Biodiversity loss and the ecology of infectious disease

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As a new discipline, the field of planetary health could follow one of two obvious pathways. In the first, practitioners of planetary health would develop a catalogue of the myriad ways in which human health is affected by anthropogenic changes to the environment. Although such an exercise might be valuable, it would not be novel, and the information by itself might not lead to solutions to the critical problems being documented. In the second pathway, planetary health practitioners would use the many adverse health consequences of anthropogenic environmental change as grounds to advocate for better environmental protections. Although there is no doubt that advocacy is necessary to prevent ever more rapid degradation of environmental health, this pathway would de-emphasise the scientific mission of planetary health and emphasise the political one. I argue that there is a third path. Planetary health could document the many cases in which human health and environmental health are simultaneously improved by the same policy or management actions. A thorough exploration of these win–win situations, with careful analysis of the mechanisms that underlie co-benefits to environmental and human health, could uncover key principles and inform new applications, while providing concrete options for policy and management.

Environmental health and human health are both multidimensional concepts. Identification of principles underlying win–win situations requires identification of specific, measurable components of both environmental and human health. In the example that follows, I specify species diversity (a key component of biodiversity) as a focal metric of environmental health and risk of exposure to zoonotic diseases as a focal metric of human health.

Burgeoning research has shown that high biodiversity frequently reduces rates of pathogen transmission and lowers disease risk for human beings, wildlife, livestock, and plants.[1–5] The widespread inhibitory effect of high biodiversity on pathogen transmission arises from a set of mechanisms that are actively being researched. First, most pathogens seem to be host generalists, in the sense that they encounter and potentially infect more than one species of host. Second, these host species differ substantially in their invasibility, susceptibility to infection, and potential to transmit infection to other hosts. Third, in many cases, the hosts most likely to acquire and transmit infection (the so-called reservoir hosts) are species that are abundant, widespread, and resilient to anthropogenic perturbations. Consequently, these reservoir species tend to persist when diversity is reduced, increasing in abundance relative to species more sensitive to disturbance. Consequently, species living in ecological communities with high diversity tend to dilute the effect of the reservoir species and reduce disease risk.[6]

This "dilution effect"[6,7] of high diversity has been best studied in plant and wildlife diseases but is also known to be widespread in human pathogens.[2,8] Its operation can be understood from the following observations. First, most human infectious diseases are zoonotic in origin.[9] These zoonotic pathogens are capable of infecting multiple species of host. Second, although zoonotic pathogens can originate in many different species of vertebrates, certain mammalian orders are over-represented as pathogen sources. Rodents, in particular, carry more zoonotic pathogens than do any other mammalian order, and far more rodent species host zoonotic pathogens than do other orders.[10] Even within the rodents, the species with fast life history traits (early maturity, large litters, and short longevity) are more likely than species with slower life histories to be reservoirs for zoonotic pathogens.[11] Third, fast life histories are typically associated with commonness and resilience to disturbance,[12] suggesting that competent reservoir species will often predominate in low-diversity communities.

The propensity for species of plants and animals with fast life history traits to be among the best reservoirs for multi-host pathogens is under active investigation. One possible underlying mechanism could be a tendency for species with fast life histories to have lower resistance or higher tolerance to infection, resulting from trade-offs between reproduction and self-defence.[13] Another possible mechanism could be a tendency for multi-host pathogens to specialise on host species that they encounter most frequently, which might be hosts with high average population density.[7]

Irrespective of the mechanisms, whenever pathogen transmission or disease risk is lower in high-diversity communities, policies that prevent the loss of biodiversity will simultaneously promote environmental protection and health protection. How common are win–win situations like this, in which a single policy or management option might improve both environmental protection and human health? Are there specific features shared in common by these situations? When trade-offs between environmental and human health prevail, can policy be adjusted to reduce the costs to the environment or to people? To what degree does the characterisation of a policy as win–win versus trade-off depend on the specific environmental health or human health outcome being considered? How frequently is the identification of win–win situations limited by data, and how can data limitations be breached?

Planetary health is uniquely poised to address these and related questions, the answers to which will undoubtedly improve both science and health. The urgency of this enterprise grows as assaults on the environment accelerate.

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