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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, appealing 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 (**Figure 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). We presented the assumptions we used to develop Table 1 and invited critique, knowing that others might generate different hypotheses for some parasite species.

Accepting that invitation, Levi et al. point out possible exceptions to some of our predictions. Their critiques can be summed up as follows. In some cases, for host groups that we assumed would be negatively impacted by human actions (e.g., primates, carnivores), there can be exceptions where a subset of host species respond positively to land-use change. We agree that it is the response of individual host species, not biodiversity *per se*, that matters. For instance, while some rodent species decrease in abundance around human settlements, other rodent species increase (Young et al. 2015). In other cases, Levi et al. conflate our focus on biodiversity adjacent to human settlements (where we assume biodiversity conservation is most relevant) with transmission that occurs within human settlements. For cryptosporidiosis, giardiasis, food-borne trematodiases, and echinococcosis, most human cases arise from infectious stages passed by other humans or domestic animals, processes that are not relevant to wild biodiversity, but which Levi *et al.* associate with low biodiversity. In these cases, increasing biodiversity (e.g., increasing the number of wild vertebrate species surrounding human settlements) will have no dampening effect on transmission, and – if anything – will contribute to an increase in transmission risk if the added species can also serve as hosts for these parasites. Although reducing human and domestic animal density may well reduce disease risk, human population control is beyond the purview of conservation biologists, making this critique irrelevant to our paper.

Levi et al. only take exception to the parts of our table that do not support the dilution effect. This selectivity is so comprehensive that they cannot find a single parasite that might decline with biodiversity loss. They support this narrow view by citing other selective studies – like Civitello et al. (2015), whose meta-analysis demonstrates that there are many reports of the dilution effect, but makes no attempt to account for publication bias against null results (the "file drawer problem") or against findings of amplification, a bias which could arise if ecologists choose to pursue lines of research likely to yield evidence for the value of biodiversity. To compound this selectivity, Levi et al. ignore or minimize studies that contradict their view (e.g., Salkeld et al. 2013, Young et al. 2013, Valle and Clark 2013). For example, new evidence suggests that contact with forest increases risk for the world's most important parasitic disease, malaria. Malaria transmission in the Brazilian Amazon is high in protected areas that allow people access to forest, but low around protected areas where people are prevented from entering (Bauch et al. 2015). Ignoring this study and the many others like it makes it impossible for Levi et al. to provide a balanced characterization of the relationship between biodiversity and disease.

Levi et al.'s Comment is based on shifting definitions. By this we mean defining a host species as "weedy" when it increases disease transmission, and then redefining that host as an integral component of biodiversity in cases where it impairs disease transmission. As an example, take the way in which Levi et al. define and re-define the role of raccoons in disease transmission. Wood et al. (2014) had argued that carnivores, like bears, that are hosts for salmon-poisoning disease would decline with increasing human disturbance. Levi et al. contend that forest fragmentation would increase the abundance of raccoons, which also host the parasite. We agree that whether raccoons outweigh other carnivores in their importance as reservoir hosts is an open question. What makes this an example of shifting definitions is that raccoons have also been claimed to dilute Lyme disease transmission (e.g., Schmidt and Ostfeld 2001, LoGiudice et al. 2003). To Levi et al., whether raccoons are beneficiaries or victims of human impacts seems to depend on which option best supports the dilution hypothesis. The same strategy is used when Levi *et al.* consider the outcomes of forest fragmentation, which they argue may lead to:

- "Smaller-bodies hosts, and hosts at lower trophic levels, most famously rodents" becoming "hyper-abundant."

- "Mesopredator release" (and thus, we presume, declines in rodent abundance).
- Declines in large-bodied ungulates, which might facilitate "surges in the abundance of small-bodied rodents" via release of competition between these two herbivore groups.
- Top predator decline, which in turn leads to increases in "large-bodied ungulates, which can become hyper-abundant in the absence in predation" (and which, as the authors themselves have demonstrated, would likely lead to decreases in smaller bodied animals, including rodents).

We agree that such changes in community composition can follow forest fragmentation (and other types of disturbance) and that these changes might affect disease transmission. However, this list includes *multiple opposing predictions.* For instance, increases in mesopredators would probably increase diseases carried by mesopredators, but reduce diseases carried by their prey (some rodents). Decreases in large predators will presumably increase disease carried by ungulates, but decrease diseases carried by rodents, which compete with ungulates. Only by conveniently defining "disease" as "rodent-borne disease" in systems where rodents increase, and "mesopredator-borne disease" in systems where mesopredators increase, can Levi *et al.* make a case for the generality of the dilution effect.

A general prediction about biodiversity is a blunt instrument for understanding the ecology of infectious disease. It is far more relevant to consider the ecologies of important hosts and vectors than it is to construct general theory about diffuse concepts such as biodiversity. This is because there are many ways to measure biodiversity, allowing one to shift its meaning to fit a pre-conceived relationship with disease. As an example, consider **Figure 2**, in which we plot three different metrics of "biodiversity" against three different metrics of "disease", where each metric is a vector of 10 randomly selected numbers. This simple exercise demonstrates that – with enough measures of diversity (e.g., species richness, forest fragmentation, proportion of focal hosts in the community) and enough measures of disease (e.g., prevalence or density of infected hosts or vectors for any of a variety of disease agents across any of a variety of host and vector species) – a significant relationship will arise in some combinations just from random chance. If one still wants to relate diversity to disease, it is essential to start with consistent, operational,

and theory-based metrics for both diversity and disease before beginning a study and before data analysis begins – instead of using shifting definitions to ensure a desired result.

Others have intuited that, for proponents of the dilution effect, use of selective interpretation and shifting definitions stems from a desire to promote biodiversity conservation (Randolph and Dobson 2012; Bausch et al. 2015). We've now seen a bias favoring the dilution hypothesis creep into every step of the scientific process – from the choice of research topics, to the interpretation of data, selection of data for publication, peer review, and promotion of results. The dilution effect is an appealing idea, and several of us have published data supporting it for particular contexts, but because human health and conservation are important challenges, the dilution effect deserves scrutiny – not protection.

Grand theories about the benefits of biodiversity may promote conservation, but human health doesn't have to be a mere pretense for protecting nature. There are certain contexts in which conservation action has predictable negative effects on particular disease groups. Several of us have published data showing how some types of conservation can reduce disease, including recent work in Africa that links ecological restoration to reductions in human schistosomiasis (Sokolow et al. 2015). Our goal as disease ecologists is to identify the circumstances in which conservation works as a disease control option. Over-generalizations impede this goal.

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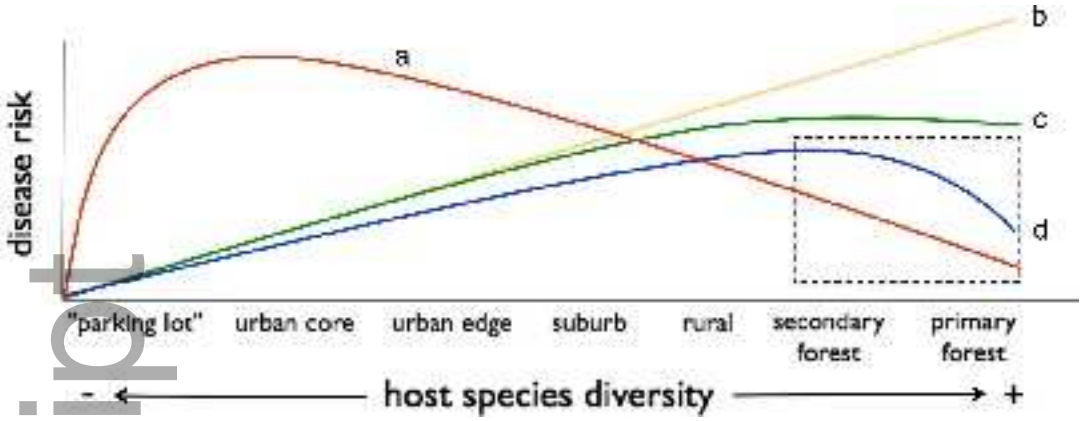
FIGURE 1. Theoretical models for the effect of biodiversity loss / land-use type on disease risk. On average, there should be zero disease risk from zoonotic diseases where host species diversity = 0 (the "parking lot ecosystem"). Possible relationships include: (a) dilution effect, (b) amplification effect, (c) amplification effect that saturates at high levels of biodiversity, and (d) amplification effect that shifts to dilution at high levels of biodiversity (suggested for Lyme disease; Wood and Lafferty 2013). The dashed box indicates how a selective frame of reference (i.e., choosing the right scenario and the right part of the relationship) can be used to assert a dilution effect when it exists (red line), or even when disease risk exhibits a net increase across levels of biodiversity loss / land-use type (blue line). Were such curves to be empirically estimated for disease agents in "real life", they would probably not be so neat; some parasites may have hosts that are negatively impacted by one land-use type but not another, making these lines irregular. This model assumes that host biodiversity and land-use type are linearly related (see x-axis labels), but this assumption is likely to be violated.

FIGURE 2. Relationships between biodiversity and disease for three different metrics of diversity and three different metrics of disease, where each metric is a vector of 10 randomly selected numbers. Lines of best fit are indicated for combinations where the relationship between diversity and disease was significant ($R^2 = 0.69$, $t_7 = -3.92$, $p = 0058$ for disease measure 1 ~ diversity measure 1; $R^2 = 0.54$, $t_7 = -2.86$, $p = 0.0244$ for disease measure 2 ~ diversity measure 2). This simple exercise illustrates that – when the most-appropriate metrics for diversity and disease are not defined *a priori* – significant results can be obtained by selective choice of metrics. Although two of the regressions here show the dilution effect, the amplification effect is an equally likely outcome.



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