Public health perspective on patterns of biodiversity and zoonotic disease

The recognition that disease ecology, conservation biology, and public health share common ground is an exciting idea, especially if the intersection of these fields results in win-win scenarios for both conservation and public health. Recently in PNAS, Civitello et al. (1) claimed that overwhelming broad evidence supports the dilution effect hypothesis, in which biodiversity loss increases disease risk. Consequently, they suggest that conservation of biodiversity offers a promising strategy to mitigate human health threats. A key element of their argument is that our previous meta-analysis (2), which failed to find unequivocal support for dilution effects in zoonotic disease systems, should be discounted.

How then should we resolve the different findings? First, the two meta-analyses ask different questions and accordingly use different data. Our analysis was designed to investigate whether the public health community should be convinced by the argument that reduced biodiversity increases the risk of zoonotic pathogens of public health concern. We used strict selection criteria to include only studies involving (*i*) pathogens that affect humans but that are maintained in wildlife communities and (*ii*) field studies where disease risk and biodiversity were measured at temporal and spatial scales germane to pathogen transmission cycles. We deliberately excluded results from laboratory experiments in which noncompetent hosts are combined with competent hosts in artificial mesocosms. We also excluded reports that used aggregate data from disparate sources (i.e., susceptible to the ecologic fallacy). We found weak support at best for the dilution effect, and that no zoonotic disease system (e.g., tick-borne diseases, hantaviruses, West Nile virus) showed uniform and predictable relationships with measured biodiversity. Civitello et al. (1) relax the criteria for effect sizes in their analyses of human pathogens and, perhaps as a consequence, reach different conclusions.

Furthermore, we identified evidence of publication bias toward publishing reports of a negative relationship between biodiversity and disease (2), whereas Civitello et al. (1) did not evaluate publication bias. We agree that current measures of publication bias have shortcomings, but without addressing this phenomenon, it is difficult to discern whether conclusions of a broad dilution effect are representative of a broad ecological pattern or an excitement to publish proof of the dilution effect (3).

Interdisciplinary collaborations involving epidemiologists, conservationists, public health agencies, and ecologists (the "One Health" approach) certainly hold merit for synergistically maintaining ecological integrity and public health (4). Nonetheless, ecological drivers of human disease are idiosyncratic and can be spatially and temporally dependent (5). Public health interventions are unlikely to be based on broad ecological generalizations if the evidence base for impacts of biodiversity on particular pathogens is only locally specific or lacking. To incorporate the science of biodiversity–pathogen relationships into public health initiatives better, we suggest an improved understanding of pathogen transmission dynamics within the context of local community ecology: incorporating multiple potential influences and multiple pathogens into modeling frameworks; demonstrating the influence of biodiversity upon the force of zoonotic infection in human populations, especially reflecting conservation measures; and devising specific recommendations for interventions.

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