Comments

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Does biodiversity protect humans against infectious disease? Comment

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Identifying how changes in biodiversity alter infectious disease dynamics is important for both basic science and policy. Biodiversity, broadly conceived, denotes "the variety of life in all its manifestations" (Loreau and Kinne 2010), and encompasses not only species richness, but also phylogenetic diversity, trait and functional distinctions among taxa, and indeed the complexity of community organization, such as food web interactions. Wood et al. (2014) note that an increase in biodiversity can at times amplify disease risk in a focal host species existing across a gradient in biodiversity, and some empirical examples do seem to demonstrate amplification (Young et al. 2013). Keesing et al. (2006) outline several mechanisms that could underlie such amplification, and it is certainly the case that if there is no biodiversity (the "parking lot ecosystem") there will be no zoonotic diseases. Where matters get more interesting, complex, and relevant to conservation is when one examines how disease risk might shift across broad-scale gradients in more realistic scenarios, such as when one compares largely intact natural ecosystems with systems degraded or fragmented by human activity (the focus of this comment).

The recent literature contains a variety of examples of situations in which high biodiversity reduces disease risk, a phenomenon termed the "dilution effect" (Keesing et al. 2006, Civitello et al. 2015). Wood et al. (2014) review a number of cases in which they hypothesize that one might find that instead of a dilution effect, an increase in biodiversity is associated with *increased* disease risk. They postulate in particular (p. 821) that prior work has focused on the developed world, and that infectious disease in developing nations in the tropics often increases with increasing diversity. We respectfully suggest that for many of the systems discussed by Wood et al. (2014), the role of diversity is not yet clear, at least if comparisons are between intact forest and degraded habitats, including in particular in tropical biomes. Such comparisons are particularly pertinent to conservation policy, and would facilitate predictions regarding whether disease risk will increase when forests of high conservation value are degraded or fragmented by human activities.

One consequence of eroding wildlife diversity in fragmented forests is that smaller-bodied hosts, and hosts at lower trophic levels, most famously rodents, can become hyper-abundant either due to increased food availability, or to ecological release following extirpation of competitors or predators (Adler and Levins 1994, Nupp and Swihart 1998, Terborgh et al. 2001, Keesing and Young 2014). The disappearance of top predators (Estes et al. 2011, Ripple et al. 2013), a widespread signature of anthropogenic impacts, releases their prey from top-down regulation and can also lead to an upsurge in infectious disease because of reduced mortality of infected hosts (Packer et al. 2003). Ecological release of prey species can occur across a gradient in body size from small rodents, which can be far more abundant in forest fragments (Nupp and Swihart 1998, Debinski and Holt 2000), to large-bodied ungulates, which can become hyper-abundant in the absence of predation or when feeding on agricultural landscapes (Ripple et al. 2013, Wilmers and Levi 2013). Shifts in competition can also lead to ecological release; McCauley et al. (2008), for instance, report that removal of large herbivores in African savanna permitted surges in the abundance of small-bodied rodents, which sustain pathogen-carrying fleas at higher abundance (see also Young et al. 2014).

Fragmentation of habitats can also lead to the ecological release of hosts through other mechanisms. For example, because of matrix subsidies and mesopredator release, generalist mesocarnivores such as raccoons and opossums often reach much higher densities in fragmented landscapes near agricultural fields and on the periphery of exurban development (Crooks and Soule 1999, Ritchie and Johnson 2009). For example, the most heavily infected hosts for the trematode vector of salmon-poisoning disease (*Nanophytes salmincola*) in Oregon are mesopredator raccoons and skunks

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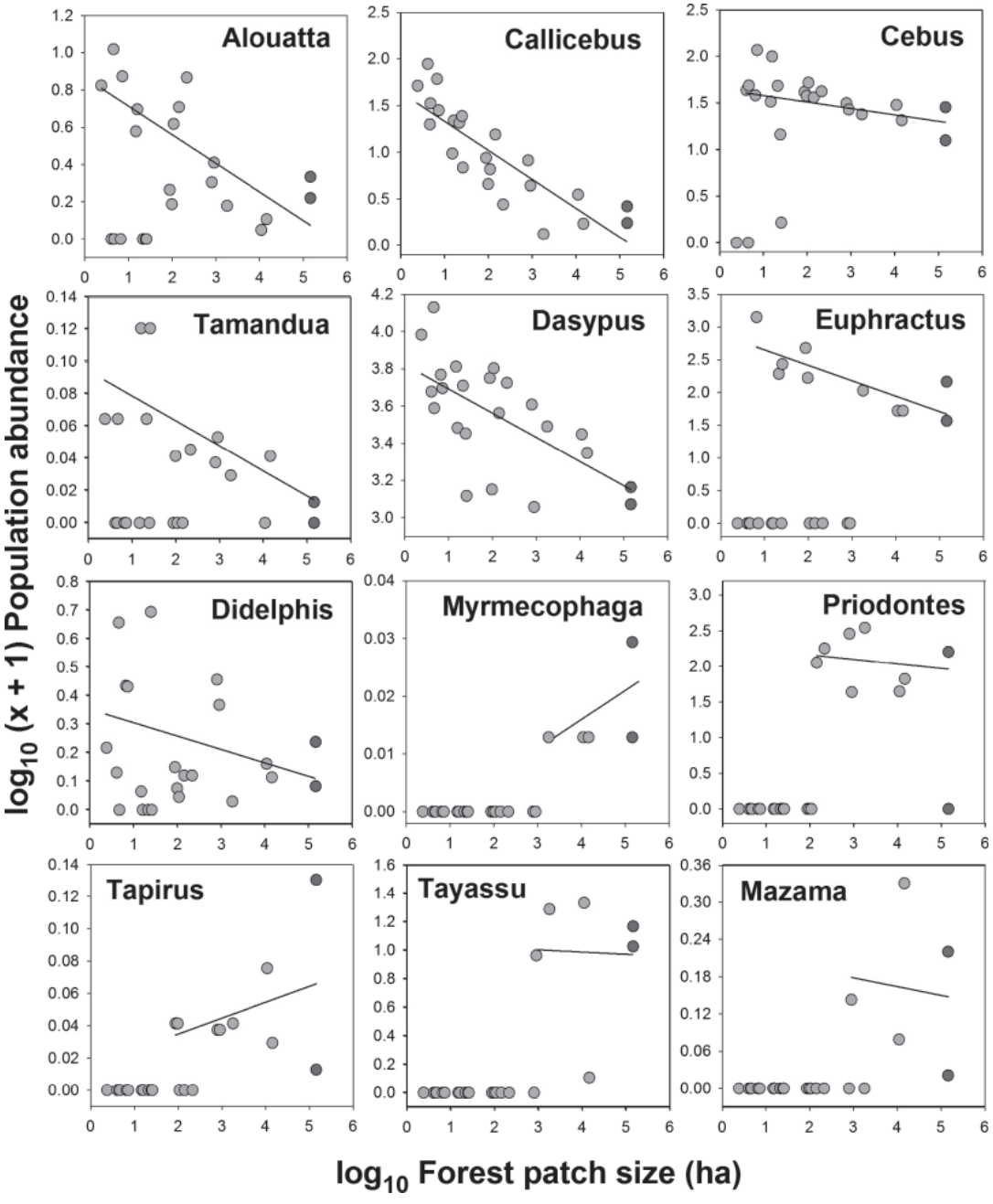

FIG. 1. Relationships between forest-patch area (measured in ha) and mammal population abundance in southern Brazilian Amazonia (from Michalski and Peres 2007). Regression line is fit including only sites where the taxa were present. Many taxa, including howler monkeys (*Alouatta*), titi monkeys (*Callicebus*), capuchin monkeys (*Cebus*), collared anteaters (*Tamandua*), two taxa of armadillos (*Dasypus* and *Euphractus*), and common opossums (*Didelphis*) increased in abundance in small forest patches, although patch-scale absences also occurred. In contrast, large-bodied species such as giant anteaters (*Myrmecophaga*), giant armadillos (*Priodontes*), tapirs (*Tapirus*), white-lipped peccaries (*Tayassu*), and brocket deer (*Mazama*), were absent in small forest fragments, which dominate hyper-fragmented tropical forest landscapes. Light and dark shaded circles indicate forest fragments and continuous forest sites, respectively. Abundances were measured with species-specific approaches including line transect surveys (primates, peccaries, and deer), burrow density estimates (armadillos), and camera trapping rates (anteaters, opossums, and tapirs).

(Schlegel et al. 1968), both of which reach much higher densities near humans (Prange et al. 2003, Gehrt 2004). Wood et al. (2014) state that the risk of contracting salmon-poisoning disease would increase with biodiversity or ecosystem intactness, because the pathogen uses dogs and wild carnivores as hosts. However, this argument does not hold if the most common hosts are mesopredators and human commensals that become much more abundant in degraded landscapes.

Similarly, Wood et al. (2014) suggest that important tropical diseases of humans should increase with biodiversity, in contrast to the predictions of the dilution effect. This conclusion was based on the faulty assumption that hosts for these diseases are more abundant in intact habitats with high biodiversity. Secondary forests, forest fragments, and edge habitats in the tropics have typically lost important avian and mammalian predators (e.g., Harpy Eagle [*Harpia harpyja*], Ornate Hawk Eagle [*Spizaetus ornatus*], jaguar [*Panthera onca*]). These degraded forests also contain fast-growing pioneer plant species with poorly defended palatable leaves (Coley and Barone 1996). As a result, many Neotropical mammals are much more abundant in fragmented and degraded forests than in continuous forests, because they enjoy both a relaxation in predation, and a boost in resources (Chiarello 1999, Michalski and Peres 2007, Canale et al. 2012, Benchimol and Peres 2015). For example, wildlife surveys across a forest fragmentation gradient demonstrate that multiple primate species, armadillos (*Cabassous unicinctus*, *Dasypus novemcinctus*, and *Euphractus sexcinctus*), anteaters (*Tamandua tetradactyla*), opossums (*Didelphis marsupialis*), and sloths (*Bradypus* spp.) are more abundant in smaller forest fragments, whereas large-bodied taxa such as tapirs (*Tapirus terrestris*), giant anteaters (*Myrmecophaga tridactlya*), giant armadillos (*Priodontes maximum*), white-lipped peccaries (*Tayassu pecari*), and brocket deer (*Mazama* spp.) (as well as top predators such as jaguars) are rare or absent (Fig. 1; Michalski and Peres 2007). Shifts in abundance are often evident, even if there is no change in species richness per se (e.g., note patterns across the larger fragments in Fig. 1). Similarly, many small rodent and marsupial taxa in the Neotropics are more abundant in fragmented forests, edge habitats, small islands, and secondary forests than in continuous tracts of relatively undisturbed primary forests (Adler and Levins 1994, Lambert et al. 2006). The disturbance-tolerant vertebrates in Neotropical communities have been previously identified to be important reservoirs of zoonotic diseases. Armadillos, sloths, opossums, and rodents, for example, are thought to be among the most important hosts for the protozoa *Trypanosoma cruzi* and *Leishmania* spp. (Christensen et al. 1982, Roque et al. 2010, Xavier et al. 2012), the pathogens that cause Chagas disease and leishmaniasis, respectively. Hyper-abundance of these mammals in landscapes dominated by small forest fragments may lead to both a higher density and infection prevalence of arthropod vectors (Gottdenker et al. 2012, Xavier et al. 2012). Wood et al. (2014) are obviously correct that hosts need some habitat, but the remaining species that amplify or transmit pathogens are, we suggest, often far more abundant in degraded than in faunally intact landscapes.

Wood et al. (2014) consistently reason that pathogens increase with biodiversity merely because their hosts require forest; they make this claim without consideration of how host species abundance varies across disturbance gradients, or empirical data on the relationship between diversity and the abundance of the reservoir host, or consideration of forests that are differentially impacted by humans. For example, the zoonotic form of *Brugia malayi* is thought to rely on leaf monkeys (*Presbytis* spp.) and macaques (*Macaca* spp.) as important wildlife reservoirs. Wood et al. (2014) claim that these species are associated with intact forest and that therefore *Brugia malayi* is an example of biodiversity increasing disease risk. However, primatological studies have found exactly the opposite pattern for these taxa (Southwick and Cadigan 1972). Of three leaf monkey and two macaque species surveyed in Malaysia, two were most abundant immediately near human habitation, moderately abundant in secondary forest, and rare in primary forest. The remaining three were much more common in secondary forest than primary forest (Southwick and Cadigan 1972). Other primatologists have described many species of macaques as "weedy" due to the extraordinary abundance they achieve near human-dominated habitat and agricultural fields (Richard et al. 1989). We agree that *Brugia malayi* is not prevalent in developed areas because its principal vector, *Mansonia* mosquitoes, breed in wild swampy habitats. However, given the relative rarity of the primate hosts for *Brugia malayi* in intact primary forests, and the abundance of these hosts in disturbed forests, the assumption that this parasite responds positively to biodiversity and intact ecosystems is clearly flawed.

After reviewing the literature concerning every pathogen and parasite that Wood et al. (2014) list as responding positively to biodiversity, we were unable to identify *any* for which there is unambiguous evidence that they increase with biodiversity across a full gradient of disturbance. For example, Wood et al. (2014) assign the trematodes, *Clonorchis* sp., *Opisthorchis viverrini, Fasciola gigantica*, and *Fasciola hepatica,* and the tapeworms, *Echinococcus granulosus* and *Diphyllobothrium latum*, to the group of parasites that increase with biodiversity. But all of these parasites are primarily transmitted by the feces of livestock, humans, and/or domestic animals, and they are associated with poor sanitation and sewage infrastructure that allows human waste into water bodies where fish are consumed (Bonsdorff 1977, Revenga 1993,

Thompson and McManus 2002, Sithithaworn and Haswell-Elkins 2003, Lun et al. 2005, Keiser and Utzinger 2009). Wood et al. (2014) note that the life cycles of *Clonorchis* sp., *O. viverrini,* and *D. latum* are also maintained by wild carnivores in addition to domestic dogs and cats, but even if the sylvatic cycles contribute to human disease, the important wild carnivore hosts may be more abundant in fragmented or agricultural landscapes due to matrix subsidies and mesopredator release (Henke and Bryant 1999, Ritchie and Johnson 2009, Levi and Wilmers 2012).

The designation by Wood et al. (2014) of the waterborne protozoa *Giardia* spp. and *Cryptosporidium parvum* as pathogens that increase with biodiversity is similarly uncertain. There are at least 19 species of *Cryptosporidium* that can be difficult to distinguish morphologically without genetic analysis (Fayer 2010). Before widespread availability of molecular diagnostics, *C. parvum* was named as the default species without genetic confirmation. Most cases of *Cryptosporidium* infection in humans are now known to be due to the cattle genotype and the human genotype, which has now been named *C. hominis* (Morgan-Ryan et al. 2002). Given the association of *Cryptosporidium* cases in humans with genotypes from human and bovine hosts, the assertion by Wood et al. (2014) that the reservoir hosts are more abundant in undisturbed areas than disturbed areas is not supported. Wood et al. (2014) similarly claim that *Giardia* is commonly associated with contaminated water in pristine areas and that possible reservoirs include many mammal species including primates, ungulates, rodents, and carnivores. Aside from the questionable assumption that a wide array of reservoir species equates to a positive response of *Giardia* to biodiversity, of the two assemblages that infect humans (A and B), it should be noted that there are a number of reservoir species associated with agricultural landscapes and domesticated animals, including cattle (assemblage A), cats (assemblage A), and dogs (assemblages A and B). While the *Giardia* genotypes that infect humans have been found in some wildlife, including primates, beavers, and muskrats, there is little evidence to suggest that humans are more likely to become infected with *Giardia* in pristine areas than in degraded landscapes, particularly when most cases are attributed to water contaminated with human waste (Shaw et al. 1977, Olson et al. 2004, Hunter and Thompson 2005).

Aside from our discomfort with Wood et al. (2014)'s specific interpretation in these cases, we also found a number of inconsistencies between the diseases listed as having a positive response to biodiversity in Table 1 of their article and the description of these diseases in the text. For example, Wood et al. (2014) acknowledge in the text that *Leishmania tropica* thrives in urban areas (Jacobson 2003) but go on to list *L. tropica* as a pathogen that responds positively to biodiversity in Table 1. Similarly, Wood et al. (2014) list *Schistosoma mekongi* as a pathogen responding positively to biodiversity. However, they reason that *S. mekongi* reservoir hosts (snails) are more abundant in disturbed habitats with fewer predators than in undisturbed habitats, which directly contradicts what would be considered a positive response of infectious disease risk to an increase in biodiversity.

Given the enormous complexity of linking land use change to vertebrate community structure, and to disease prevalence and dynamics, how then should we determine whether overall species diversity and intact forests and their food web interactions are good for our health? The question of whether or not dilution or amplification effects predominate for particular infectious diseases (or entire suites of pathogens and parasites) across gradients in disturbance is a challenging empirical problem that requires a multifaceted approach, and not an issue that can be entirely resolved by a priori reasoning. The solution we suggest lies with a landscape-scale epidemiology approach (from local patches to entire biogeographical regions) that combines (1) detailed knowledge about hosts for pathogens combined with molecular techniques that ensure that the pathogens being detected are the same as those that infect humans, and (2) comprehensive vertebrate diversity and abundance assessments across a gradient of land use change (as in Michalski and Peres 2007, Fig. 1), all (3) linked to measures of disease risk such as vector abundance and infection prevalence, or infection prevalence in humans. This is a tall order. To be most effective, these observational, correlative approaches should be combined when possible with experimental manipulation of host communities and the development of multispecies host–vector–pathogen models that explicitly include direct and indirect community feedbacks, ideally across gradients that incorporate nearly intact natural ecosystems (viz., those of greatest conservation concern). Experimental communities consisting of random subsets of species can provide useful information about how biodiversity impacts disease risk, but comparisons of patterns across actual communities that vary non-randomly in composition as biodiversity changes seem more pertinent to management and conservation questions. Most modeling efforts in the epidemiological literature have until recently focused on single host–pathogen systems (Lloyd-Smith et al. 2009), but this is beginning to change (e.g., chapters in Ostfeld et al. 2008; Miller and Huppert 2013, Mihaljevic et al. 2014). Conceptual models have begun to elucidate the circumstances in more detail when one might observe a positive versus negative effect of biodiversity on disease risk (Dobson 2004, Joseph et al. 2013), but there is a need for models tailored to the complexities of multispecies

communities, where potential hosts are engaged in predatory and competitive interactions, as are potential vectors, and to examine these interactions across multiple spatial scales. For example, multispecies disease models often ignore the complexity of species interactions in entire food webs and simply assume that increasing host diversity is an additive process such that total vertebrate population density always increases substantially with host diversity (Dobson 2004), when in fact the opposite is often observed when important apex predators or competitors are extirpated, and density compensation emerges in the remnant community (Henke and Bryant 1999, Peres and Dolman 2000, Fedriani et al. 2001, Michalski and Peres 2007). For models to reflect what is known empirically, they cannot ignore species interactions and must carefully consider the complex relationships among diversity, species composition, trophic interactions, and the abundance of reservoir hosts. Indeed, future research may determine that the effects of diversity having to do with top-down effects (as in Packer et al. 2003, Levi et al. 2012, 2015), bottom-up effects of resource availability (Smith 2007, Smith et al. 2015), or competitive interactions (McCauley et al. 2008, Young et al. 2014) are much more important than changes in host diversity or composition, per se. Without conducting comprehensive and coupled comparative, experimental, and theoretical analyses, we cannot make inferences about whether the risks of contracting these pathogens and parasites are likely to increase or decline with biodiversity, or more broadly with generalized degradation in anthropogenically impacted landscapes. The detailed phylogenetic structure of species losses (see Parker et al. 2015), and shifts in patterns of community organization along gradients in such degradation (e.g., the relative importance of top-down predation) are likely to be key considerations. Conservation after all is concerned not merely with merely sustaining lists of species, but with maintaining the integrity of ecological interactions that govern the relative abundance and selective milieus of community members.

Given the catastrophic loss of biodiversity that accompanies human activities across the globe (Ripple et al. 2013, 2015, Dirzo et al. 2014), it is essential that we address when and by what mechanisms this loss will also foster increases in disease transmission. However, our systematic examination of the evidence in Table 1 of Wood et al. (2014) finds that the conclusions drawn from their hypotheses are not warranted. Beyond these particular case studies, it is worth noting that other authors have recently found in careful surveys of the literature increasing evidence, across a wide range of systems, to support the proposition that the conservation of biodiversity can also serve to reduce the transmission of infectious diseases (Civitello et al. 2015; Johnson et al. in press). We conclude by emphasizing the urgent need to embed analyses of infectious disease epidemiology into broader themes in community ecology such as assembly theory and trophic cascades analyses in order to address the increasingly intertwined issues of managing infectious disease risks and conserving biodiversity.

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Does biodiversity protect humans against infectious disease? Reply

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The dilution effect is the sort of idea that everyone wants to be true. If nature protects humans against infectious disease, imagine the implications: nature's value could be tallied in terms of *human suffering avoided*. This makes a potent argument for conservation, convincing even to those who would otherwise be disinclined to support conservation initiatives. The appeal of the dilution effect has been recognized by others: "the desire to make the case for conservation has led to broad claims regarding the benefits of nature conservation for human health" (Bauch et al. 2015). Randolph and Dobson (2012) were among the first to critique these claims, making the case that promotion of conservation to reduce Lyme disease risk, although well intentioned, was flawed. Along with Randolph and Dobson's critique, there have been several calls for a more nuanced scientific assessment of the relationship between biodiversity and disease transmission (Dunn 2010, Salkeld et al. 2013, Wood and Lafferty 2013, Young et al. 2013). In response, supporters of the dilution effect have instead increased the scope of their generalizations with review papers, press releases, and, like Levi et al. (2015), letters. These responses have been successful; it is not uncommon to read papers that repeat the assertion that biodiversity generally interferes with disease transmission and that conservation will therefore generally benefit human health. Here, we explain how Levi et al. (2015) and other, similar commentaries use selective interpretation and shifting definitions to argue for the generality of the dilution effect hypothesis.

Levi et al.'s critique centers on our table of hypotheses for how some parasitic diseases of humans might respond to biodiversity loss (Wood et al. 2014). Feeling that a consistent, systematic evaluation was needed, we started with the approach long used by public health scientists and parasitologists: determine the key hosts and vectors in a life cycle and ask how they are likely to change under different circumstances. The circumstances of interest to us were land-use changes that result in biodiversity loss in areas surrounding human communities. We applied this basic logic to the epidemiology of the 69 most important parasites of humans. This exercise showed that, depending on the parasite species, there were hypothetical positive, negative, and neutral associations between biodiversity and parasite transmission. Although we made hypotheses about the overall associations, we indicated that, for any given zoonotic disease agent, the actual shape of this relationship may be complex and dependent on the sensitivities of the hosts and vectors (Fig. 1). We emphasized in our paper that Table 1 contains hypotheses, not conclusions: "Because the biodiversity–disease relationship is untested for many human disease agents, our tabulation is only a starting point for investigating the generality of the dilution effect. However, it provides a systematic, transparent, and reproducible set of predictions that can be a common foundation for discussion" (Wood et al. 2014: Table 1).

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