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Conservation of biodiversity as a strategy for improving human health and well-being

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The Earth's ecosystems have been altered by anthropogenic processes, including land use, harvesting populations, species introductions and climate change. These anthropogenic processes greatly alter plant and animal communities, thereby changing transmission of the zoonotic pathogens they carry. Biodiversity conservation may be a potential win–win strategy for maintaining ecosystem health and protecting public health, yet the causal evidence to support this strategy is limited. Evaluating conservation as a viable public health intervention requires answering four questions: (i) Is there a general and causal relationship between biodiversity and pathogen transmission, and if so, which direction is it in? (ii) Does increased pathogen diversity with increased host biodiversity result in an increase in total disease burden? (iii) Do the net benefits of biodiversity conservation to human well-being outweigh the benefits that biodiversity-degrading activities, such as agriculture and resource utilization, provide? (iv) Are biodiversity conservation interventions cost-effective when compared to other options employed in standard public health approaches? Here, we summarize current knowledge on biodiversity–zoonotic disease relationships and outline a research plan to address the gaps in our understanding for each of these four questions. Developing practical and self-sustaining biodiversity conservation interventions will require significant investment in disease ecology research to determine when and where they will be effective.

This article is part of the themed issue 'Conservation, biodiversity and infectious disease: scientific evidence and policy implications'.

1. Introduction

The need to feed, clothe and house humanity has altered most of the Earth's ecosystems [1]. The dominant anthropogenic processes include the conversion of natural landscapes for agriculture and urbanization, the accidental or purposeful introduction of non-native species, and direct harvesting of wild populations (figure 1). These activities have greatly altered the composition of biotic communities [3] and they sometimes (but not always) reduce local diversity [3,4]. Human activities have substantially impacted ecosystems, including changes in vegetation structure and microclimates, nutrient cycling, water purification and the emergence of infectious diseases [5,6].

The effects of anthropogenic processes on the transmission of infectious diseases have gained substantial attention in the past decade due to the simultaneous erosion of biodiversity [4] and the increase in emerging disease events [7]. If these two processes are causally linked, then conservation of biodiversity might benefit humans by reducing zoonotic disease risk [8]. However, questions have been raised about the causality and generality of relationships between biodiversity and zoonotic disease risk [9–11]. A fundamental question is whether conservation interventions will increase overall human well-being, including

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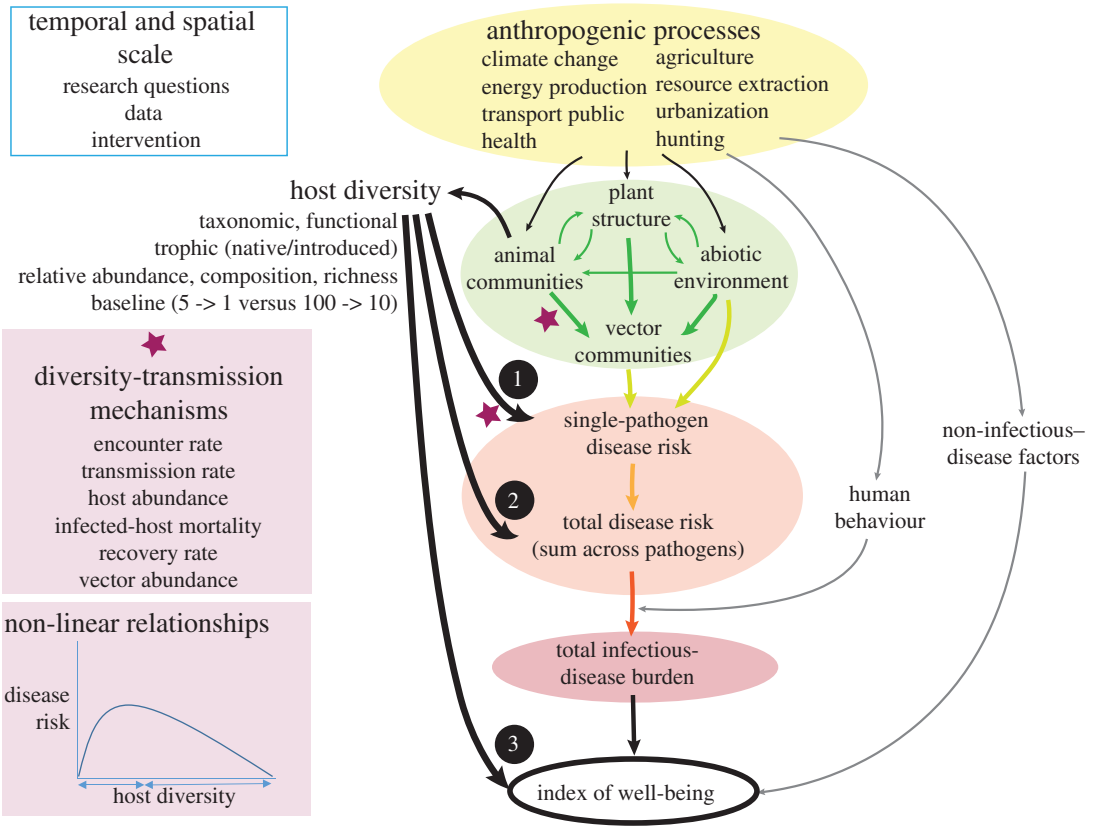

Figure 1. A framework for developing biodiversity conservation interventions to improve human well-being by reducing the total infectious-disease burden. The causality and direction of the effect (positive or negative) of the three numbered black arrows determine the impact of biodiversity conservation interventions on well-being, and are the topics of sections 2–4 in the text. Anthropogenic processes influence biotic and abiotic factors, human behaviour and non-infectious–disease factors. Host diversity for zoonotic pathogens can be quantified in many different ways and is relative, such that the baseline for comparisons is key. Beige boxes illustrate six proposed potential mechanisms by which host diversity can influence transmission [2], and an example of a nonlinear relationship between disease risk and host diversity. Stars indicate possible linkages between host diversity and disease risk. The blue box indicates that spatial and temporal scales are important at all stages from research to intervention.

impacts on the total burden of infectious diseases, as well as other effects on physical, mental and societal well-being.

Determining the effectiveness of biodiversity conservation as a public health strategy requires addressing four questions about the link between biodiversity and the transmission of pathogens, within a context of limited resources and economic trade-offs. First, is there a general and causal relationship between host biodiversity and disease risk, and which direction is it in? Second, if this link is causal and negative for the majority of individual pathogens, does the increased diversity of pathogens that are present in more diverse host communities result in a net total increase or decrease in the burden of infectious disease? Third, is the net benefit of biodiversity conservation to human well-being greater than the benefits provided by diversity-degrading anthropogenic process (e.g. agricultural land-use change or harvesting a species)? Fourth, are biodiversity conservation interventions feasible and cost-effective when compared to other options employed in standard public health approaches, such as vaccination or treatment?

In assessing biodiversity conservation interventions, we consider the two most commonly proposed interventions to increase biodiversity—reintroducing species, and habitat conservation, restoration or alteration. Habitat modification can sometimes target specific species (or suites of species) and lead to both increases and decreases in of different hosts. Decreases in abundant species can lead to increases in diversity metrics that include community evenness.

Here, we consider evidence available to answer each of these four questions, and we outline steps needed to bridge the gap to assess the viability of biodiversity conservation as a public health strategy. Most previous work has addressed the first question—whether host biodiversity is correlated with disease risk—and thus our treatment of the four questions is skewed towards this topic. Nonetheless, we discuss all four questions in our review because all are fundamentally important, despite the paucity of evidence on which to base firm conclusions for the latter three [12].

2. Disentangling the causal effects of biodiversity on disease from other effects of anthropogenic change

Urbanization and agricultural expansion have greatly changed biological communities [3], reducing vertebrate species diversity in many habitats [4,13]. While land use influences disease in myriad ways [14], researchers have focused on determining whether erosion of biodiversity will lead to an increase or decrease in disease risk—hypotheses called the 'dilution effect', and 'amplification effect', respectively [8,15,16]. The name dilution effect originates from the mechanism proposed for the Lyme disease system; the addition of less competent host species (birds, raccoons, opossums and skunks) to communities consisting of highly competent hosts (white-footed

Table 1. Summary of different types of evidence for host biodiversity–disease risk relationships in published studies of zoonotic pathogens. The second column summarizes the correlational analyses presented in the paper between diversity and disease risk. The colour of cells in this column indicates the sign of the significant coefficient(s) between diversity and disease risk as reported in the cited study, with green indicating only negative significant relationships, red indicating only positive significant relationships and yellow indicating either both positive and negative significant coefficients, no significant correlations or uncertain correlations (see notes). The text in each cell in this column indicates the level of significance (not significant (n.s.) = $p > 0.05$; *$0.05 > p > 0.01$; **$p < 0.01$) and sign of the coefficient ($\pm$ = disease risk decreased/increased with host diversity) for all the analyses reported in the paper. Columns 3–6 indicate the presence of data for each of the four types of evidence described in the main text in support of mechanistic relationships between diversity and disease risk. Pathogen abbreviations: SNV, Sin Nombre virus; a hantavirus; WNV, West Nile virus.

pathogen/disease	diversity–disease risk correlation	community assembly	host competence	diversity–host/vector abundance	diversity–disease vector contact	ref.
anaplasmosis	2 (+ *,− *)					[23]
Andes virus	5 (− *), 11 (n.s.)					[24]
hantavirus	1 (? **)[a]					[25]
Lyme disease	1(+ *), 3 (n.s.)					[26]
Lyme disease	1 (n.s.)					[27]
SNV	1(− **)					[28]
SNV	1(− **)					[29]
SNV	1(− *)					[30]
SNV	2(− *,**), 4 (n.s.)					[31]
SNV	1 (n.s.)					[32]
WNV	8 (− *−**), 4 (n.s.)					[33]
WNV	2 (− *,**) 2 (n.s.)					[34]
WNV	2 (n.s.)					[35]
leptospirosis	1 (− **)					[36]
Chagas disease	?[b]					[37]
gut bacteria[c]	1 (− *)					[38]
key	negative	data present				
	mixed/n.s.	partial data				
	positive	no data				

[a]The experiment in this study removed species from plots and measured the change in host infection prevalence, but did not indicate how communities naturally assemble in this region, so the direction of the relationship between naturally occurring diversity and disease risk is difficult to interpret.
[b]The statistical analysis was not reported in the paper.
[c]This study correlated combined prevalence of four groups of pathogens: *Escherichia coli*, *Giardia* spp., *Salmonella* spp. and *Cryptosporidium* spp.

mice, eastern chipmunks and shrews) is hypothesized to 'dilute' the transmission of the Lyme disease bacterium to larval ticks by these species, thereby reducing infection prevalence in nymph ticks [17,18].

A suite of mechanisms has been proposed to explain how diversity (usually of hosts, but possibly also of vectors or microbiota, and measured in a variety of ways; figure 1) could influence transmission and result in either a dilution or amplification effect. These mechanisms include alterations in host and vector contact rates, abundance, vital rates and infectiousness (figure 1, beige box with star) [2]. For each mechanism, either dilution or amplification can occur as host composition and diversity changes. Anthropogenically altered habitats tend to have fewer large predators, smaller-bodied species, and more introduced species than undisturbed habitats [19,20]. The traits of species, including their contact rates, their infectiousness for each pathogen, and their impact on other host and vector species will determine whether they are amplification or dilution hosts [21,22]. Determining whether increasing biodiversity will result in dilution or amplification requires data from multiple pathogen systems, and multiple studies of the same pathogen system to understand how geographical and ecological variation influence transmission.

A recent meta-analysis of 61 experimental and observational studies of the dilution effect in plants and animals found a strong overall negative correlation between diversity and disease risk, and negative (but not necessarily significant) coefficients in 182 of 202 effects [16]. Most studies focused on a single pathogen, and correlated host diversity (usually species richness) with some measure of disease risk—infection prevalence in vectors or the density of infected vectors for vector-borne diseases, and the density or prevalence of infected hosts of a focal species for directly transmitted pathogens. We examined mechanistic evidence for biodiversity influencing disease risk in the 16 studies from this meta-analysis that examined the effect of host diversity on zoonotic pathogens to outline future research needs (table 1). We focused on zoonotic pathogens, since these are the pathogens with direct impacts on human health. Although the majority of comparisons in these studies were non-significant and there were

sometimes mixed results for the same pathogen, the majority of studies found at least one significant negative correlation (table 1). Thus, overall, the available data suggest that there is some correlational support in many zoonotic systems for a dilution effect, and that some species or species groups are more important than others in transmission.

The next step required to develop biodiversity conservation as a public health intervention is to determine whether correlational relationships between host biodiversity and pathogen transmission are causal and to determine the mechanism and host species that are most important in reducing disease risk. Targeted biodiversity conservation interventions may be more effective, especially if they are guided by a mechanistic understanding of transmission ecology. For example, some conservation actions (e.g. habitat preservation or restoration) may reduce host species diversity by reducing human commensals or invasive species, both of which sometimes make up significant components of host diversity in human-dominated areas, and can act as important reservoirs of disease [19]. In addition, although many previous studies of the dilution effect used relatively simple measures of diversity (e.g. species richness), the impact of biodiversity conservation interventions is likely to differ depending on the identity and relative abundance of species in the community [26].

Theory [2,21,39,40] suggests that determining causal relationships between naturally occurring patterns of host diversity on pathogen transmission requires three types of data:

(1) Predictable patterns of community assembly across naturally occurring diversity gradients including richness, composition and abundance.
(2) Estimates of host competence, including susceptibility to infection, and infectiousness (including magnitude and duration).
(3) Data showing the effect of 'diluting' species on: (i) 'amplifying' hosts or vector abundance, (ii) contact rates among amplifying hosts, (iii) contact rates between vectors and amplifying hosts, or (iv) changes in amplifying host susceptibility to infection or infectiousness (figure 1).

Establishing a causal link between diversity and transmission requires integrating these three datasets into a mechanistic multi-host mathematical model of pathogen transmission. Models can be used to quantify the magnitude and direction of the effect of natural variation in species diversity on transmission, independent of habitat change effects, or other factors that change across the natural gradient. A combination of experimental approaches, which reduce issues with confounded variables, and observational studies, which have less experimentally introduced artefacts, larger spatial scale, sometimes larger sample sizes and less restricted scopes of inference is often most effective. Theory shows that diversity can reduce transmission through some mechanisms while increasing it through others [2,21,39,40], and both theory and the empirical results collected thus far indicate that some species play a much larger role than others [18,30,41]. An integrative approach is needed to determine the net effects of diversity, and the role of each species in transmission.

Some studies on zoonotic pathogens have already collected one or more of these three types of data, but much more research is needed to show that observed correlations are causal and to identify the mechanisms by which diversity is influencing disease risk (table 1). Many of these data gaps are due, in part, to the greater difficulty of quantifying species interactions among hosts (or hosts and vectors), especially in comparison to measuring correlations between estimates of species richness or diversity with disease risk. However, additional research to fill these gaps is needed, because correlations between disease risk and diversity can be misleading in both directions; they can provide spurious positive evidence of causal relationships (in either direction), and they can hide causal relationships between host diversity and transmission. This is because many factors other than host species diversity influence transmission (e.g. climate, habitat and vector abundance) and can mask dilution or amplification effects, resulting in non-significant correlations between host diversity and disease. Similarly, covariance between host diversity and other factors can also create spurious significant correlations.

The likelihood that there will be confounding factors in observational field studies is very high, because the same disturbance or habitat gradient that creates gradients in host species diversity also alters many other aspects of transmission. For example, for vector-borne disease such as Lyme disease or West Nile encephalitis, increasing forest fragmentation can simultaneously increase vector abundance [42,43] and decrease host diversity, and the former may be confused with the latter as the cause of increased disease risk. Habitat differences can also create spurious correlations for directly transmitted pathogens. For example, observational field studies of hantaviruses have found correlations between host diversity and disease risk, measured as the density of infected small mammal hosts, and have hypothesized that these patterns result from increased competition [31] or predation [32], or reduced contact rates due to behavioural interference [30] as biodiversity increases. However, differences in habitat (rather than host diversity) among sites in these studies could also result in differences in host abundance and behaviour.

The issue of confounding variables is especially important when disease risk or incidence data come from much larger spatial scales than the scale of transmission. Variability in transmission for most zoonotic pathogens exists at the scale of tens of metres or hectares [44,45]. By contrast, many studies that use human incidence data often analyse variation among counties [33,34], because this is the spatial unit for which these data are often publicly available. In addition, analyses at smaller spatial scales are challenging for low-incidence pathogens like WNV, Lyme disease and hantaviruses, because the majority of smaller spatial units have zero cases. Aggregation of human incidence and diversity data over large spatial scales introduces many additional confounding variables and noise including variation in human behaviour influencing exposure to the pathogen, climate and habitat differences [10,46,47]. For example, correlations between US statewide Lyme disease incidence and host species diversity data [17] are difficult to interpret because of confounding variables associated with large-scale variation in climate and tick questing behaviour. Latitudinal gradients in climate alter the phenology of nymphal and larval-stage ticks, which can decrease infection of larval ticks in more southern states (where host diversity was also higher), and latitudinal differences in questing behaviour have been shown to reduce human–tick encounters in more southern states [48].

Local-scale studies and more mechanistic data help avoid many of these confounding variables. This is illustrated by a recent study that collected both mechanistic and disease-risk data on a directly transmitted fungal pathogen in *Daphnia* and showed that an apparent disease risk–diversity correlation was in fact spurious, and stemmed from differences in habitat structure [49]. If habitat structure, rather than host diversity, is the causal variable influencing pathogen transmission, then interventions that focus on habitat structure will be more effective than those that alter host diversity without changing the important aspects of habitat structure.

It is important to note that data gaps in studies of the dilution effect (table 1) do not mean a dilution effect is not operating in these systems. The data gaps simply indicate that causal inference is not warranted. If correlations are spurious and interventions increase host diversity but not the actual causal factor, the intervention will fail to reduce disease risk. Similarly, there may be missed opportunities in the systems where non-significant correlations have been found if dilution effects are being masked by other factors.

An additional challenge that arises in assessing the causality and generality of relationships between diversity and disease risk is that the relationship is usually nonlinear and may be unimodal with a peak at some intermediate level of diversity for most diseases (figure 1, lower left box). When habitats are so degraded that few host species remain (such as in highly urbanized cities), the likelihood that some pathogens will persist is often low. For example, the risk of Lyme disease in downtown New York City is near zero because there are no deer, little leaf litter and therefore few ticks. To reduce disease risk by conserving or restoring biodiversity, it is obviously important to determine on which side of the peak a given location falls before attempting an intervention.

3. Determining the net impact of increased biodiversity on total disease burden: accounting for multiple pathogens and human behaviour

Most individual studies of the dilution effect examine only a single pathogen [10,16], making it impossible to predict the total or net patterns of disease risk and diversity. This is a major shortcoming in our understanding of biodiversity–disease processes, and raises an important question: how would conservation interventions that increase host biodiversity affect total disease burden in humans (figure 1)? Answering this question would require measuring the impact of diversity on disease risk for all the important zoonotic pathogens in the study area, and converting them to a common currency (e.g. disability-adjusted life years [50,51]). There is strong evidence that pathogen richness increases with host species richness [52], and the vast majority of hosts are infected with many different pathogens [53]. However, interactions between pathogens can be negative, positive or undetectable [54–56], and both pathogen and host dynamics occur within a tangled bank of species interactions [57]. A rigorous study of the net impact of host diversity on total disease burden has never been done for any habitat or location. It is obviously a daunting task, but is required to assess the net impact of interventions focused on conserving biodiversity.

Broad-scale patterns suggest that the number or burden of infectious diseases is higher in tropical than in temperate regions, and decreases with wealth, but shows mixed patterns with biodiversity [58–60]. However, the spatial scale of these studies (countries) is too large to determine whether these correlations are causal. Further, most conservation interventions are likely to take place within countries, at much smaller scales, making these patterns of limited value for guiding public health interventions.

An important step in determining the impact of biodiversity conservation is to determine how an intervention might affect human behaviour and contact with wildlife or vectors. Variation in human behaviour determines the relationship between disease risk and incidence (figure 1) [61] and can sometimes overwhelm differences in disease risk. For example, while the density of infected ticks carrying the Lyme disease bacteria, *Borrelia burgdorferi*, is higher in forested areas than on grass or forest edges [45], human incidence often increases with edge habitat [62], probably due to higher human use of forest edges than intact forest. Similarly, some studies have found opposing trends; forest fragmentation was correlated with increased disease risk (the density of nymphal ticks and prevalence of the Lyme disease bacteria) but decreased human incidence [63]. If a biodiversity conservation intervention entailed creating a large forested park in a residential area, it would be important to ensure that use of the park did not increase exposure to vectors or infected wildlife so much that other benefits of the park were outweighed by increased exposure to ticks or infected wildlife and resultant disease burden.

Our understanding of the overall impact of biodiversity conservation interventions could be greatly improved by studying changes in multiple infectious diseases as large-scale development projects are implemented, as has been done with dams and several water-borne diseases [64,65]. These projects represent landscape-scale experiments that can provide unparalleled insight into the impact of conservation interventions. In addition, developing knowledge of net impacts on public health in collaboration with development organizations will maximize the impact of disease ecology research on policy decisions [66].

4. Balancing biodiversity conservation interventions with benefits of diversity-degrading anthropogenic processes in contributing to human well-being

Biodiversity conservation interventions may reduce or increase the total burden of infectious diseases, and they also have other impacts on human health and well-being [12,64]. Well-being includes physical, mental and social aspects and is not merely the absence of disease or infirmity [67]. Increasing biodiversity through habitat conservation or restoration may have positive effects on several aspects of well-being, including both physical and mental health benefits associated with spending time in nature [68,69] (e.g. decreased medical recovery time [70]).

However, while conservation of land or species may provide some increased ecosystem services [5], it may also reduce the food or resources that are obtained from agriculture or hunting [71,72]. A full assessment of the impact of biodiversity conservation interventions must include both effects on

infectious diseases and effects on other aspects that contribute to human well-being, such as nutrition, non-infectious diseases and economic wealth, and weighing them in a common currency [12,71,73]. For example, disease risk may increase with forest clearing for agriculture [46,72], but this may be outweighed by benefits of the food grown on that land [74]. More inclusive assessments could use more inclusive indices of human well-being such as those used in the United Nations' Human Development Index [75] and the World Happiness Report [76], but these efforts have just begun.

5. Targeted approaches and cost-effectiveness of biodiversity conservation interventions and other strategies

If biodiversity conservation can be shown to be causally effective in reducing disease burden in a region and to have a net positive impact on human well-being, then the next question is whether such conservation actions are feasible and cost-effective at the appropriate scale. A recent review suggested that anthropogenic land use often correlates with increased disease incidence, transmission or risk for individual diseases [14]. One might therefore hypothesize that protecting land from anthropogenic disturbance might reduce overall disease risk. A recent study examined how three types of diseases correlated with the size of two types of protected natural areas (strictly protected areas and sustainable-use protected areas) and other land-use activities (roads and mining), in municipalities in the Brazilian Amazon, while controlling for socioeconomic and climatic variables [46]. Significant negative correlations existed between strictly protected area size and malaria incidence, acute respiratory infection and diarrhoeal disease in Brazilian municipalities, supporting this biodiversity conservation intervention as a strategy to benefit public health. However, malaria incidence increased with the size of sustainable-use protected areas and this effect was twice as large as the reduction in malaria incidence with strictly protected areas. This pattern of increasing malaria incidence with the size of sustainable-use protected areas is consistent with another recent study that found increased malaria incidence with forest cover around cities in Brazil [77]. The contradictory correlations with different types of protected areas point to a key challenge in developing biodiversity conservation interventions as public health strategies—our understanding of the mechanisms driving disease incidence patterns is still too poor to predict the outcome of broad-scale land-use interventions on even the most well-studied diseases like malaria [46,78].

These challenges and the mixed patterns evident in previous studies of zoonotic pathogens (table 1) suggest that more targeted strategies might be useful. This is supported by many studies of multi-host pathogens that show that one or two species appear to play the dominant role in transmission [18,41,79,80]. Interventions targeting these focal species might be more effective and more feasible in mitigating disease than conserving biodiversity generally or protecting land, and they might also have fewer detrimental secondary effects.

Designing effective targeted interventions requires identifying the key hosts in transmission and developing interventions that target those hosts. This requires a deep understanding of both disease and population ecology, which is a substantial challenge. For example, for Lyme disease, white-footed mice, *Peromyscus leucopus*, were initially thought to be the dominant host for infecting larval ticks with the Lyme bacteria *Borrelia burdorferi*, and were targeted with a vaccine in an experimental study [81]. Unfortunately, the impact was only modest with significant reductions in prevalence in nymphal ticks at three of six sites and 19% and 25% reductions overall in the 2 years of the study. Subsequent work provided a possible explanation for the smaller than expected impact; other small-mammal hosts, including chipmunks and shrews, infect at least as many ticks as mice, especially in years without oak masting, when mice are less abundant [18,82,83]. In addition, reservoir hosts for Lyme disease vary substantially across different habitats and continents [44]. This variability suggests that interventions, such as restoring key predators [84], that reduce the abundance of multiple reservoir hosts might be more effective than simply targeting mice.

Species reintroductions to reduce pathogen transmission are intriguing, because they potentially offer self-sustaining impacts. For example, a recently suggested intervention to suppress Lyme disease incidence was the reintroduction of wolves to reduce coyote predation on foxes, with the aim to increase predation on small-mammal host populations [84]. This intervention is based on recent work in the Midwestern USA that showed that higher wolf abundance was correlated with lower coyote density which in turn was correlated with higher fox abundance [85]. Reintroducing wolves was hypothesized to increase predation on small mammals, because foxes can occur at higher densities than coyotes and have a more carnivorous diet focused on small mammals [84]. The introduction of wolves might also reduce the density of deer, the most important host for adult ticks [86], although it is unclear whether densities would be depressed enough to impact tick population dynamics [44].

This work is so far purely correlational and rests on untested assumptions (i.e. that wolf reintroduction will increase small-mammal predation rates, and decrease the abundance of infected nymphal ticks–the metric of disease risk for human Lyme disease incidence; [47]). Clearly, further study is needed to understand whether the correlational relationships among predator densities are causal and to explore the mechanistic interactions among predators and prey species to determine the potential efficacy of this approach. In addition, the general public sometimes opposes reintroducing large predators, so this type of intervention might be challenging politically as well as logistically. Nonetheless, we discuss this example because it illustrates a potential targeted biodiversity conservation intervention suggested by three decades of research on Lyme disease [44,87], and, as noted above, targeted approaches may be more effective, cheaper, and may have fewer incidental impacts on other aspects of human well-being.

The final question for developing biodiversity conservation interventions is to determine whether targeted or broad-scale interventions can be cost-effective compared to other more traditional public health interventions. Most current public health spending is devoted to the development and distribution of vaccines and drugs for treatment, promoting personal protective measures (including avoiding contact with vectors or human–wildlife contact), sanitation, development (e.g. screens on windows) and vector control. Some of these methods are relatively cheap, including hand washing, using insecticide-treated bed-nets for mosquito-borne disease,

or checking oneself for ticks. Others require perpetual action and often come with substantial costs, including vaccination, drug treatment and vector control.

The most rigorous approach to assessing the impact of public health control interventions is analyses that explicitly compare the cost of intervention to a monetary value of disease averted, across a range of transmission intensities. These analyses have guided the roll-out of vaccines, bed-nets and HIV treatment [88–90], and have been done for some ecosystem services [91]. Similar cost–benefit analyses are needed for biodiversity conservation interventions once they can be shown to be efficacious [12].

Previous research provides initial estimates of some of the costs of biodiversity conservation interventions, but much more work is needed for estimating both costs and benefits. For example, setting aside land for protected areas, altering urban planning and large-scale restoration to reduce forest fragmentation all carry both initial costs as well as recurring management costs that vary with the local setting [62,92]. Recurring costs for maintaining protected areas measured per square kilometre per year decrease approximately four orders of magnitude as the size of protected areas increase from 1 to 10 000 km^2 [92]. Costs for reintroductions of extirpated species can also be substantial; initial costs for wolf-reintroduction to Yellowstone National Park included several hundred thousand dollars for translocation and release, in addition to substantial resources to address lawsuits by parties opposed to the reintroduction. Some smaller-scale biodiversity conservation interventions can be cheaper, including removal of invasive vegetation that provides attractive habitat for overly abundant deer [93], or installing owl or bat boxes to increase predation on small mammals and mosquitoes, respectively. However, the effectiveness of these inexpensive measures on large spatial and temporal scales is unknown.

Once the efficacy of biodiversity conservation interventions can be determined, the next step is to combine the cost of the intervention with the benefits of the disease burden alleviated. This is often done for different monetary valuations of disease burden. The final step is to compare the cost–benefit calculations for biodiversity conservation interventions with other approaches to determine which might be most efficacious. Some biodiversity conservation interventions (e.g. species reintroductions, protected natural areas) may have lower recurring costs than many standard public health strategies. This advantage would increase their net benefit in long-term assessments.

6. Conclusion

Humans are continually altering Earth's ecosystems. The high and increasing living standards in developed and rapidly developing countries, respectively, result in the conversion of additional land to agriculture and urban area, increased energy use, climate change and biotic homogenization. These changes present new challenges as novel pathogens spill over from wildlife when human activities encroach into natural areas, pathogens are introduced to new regions, novel host species are introduced to new continents or islands, populations of animals increase or decrease as land-use changes, and climates shift to become warmer, and wetter or drier. These challenging threats to public health may demand novel interventions and strategies. We summarized research needed to develop biodiversity conservation interventions into public health tools. Substantial increases in knowledge are required before the impacts and cost-effectiveness of these interventions on human well-being can be accurately determined, and we have outlined methodologies to advance this approach. If diverse communities can be shown to provide net benefits to human well-being, this could provide powerful motivation for preserving Earth's remaining biodiversity.

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