012, Civitello et al. 2015a, Johnson et al. 2015b). Here, our main focus is on the first step—the spillover process.

## THE LOGICAL BASIS FOR THE ARGUMENT THAT HIGH DIVERSITY INCREASES DISEASE EMERGENCE

The notion that high native biodiversity increases the threat of human exposure to zoonotic diseases rests on the assumption that all vertebrates are potentially dangerous because any one might be the source of spillover that results in the next deadly EID (see Jones et al. 2008, Dunn et al. 2010). The precise mechanisms proposed to link high vertebrate diversity to high risk of zoonotic emergence are typically not specified. Here, we provide a logical structure and evaluate the evidence for this connection. In a later section, we evaluate an alternative causal pathway involving vertebrate diversity and the host range of pathogens.

The primary logic by which greater diversity could lead to higher zoonotic risk requires a three-part causal chain connecting vertebrate diversity to pathogen diversity to risk of human exposure to a zoonotic EID (Fig. 1). First, the logic requires that the more species of mammals and birds there are in any given location, the more total vertebrate-borne pathogen species will occur there. In order for this to be the case, each species of mammal or bird must host at least some unique pathogens. Alternatively, if most pathogens are widely distributed across hosts, then the number of host species will be less important. The second necessary link in the causal chain is that the more total species of pathogens there are in an area, the more potentially zoonotic species should occur there. This supposition requires that the zoonotic pathogens are roughly equally distributed across all vertebrate pathogens. If the zoonotic pathogens are found predominantly in a few host taxa, then the number of host species will be less important. And finally, the logic of this connection requires that the more potentially zoonotic pathogens that exist in an area, the more human disease we can expect (with human disease variously measured

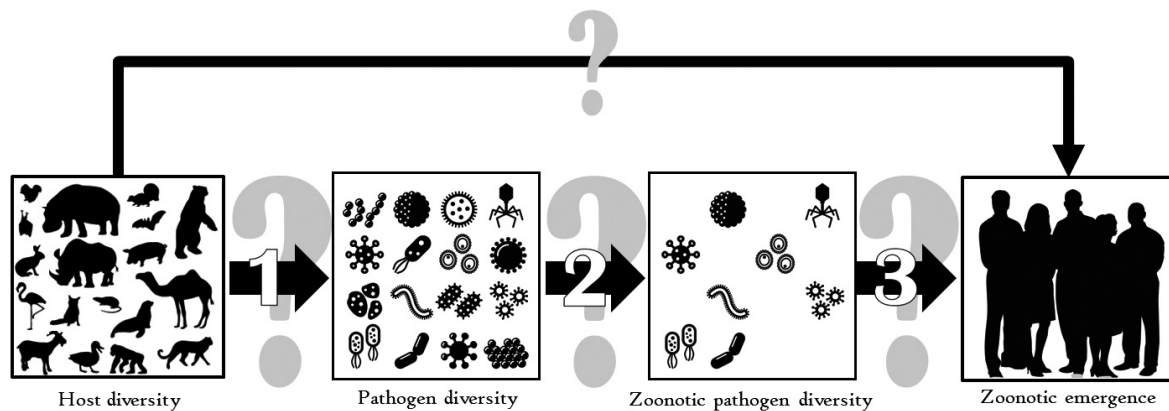


Fig. 1. The necessary logical steps underlying the argument that high host diversity leads to high probability of the emergence of a zoonotic disease. High diversity of vertebrate hosts must result in high total diversity of pathogens within the vertebrate community, which in turn must lead to high diversity of actual or potential zoonotic pathogens (those that can infect humans and cause disease), which in turn must increase the probability of new emergence events. Although a link between host diversity and parasite diversity is relatively well established, effect of host diversity on viral and bacterial pathogens (arrow 1) is not. Evidence does not support a link between overall pathogen diversity and that of actual or potential zoonotic pathogens (arrow 2). Some evidence supports correlations between diversity of zoonotic pathogens and the likelihood of zoonotic emergence (arrow 3), but with important caveats described in the text.

as prevalence, severity, or the probability of a new emergence event). What is the evidence for the three links in this causal chain (Fig. 1) connecting vertebrate diversity to zoonotic EIDs?

#### *The first link: Does higher vertebrate diversity lead to more total pathogen diversity?*

Natural ecosystems are rich in parasites and pathogens (Hudson et al. 2006). To the extent that each free-living species has at least some unique pathogens, high diversity of free-living species should lead to high diversity of pathogens; in other words, diversity begets diversity (Hechinger and Lafferty 2005, Dunn et al. 2010, Johnson et al. 2015a). However, because pathogens may be shared among several or many hosts, adding more species of hosts might not lead to a linear or even a predictable increase in species richness of pathogens. For example, Koh et al. (2004) found that the correlation between vertebrate diversity and parasite diversity was considerably weaker when the parasites had low host specificity. Johnson et al. (2016) found that the diversity of amphibian parasites increased with that of their hosts, but whereas parasite diversity increased consistently with

the area sampled, host diversity saturated at larger areas.

In a synthesis of the available evidence concerning helminths, Dobson et al. (2008) argued that “patterns of parasite diversity do not clearly map onto patterns of host diversity,” pointing out, for example, that some parasites of fish (e.g., monogeneans) are more diverse, but others (e.g., gut parasites) less so, where hosts are more diverse. Focusing on North American carnivores, Harris and Dunn (2010) found that in general, the species richness of carnivore parasites increased with increasing host species richness. However, in some geographic areas, such as the northern portion of North America, the correlation between host and pathogen diversity was weak. Examining 38 case studies of protozoan and metazoan parasites infecting animal hosts (both vertebrates and invertebrates) identified through literature searches, Kamiya et al. (2014) found significant positive correlations between parasite and host diversity (effect size [ $r$ ] = 0.55). Their meta-analytic methods suggested that the correlation was somewhat weaker for mammals ( $N = 7$  host taxa,  $r = 0.43$ ) than for birds ( $N = 11$  host taxa,  $r = 0.65$ ), although both relationships were

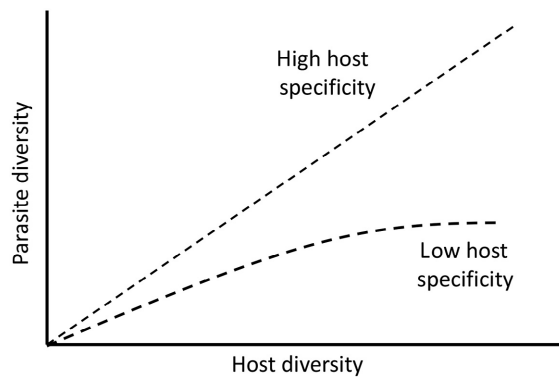


Fig. 2. Schematic diagram of how parasite diversity is expected to vary with host diversity when parasites show high host specificity (upper curve) and when they show low host specificity (lower curve). In the latter case, the sharing of parasites between hosts means that the diversity of parasites will saturate as host diversity increases, resulting in little or no additional increases in parasite species at high levels of host diversity.

statistically significant. Whether diversity of viral and bacterial parasites correlates with diversity of their hosts has not been similarly analyzed.

Based on these studies, there appears to be moderate support for the supposition that greater species richness of vertebrates leads to greater richness of vertebrate-borne pathogens, but there are caveats. One key limitation is that the effect of vertebrate diversity on that of viruses and bacteria, which remain the dominant groups of emerging infectious diseases of humans (Jones et al. 2008), is largely unknown. Another caveat is that nonlinear relationships caused by host sharing (non-specificity by pathogens) or by different species–area curves for hosts and pathogens might weaken the correlation within some regions of parameter space. For instance, with extensive host sharing among pathogens, pathogen richness might rise with increases in host richness from low to moderate levels. But above those moderate levels of host richness, pathogen richness might increase modestly or not at all, as few unique pathogens are added (Fig. 2). Future studies should incorporate the relationship between host diversity and the diversity of bacteria and viruses specifically, and also explore the shape of the relationship between host and pathogen diversity.

*The second link: Higher diversity of all pathogens leads to higher diversity of potentially zoonotic pathogens?*

If communities with more species of vertebrates support more total species of pathogens, we might expect these diverse communities to pose a higher risk of zoonotic emergence. But this would be true only if communities supporting more total species of pathogens also support more potentially zoonotic pathogens. To what degree is total pathogen richness correlated with the richness of potentially zoonotic pathogens?

We are not aware of any direct tests of possible correlations between total pathogen richness within vertebrate communities and the richness of potential or actual zoonotic pathogens. Such tests would require reliable estimates of total pathogen richness within vertebrate communities, which appear to be rare or absent. Nevertheless, if zoonotic pathogens are equally likely to arise from any host species within the vertebrate community, then this correlation would be plausible and even expected. However, some host taxa are much more likely than others to act as sources of zoonotic transmission. In particular, rodents, and secondarily carnivorans, are more likely to act as hosts for zoonotic pathogens than are birds, other mammals, or other vertebrate taxa (Johnson et al. 2015a, Han et al. 2016). Far more species of rodents ( $N = 244$ ) host zoonotic pathogens than do species in other mammalian orders, and rodents carry 85 unique zoonotic pathogens, which is more than the number hosted by any other mammalian order (carnivorans are second in both respects, with 139 species hosting 83 unique zoonotic pathogens; Han et al. 2016). Other well-studied groups of mammals, including the chiropterans, ungulates, and primates, are the sources of far fewer zoonotic pathogens, although chiropterans might be particularly important hosts for zoonotic viruses (Luis et al. 2013). Within the rodents, species with fast life history traits (e.g., early age at maturity, large litters, short life span) are more likely than those with slow life history traits to act as reservoirs for zoonotic pathogens (Han et al. 2015). These life history traits tend to be correlated with commonness rather than rarity (Stearns 1992, Blackburn et al. 1996). Similarly, Johnson et al. (2015a) found that wild rodents were the reservoirs for most (55/95) zoonotic



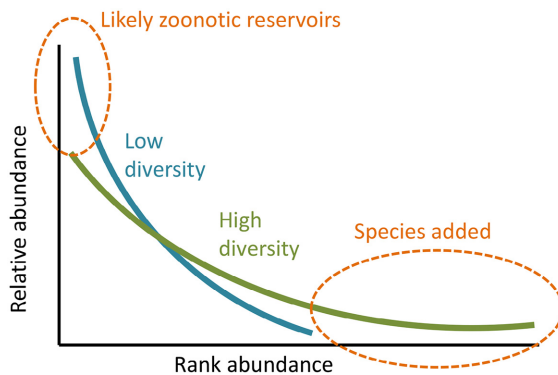


Fig. 3. Schematic representation of typical rank–abundance curves, in which the relative abundance of each species is represented on the vertical axis and the rank of each species, from highest to lowest abundance, is given on the horizontal axis. Contrasted are two scenarios, a relatively low-diversity community in blue and a relatively high-diversity community in green. The curves represent the common observation that higher-diversity communities include more species that are rare and fewer that are common. The species added (right-hand orange circle) in higher-diversity communities are not likely to be the sources of zoonotic pathogens, whereas the most abundant species in lower-diversity communities (left-hand orange circle) are often the sources of zoonotic infection.

viruses, that zoonotic virus spillover from wild-life to humans was most frequent in and around human dwellings and in agricultural fields, and that rodents were the most likely hosts to be implicated in transmission that occurred around human dwellings and agricultural fields.

As a consequence, the pathogens occurring within some elements of the vertebrate host community, namely those in chiropterans, ungulates, and primates (and within most other mammalian orders; Han et al. 2016), appear to be less important to zoonotic disease emergence than those occurring in the rodents and carnivorans. It is therefore critical to ask whether communities with high vertebrate diversity necessarily contain more species of hosts for pathogens with high zoonotic potential.

Ecologists have traditionally compared species diversity between different communities using rank–abundance curves, also called dominance–density curves or Whittaker plots (Whittaker 1965, Krebs 1999), which are frequency distributions of

species ordered by their abundance (or the log of their abundance). Most, if not all, communities are characterized by a few highly abundant species and many more rare species (Fig. 3). When communities differing in species richness are compared using rank–abundance curves, more diverse communities frequently have longer tails; in other words, more diverse communities have relatively more rare species (Magurran 2004). One would expect, therefore, that moving along a gradient from less diverse to more diverse vertebrate communities, one would see an accumulation of rare species, with the more abundant species becoming proportionally less dominant (Magurran 2004; Fig. 3). If these rare species were likely to act as hosts for actual or potential zoonotic pathogens, then one would expect more diverse vertebrate communities, potentially with more species of pathogens overall, to be sources of more zoonotic pathogens. However, the evidence suggests that the more common and widespread species of vertebrates, such as rodents, rather than the rare ones occurring only in high-diversity communities, are more likely to act as reservoir hosts for zoonotic pathogens. This pattern has now been observed repeatedly in nature (Keesing et al. 2010, Ostfeld et al. 2014, Han et al. 2015, Johnson et al. 2015b). Not only do rare species appear less likely to act as zoonotic reservoirs, but their very rarity will often reduce their potential for zoonotic spillover into humans.

Han et al. (2016) found a positive correlation across mammalian orders between the total number of species and the number of species known to host zoonotic pathogens. In other words, the more species there are within a mammalian order, the more species within that order host zoonotic pathogens. Based on Figure 2 in Han et al. (2016), about 10% of species within any given mammalian order act as hosts for zoonotic pathogens, with somewhat lower percentages for chiropterans and soricomorphs and somewhat higher percentages for carnivorans and artiodactyls. Thus, in general, the more species-rich orders are expected to host more zoonotic pathogens, but this observation is not directly relevant to the question of whether more diverse vertebrate communities necessarily contain more species that serve as zoonotic reservoirs.

On the basis of this evidence, we suggest that increases in total pathogen richness that likely

accompany increases in species richness of vertebrate hosts generally do not cause increases in the richness of potential or actual zoonotic species, severing the causal chain linking high diversity to high human disease potential. At the very least, this necessary link in the causal chain currently has no support.

A rigorous evaluation of the hypothesis that areas with higher diversity of vertebrates have higher diversity of zoonotic pathogens will require new information on hosts and pathogens along diversity gradients. A two-part process could potentially provide the necessary data. The first step would be to determine how frequently lower-diversity communities of vertebrates consist of random, vs. non-random, subsets of higher-diversity communities. Existing evidence suggests that low-diversity communities typically contain nested subsets of their higher-diversity analogs (Wright et al. 1998), but these investigations are typically based on presence-absence data about each species. Data on changes in the relative abundance of species along the diversity gradient would be even more helpful for quantifying community structure, particularly to see whether it conforms to the hypothesis illustrated in Fig. 3. The second key step for evaluating the connection between diversity and zoonotic species richness would be to determine, for most or all of the species present in these communities, which species transmit which zoonotic pathogens, and how efficiently. Available databases of pathogens detected in members of vertebrate communities are inadequate for addressing the hypothesis. These databases (e.g., Johnson et al. 2015a) consist of both direct and indirect (e.g., immunoassay) measures of pathogens in potential hosts, but cannot by themselves determine whether pathogen-positive hosts are reservoirs (amplifying hosts) or dead-ends (buffering, or dilution hosts). Consequently, the mere detection of pathogen exposure within a particular host should not be considered evidence that the host transmits the pathogen. For example, for the many zoonotic pathogens transmitted by generalist vectors, most species within a vertebrate community might test seropositive, indicating exposure, but few permit pathogen amplification and onward transmission (Ostfeld and Keesing 2012). Together, these two sets of data—the community composition of species across diversity gradients and the transmission potential of these

host species for zoonotic pathogens—would indicate how the risk of human exposure to emerging or recognized zoonotic pathogens changes along gradients in vertebrate diversity.

*The third link: Higher diversity of potentially zoonotic pathogen species leads to more human disease?*

The last link in the proposed causal chain between biodiversity and human disease connects zoonotic pathogens to human cases. If each species of zoonotic pathogen in a community had an additive or synergistic effect on human disease incidence or severity, then one would expect that the existence of more zoonotic pathogens in a region would lead to more human disease. An alternative would be that the presence of multiple pathogens would reduce the net impact on health, for example, through mutual interference that reduced each pathogen's impacts.

Dunn et al. (2010) considered one perspective on this question. Focusing just on leishmaniasis, trypanosomes, malaria, schistosomes, filariae, spirochetes, and leprosy, they asked whether the status of these diseases (endemic, sporadic, non-endemic) in a country was correlated with the total species richness of human pathogens (bacteria, viruses, helminths, and protists) in that country. For example, they asked whether the likelihood of leishmaniasis being endemic in a particular country was a function of the total number of different pathogens in that country. They found that pathogen richness was a significant positive predictor of disease status (greater endemicity). Interestingly, Dunn et al. (2010) found that per capita spending on health care was the strongest negative predictor, and minimum actual evapotranspiration the strongest positive predictor, of endemic status of the selected diseases. Although their analysis was not restricted to zoonotic pathogens, their results provide some support for the proposed link between zoonotic pathogen richness and these particular human diseases. To our knowledge, however, the effect of total pathogen richness on the most epidemiologically important zoonotic diseases—those caused by viruses and bacteria—has not been addressed.

One other way that greater zoonotic pathogen richness could influence human disease is through simultaneous infections (coinfections).

Both positive and negative effects of coinfection, as compared to single infection, on human and animal hosts have been detected, suggesting that both mutual inhibition and facilitation among pathogens occur in a variety of disease systems (Vaumourin et al. 2015). However, a quantitative review of the effects of coinfection on human health showed a strong tendency for coinfections to correlate with poorer human health and increased pathogen abundance when compared to single infections (Griffiths et al. 2011), although this study did not indicate how many of these pathogens were zoonotic. Griffiths et al. (2011) make the critical point that the correlation between coinfection and reduced health of individual hosts could be caused by an underlying negative health condition that makes coinfection more likely (i.e., poor health causes coinfection), or by multiple infections facilitating one another to the detriment of host health (coinfection causes poor health). The direction of causality is difficult to determine without experimental manipulations. Recent experimental research in diseases of wildlife and plants (Johnson and Hoverman 2012, Johnson et al. 2013, 2016, Rottstock et al. 2014) shows that higher pathogen diversity can cause lower disease severity, suggesting that mutual interference might be common. These studies indicate that pathogen diversity is often a poor metric for assessing epidemiological impact, which might be better measured by prevalence of infection (Johnson et al. 2015b). The lack of similar experimental studies of multiple vs. single infections on human hosts makes it difficult to apply these results to human epidemiology. Although the limited evidence currently available supports a positive correlation between numbers of zoonotic pathogens and incidence or severity of human disease, the generality of this correlation cannot be thoroughly assessed at present. Potentially counteracting the effect of zoonotic pathogen diversity and human disease is the observation that diversity of taxa and their abundance are often negatively correlated in ecological communities due to enhanced competition or impact from natural enemies. If higher pathogen diversity reduces pathogen abundance, disease risk might be decreased. However, if the increase in diversity results in a more virulent pathogen replacing a less virulent one, then disease risk will be increased (Strauss et al. 2016). More attention

should be focused on the frequency of positive correlations between coinfections with zoonotic pathogens and human disease prevalence or severity, on the relationship between pathogen diversity and abundance, and on the causal pathways involved.

### DOES GREATER VERTEBRATE DIVERSITY CAUSE GREATER RISK OF ZOOONOTIC EMERGENCE?

Putting all of the evidence together, there is some support for the link between increased diversity of vertebrates and increased total diversity of pathogen species, and for the link between more zoonotic pathogen species and more potential for zoonotic emergence. However, there is no evidence supporting the existence of a correlation between the total number of pathogen species and the number of zoonotic species. Consequently, a causal connection between vertebrate diversity and zoonotic disease emergence based on the proposed underlying component mechanisms is not expected based on current evidence.

One alternative mechanism that could theoretically cause high vertebrate diversity to increase risk of zoonotic emergence involves changes in the host range of pathogens. Potentially, high vertebrate diversity within local communities could cause pathogens to evolve the ability to exploit a larger number of potential hosts (Ricklefs 2010). If pathogens occurring within more diverse vertebrate communities have a larger host range and are therefore more likely to spill over into humans, high diversity and zoonotic emergence could be correlated. To our knowledge, this pathway has not been explored empirically. Such a mechanism would require that the niche breadth of pathogens (the number of hosts infected) increases with increasing host diversity. A positive correlation between host diversity and niche breadth might occur for pathogens, but data on the effects of species diversity on niche breadth in free-living organisms suggest that it is unlikely. For example, niche breadth tends to *decrease* with increasing species diversity in both birds (Belmaker et al. 2011) and mammals (Pagel et al. 1991).

Thus far, we have examined the evidence for an underlying mechanistic pathway connecting vertebrate diversity to zoonotic emergence. Most research on this question, however, has looked

at the net effect of vertebrate diversity, without considering details of the potential causal pathway. For example, in their widely cited paper, Jones et al. (2008) used a global database to quantify risk factors for hundreds of emerging infectious diseases of humans that have arisen since the mid-20th century. Jones et al. (2008) stated that “disease emergence is largely a product of anthropogenic and demographic changes, and is a hidden ‘cost’ of human economic development,” suggesting that human activities that disturb natural ecosystems frequently trigger EIDs. However, they also contend that high native biodiversity is a risk factor, stating “Wildlife host species richness is a significant predictor for the emergence of zoonotic EIDs with a wildlife origin.” Whether anthropogenic environmental changes or native biodiversity has a greater influence on disease emergence is clarified by their Table 1, which shows that the probability of an emergence event (zoonotic disease with wildlife origin) is only about 1% higher in areas with high wildlife diversity than in areas of low diversity (regression coefficient = 0.008–0.013, odds ratio = 1.01). In contrast, the probability of emergence was 75–90% higher in areas of high vs. low human population density, suggesting that wildlife diversity plays at most a very small role.

In another global analysis, Dunn et al. (2010) found that “[human] pathogen richness (number of kinds) is largely explained by the number of birds [sic] and mammal species in a region” and that pathogen prevalence in humans is positively correlated with regional pathogen richness. However, they describe how these correlations could be spurious rather than causal: If “mammal and bird richness directly influences human pathogen richness by serving as alternative hosts... We would expect to see pathogens with bird reservoirs best predicted by bird richness and pathogens with mammal reservoirs best predicted by mammal richness. Instead, mammal richness is the stronger predictor of the richness of pathogens both with non-human mammalian reservoirs and those with non-human [sic] bird reservoirs. Since no biological link is expected between these two variables, the correlation implies that mammal richness captures additional variables important for pathogen diversity rather than causing such patterns.” Thus, they conclude that this correlation might not represent

a causal relationship between vertebrate richness and human pathogen richness or disease.

A more recent global analysis by Murray et al. (2015) has been interpreted as supporting a link between mammalian diversity and human infectious disease. Murray et al. assessed the determinants of biogeographic patterns of 187 human pathogens occurring in 225 countries. They found that human infectious diseases are spatially structured in ways that are “broadly consistent with classic zoogeographic classifications, including regions reminiscent of Nearctic, Neotropical, Ethiopian/African, elements of the Saharo-Arabian, elements of the Mediterranean, Palearctic/Eurasian, Oriental, Australian, and Oceanian.” Exploring extrinsic correlates of the biogeographic patterns of infectious disease assemblages, Murray et al. (2015) identified the similarity of the mammalian assemblages as the most significant positive predictor. Overall, the similarity between pairs of countries in their mammalian species assemblages explained 17.9% of the similarity in their disease assemblages. However, they noted that the correlation between mammal assemblages and disease assemblages occurred for “disease classes that have no contemporary connections to mammals” and argued that the spatial structure of human disease assemblages is “likely governed by the same processes that govern patterns of biodiversity more generally...” They argued that, “Although host species richness or distributions may causally relate to pathogen species richness or distributions in some instances, these correlations do not imply causation.” In other words, Murray et al. (2015) do not assert that the diversity of human diseases is causally related to mammalian biodiversity, but rather that both disease diversity and mammalian diversity respond independently to the historical impacts of local and regional diversification, dispersal, and extinction.

In examining human infectious diseases in which outbreaks have been observed in the Asia-Pacific region, Morand et al. (2014) found that numbers of vector-borne diseases, but not zoonotic diseases, were positively correlated with species richness of birds plus mammals. In other cases, increased diversity of human pathogens with decreasing latitude has been documented (e.g., Guernier et al. 2004, Dunn et al. 2010). To the extent that latitude is a proxy for vertebrate species richness, these analyses would support a link



between host diversity and some metric of human disease. However, Han et al. (2016) compared geographic gradients in the species richness of mammalian hosts for zoonotic pathogens with gradients in the richness of zoonotic pathogens worldwide. Contrary to the expectation that high mammalian diversity leads to high diversity of zoonotic pathogens, Han et al. (2016) found no correlation between zoonotic host diversity and zoonotic pathogen diversity. Although zoonotic host diversity was highest at low latitudes, diversity of zoonotic pathogens was quite evenly distributed from low to high latitudes. They found that mammalian hosts in the subarctic zone harbor more zoonotic pathogens than do mammalian hosts from other regions.

### POSITIVE OR NEGATIVE EFFECTS OF BIODIVERSITY ON INFECTIOUS DISEASE

At the same time that assertions have been made about high biodiversity increasing zoonotic emergence, evidence has been accumulating that high biodiversity reduces the transmission and prevalence of many pathogens (Keesing et al. 2010, Ostfeld and Keesing 2012, Civitello et al. 2015a). On the one hand, then, some have said that biodiversity has a negative effect on human health by increasing the emergence of new pathogens, while others have said that biodiversity generally protects human health by reducing transmission. Is it possible to reconcile these two contrasting views?

Field and laboratory studies of the effects of diversity on transmission or prevalence of infectious disease have proliferated in recent years. A review by Cardinale et al. (2012) demonstrated that the effect of diversity on disease prevalence was significantly negative in 80% of the empirical tests of diversity–disease relationships, supporting the widespread occurrence of the “dilution effect” (Keesing et al. 2006). Of the remaining tests, 12% showed a significantly positive effect of diversity on prevalence (an “amplification effect” [Keesing et al. 2006]), and 8% showed no significant effect. However, relatively few of the studies reviewed by Cardinale et al. (2012) evaluated zoonotic diseases. A meta-analysis by Civitello et al. (2015a) confirmed that the negative relationship between diversity and disease prevalence was indeed widespread and significant for both

zoonotic and non-zoonotic diseases. Civitello et al. (2015b) found no evidence for the hypothesis (Salkeld et al. 2015) that support for the negative diversity–disease relationships arises from a confirmation bias.

In contrast to the strong evidence that biodiversity reduces the transmission and prevalence of existing diseases, the evidence that biodiversity increases zoonotic emergence is weak. As described above, zoonotic pathogens tend to persist in vertebrate species that are common and widespread rather than in the rarer species that occur only in more diverse communities. When a positive correlation between vertebrate diversity and zoonotic emergence is found, either the effect size tends to be quite small (Jones et al. 2008) or the correlation is probably spurious, as often described in the studies themselves (Dunn et al. 2010, Johnson et al. 2015a, Murray et al. 2015).

A key area of agreement between the two schools of thought is that *disturbance* of native biodiversity is thought to increase either risk of transmission or human exposure rates to zoonotic pathogens. Some of the strongest support for a negative relationship between diversity and disease prevalence comes from experimental manipulations of diversity and from relatively small-scale comparisons of communities with different degrees of diversity loss caused by human activities (Ostfeld and Keesing 2012). Similarly, when discussing the determinants of zoonotic emergence, Jones et al. (2008) emphasized the strong impacts of environmental changes caused by human activities and increased human population density resulting from economic development. Murray et al. (2015) suggested that “faunal convergence among regions, which might arise as a result of non-random biodiversity loss of the spread of invasive species, could coincide with convergence in infectious disease assemblages and, in turn, disease risks.” Johnson et al. (2015a) emphasized that pathogen spillover from wildlife to humans has occurred most frequently in highly disturbed habitats (dwellings, agricultural fields) and also via occupational exposure to animals via hunting, laboratory work, veterinary practice, and wildlife management.

Perhaps a general agreement is emerging that human activities can increase disease risk when either (1) biodiversity loss increases transmission rates and prevalence of pathogens in hosts or (2)

changes in land use and behavior increase contact rates with pathogen reservoirs, or both. Although these land use and behavioral changes can be associated with anthropogenic reductions in biodiversity, they can also affect disease risk independently of any impacts on diversity. An important point is that both pathways apply to areas anywhere along a gradient from high to low native diversity. Highly biodiverse areas might pose little risk to humans, even with diverse pathogens present, if human behaviors eliciting exposure (e.g., entry into risky habitat, occupational exposure) are rare or absent.

#### RELEVANCE OF BIODIVERSITY–DISEASE RELATIONSHIPS TO POLICY

More than three decades of intensive research has documented the myriad ways that biodiversity supports ecosystem services necessary for human well-being (Daily and Matson 2008, Cardinale et al. 2012), resulting in the recent establishment of the Intergovernmental Platform on Biodiversity and Ecosystem Services (IPBES). A major function of IPBES is to support environmental policies that conserve biodiversity and its ability to support ecosystem services. Recognition of the importance of biodiversity in reducing pathogen transmission and disease risk has lagged behind its relevance for other ecosystem services such as food and water production, water and air filtration, and nutrient cycling. Consequently, the importance of the relationship between biodiversity and disease for environmental policy has received relatively little attention.

Exploring the policy relevance of the relationship between biodiversity and disease risk requires specifying which aspects of biodiversity can be managed or otherwise affected by environmental policy. In any region, the native biodiversity of both parasitic organisms and their hosts provides a baseline for infectious disease risk by representing the collection of potential and actual zoonotic pathogens that the local human population might encounter. The value of knowing the diversity of this native baseline in predicting disease risk is not well established. What is clear, however, is that this native biodiversity is a result of evolutionary and biogeographic processes occurring over long time periods (Brown et al. 2001, Dunn et al. 2010, Murray et al. 2015).

Most of the research on diversity–disease relationships that is interpreted as suggesting that biodiversity is “bad” for human health compares biogeographic regions that differ in native diversity. Even if it were found to be empirically supported, the suggestion that areas with high native biodiversity pose a greater threat of zoonotic emergence is not likely to be relevant to environmental policy. Modern environmental policies do not affect the level of biodiversity that has evolved within a region, but they can strongly affect the proportion of native biodiversity that remains extant in a location. They can also affect which members of the ecological communities persist or disappear. The consistent evidence demonstrating that high biodiversity reduces disease risk arises largely from local- or regional-scale comparisons, or experimental manipulations, of elements of the native diversity (Keesing et al. 2010, Cardinale et al. 2012, Civitello et al. 2015a). As this research increasingly elucidates the important functional components of biodiversity, the drivers of diversity loss, and the mechanisms linking diversity loss to disease risk, it will become increasingly relevant to environmental policy and management.

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