ESSAY

ENVIRONMENT

Environmental change and the ecology of infectious disease

When and where do human impacts increase disease risk for people and wildlife?

By **Chelsea L. Wood**

More than half of all the infectious diseases that affect humans are zoonoses—pathogens naturally transmitted from animals. Because a substantial proportion of these diseases originate in wildlife, environmental context drives patterns of transmission. But despite the strong influence of environment on zoonotic pathogens, considerable uncertainty exists as to whether and how anthropogenic environmental change modulates disease risk.

Does loss of biodiversity generally increase or decrease disease agent transmission? In other words, do human impacts on biodiversity increase the prevalence of diseases by eroding natural "checks and balances" on transmission, or do they decrease prevalence when they remove the free-living biodiversity on which disease agents depend? As human impacts on ecosystems accumulate and the perceived threat of zoonotic disease grows, answers to this question are urgently needed. In addition to offering the possibility of improved disease control, research on the role of biodiversity (and biodiversity loss) in disease transmission also presents a timely opportunity for refining fundamental principles in ecology. Parasites compose—by some estimates—more than half of Earth's species, and new data suggest that their influence, though sometimes hidden, can be substantial.

My doctoral research explored the ecology of infectious disease in a changing world: specifically, in a world subject to biodiversity loss, habitat disturbance, and other anthropogenic impacts. I focused on two fundamental questions: (i) what are the effects of anthropogenic environmental change on the abundance, diversity, distribution, and transmission of parasites and pathogens and (ii) how can we harness our knowledge of the ecological dimensions of disease agent transmission to address human disease burdens? I tackled these questions with diverse approaches, including empirical observation at the macroecological scale to investigate patterns of parasite distribution across natural variation in anthropogenic pressures and synthesis to assess generality of patterns across ecosystems and disease agents.

Anthropogenic environmental change can radically impoverish marine biodiversity, and fishing is among the most important drivers of biodiversity loss in ocean ecosystems. What is the effect of such fishing-driven biodiversity loss on marine parasite assemblages? As a Ph.D. student, I used exploited marine fishes and their metazoan parasites as a model system to explore this question, first generating several hypotheses (*1*), and then testing these hypotheses by using fished and unfished coral islands of the Line Islands archipelago as replicates in a natural experiment. This work showed that fishing can increase the abundance of some parasite taxa and decrease the abundance of others, depending on parasite traits (*2*, *3*). Specifically, parasites with complex life cycles were vulnerable to fishing-driven declines, probably because they require multiple host species, some of which are the top predators most sensitive to fishing impacts. In contrast, directly transmitted parasites tended to increase in abundance, probably because of fishing-driven compensatory increases in the abundance of their hosts.

A similar result arose from a comparison of the parasites of exploited host species inside and outside of marine reserves in central Chile, where a subset of parasites with particular traits (in this case, short transmission distances) responded to protection of their hosts with marked increases in prevalence (*4*). These general patterns were also confirmed in a meta-analysis of studies reporting the composition of parasite assemblages in marine reserves and matched open-access areas (*5*).

These results are surprising, because they contradict a portion of the disease ecology literature that suggests that high biodiversity should buffer against disease transmission by diluting contacts among competent hosts. Instead, my work to date implies that—although there may be a few "winners" among parasites in fished ecosystems—many parasites will decline alongside their hosts.

The conclusion that I reached from my empirical work on marine parasites was corroborated by a study that I led to synthesize how biodiversity loss mediates the risk of zoonotic disease for human populations (*6*). Just as I observed among fish parasites, environmental change appears to have variable effects on transmission of human disease agents, with many disease agents responding negatively to biodiversity loss. This is in direct disagreement with the assumption, held by many ecologists, that disease control can be achieved

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Category Winner

Environment: Chelsea Wood for the essay, "Environmental change and the ecology of infectious disease." Dr. Wood received her Bachelor of Arts degree from Dartmouth College and her Ph.D. from Stanford University. She did postdoctoral research in Pieter Johnson's lab at the University of Colorado at Boulder, and is currently a Fellow in the Michigan Society of Fellows and an Assistant Professor in the Department of Ecology and Evolutionary Biology at the University of Michigan. She is interested in how parasites and pathogens respond to human impacts on the environment.

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through biodiversity conservation. I bolstered this finding by drilling down into the mechanisms that link a single, well-studied zoonotic disease—Lyme disease—to biodiversity in northeastern North America and found that Lyme disease risk actually increases with biodiversity along broad-scale disturbance gradients (e.g., urban to suburban to rural), contrary to the expectation of many ecologists (*7*, *8*).

To date, my research has shown that the effects of anthropogenic biodiversity loss on disease transmission can be positive, negative, or neutral, depending upon ecological context: the disease agent, the ecosystem, and the type and magnitude of the impact. Now that it is becoming clear that anthropogenic impacts have complex effects on disease risk, the next challenge for disease ecology is to identify general principles that govern the disturbance–disease relation and thereby develop the capacity to predict when, and under what conditions, environmental change might lead to increased disease risk for humans and wildlife.

My synthetic work on zoonotic disease suggested that biodiversity conservation was not an effective tool for disease control; but what options for ecological intervention might be available to reduce human and wildlife disease burdens? These studies suggest a variety of potential options that will need to be pursued by cross-disciplinary teams that include ecologists, epidemiologists, physicians, and social scientists. This research represents an unusual opportunity—both for building an enhanced understanding of ecological complexity and for equipping policy-makers to protect the world's human populations from infectious disease. ■

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