Biodiversity Loss Affects Global Disease Ecology

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Changes in the type and prevalence of human diseases have occurred during shifts in human social organization, for example, from hunting and gathering to agriculture and with urbanization during the Industrial Revolution. The recent emergence and reemergence of infectious diseases appears to be driven by globalization and ecological disruption. We propose that habitat destruction and biodiversity loss associated with biotic homogenization can increase the incidence and distribution of infectious diseases affecting humans. The clearest connection between biotic homogenization and infectious disease is the spread of nonindigenous vectors and pathogens. The loss of predators and hosts that dilute pathogen transmission can also increase the incidence of vectorborne illnesses. Other mechanisms include enhanced abiotic conditions for pathogens and vectors and higher host-pathogen encounter rates. Improved understanding of these causal mechanisms can inform decisionmaking on biodiversity conservation as an effective way to protect human health.

Keywords: biodiversity, emerging disease, vectorborne disease, public health, ecosystem services

Patterns of health and disease have undergone dramatic transitions during human history, reflecting social and ecological changes (Barrett et al. 1998, McMichael 2001). Beginning about 10,000 years ago, the first epidemiologic transition associated with the acceleration of emerging disease occurred during the shift from hunting to agriculture. Along with permanent and larger settlements, animal domestication, and changes in human diet and social organization came a rise in zoonotic infection (Wolfe et al. 2007). As communities became densely crowded, more virulent forms of human pathogens evolved (Ewald 1994). A second transition occurred during the Industrial Revolution. The incidence of infectious disease and infant mortality decreased, lengthening life expectancy. At the same time, noninfectious chronic disease rose, accounting for a greater share of overall mortality. Greater water and air pollution caused higher rates of cancer, allergies, birth defects, and impaired cognitive development. These transitions were not uniform: In some less developed parts of the world, infectious diseases remained a recurring problem and chronic diseases became more common (Barrett et al. 1998).

It has been proposed that we are in the midst of a new epidemiologic transition, in which globalization and ecological disruption appear to be associated with newly emerging infectious diseases as well as reemerging infections previously thought to be under control (Barrett et al. 1998, McMichael 2001). Such disruption and population expansion are especially important in the dramatic rise in zoonotic diseases (Wilcox and Gubler 2005, Jones et al. 2008). At the same time, greater mobility and trends in urbanization have contributed to the global nature and reach of these diseases. Although it is generally accepted that we are in the midst of an accelerating global extinction (Pimm et al. 1995), the connections between biodiversity decline and emerging and reemerging disease in this period have received little attention. In this article, we discuss the connections between biodiversity loss and human health at multiple hierarchical levels to understand the mechanistic basis of this transition.

Perhaps the clearest link between biodiversity and human health is through the spread of invasive species and pathogens. Globalization and the transfer of exotic organisms have resulted in widespread biotic homogenization—the replacement of local biota with nonindigenous species. These exotic species can cause extinctions of local taxa, resulting in the loss of diversity at many levels, from genetic variation to species number. The spread of vectors and disease has also been an integral part of human history, affecting people and wildlife. During recorded history, some insect vectors have dispersed into new habitats by phoresy (i.e., one organism transporting another), flight, or wind, but human-aided transport has been responsible for the arrival and spread of most invasive vectors, such as fleas, lice, and mosquitoes. Yellow fever,

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dengue, malaria, and West Nile encephalitis are some of the diseases that have breached biogeographic barriers through anthropogenic movement (Lounibos 2002).

Biodiversity loss can also have a direct effect on zoonotic disease transmission. Plant or structural diversity and habitat complexity can influence the composition, abundance, and distribution of animals that play important roles in the transmission cycles of some human diseases. For example, deforestation and habitat fragmentation or modification, and the accompanying loss of structural diversity, can lead to changes in human contact rates with a variety of pathogens and disease vectors (Vittor et al. 2006). Changes in the diversity or composition of animal hosts may be closely associated with the incidence of zoonotic diseases such as Lyme disease or West Nile virus (WNV) in humans (LoGiudice et al. 2003, Ezenwa et al. 2006). At the same time, it is important to note that parasites can be important drivers of biodiversity and components of ecosystem health (Hudson et al. 2006). Some disease dynamics may operate independently of biodiversity if they rely on nongeneralist vectors or a certain abundance of reservoir hosts (Kilpatrick et al. 2006). Finally, although additional hosts can reduce the transmission rates of particular diseases, they can also harbor other pathogens.

The links between biodiversity and human health occur from the microbial level to that of the habitat. Mechanistic pathways that lead from changes in biodiversity to human health can occur at the genetic, microbial, organismal (host or vector species), community, and habitat levels. The range of pathways is summarized in table 1. In this review, we discuss case studies in which these links have been investigated in some detail. We then summarize general lessons from this research and identify several key questions that remain to be answered. We conclude by describing how decisionmakers can use such information to design conservation initiatives that protect both biodiversity and human health.

Malaria

Malaria is an infectious disease transmitted by the female *Anopheles* mosquito. Four species of the malarial parasite *Plasmodium* commonly infect humans and cause various forms of disease. Despite the eradication of malaria from most temperate areas, populations at risk continue to grow, with at least 300 million cases per year and 1 million deaths (WHO 2007). Flulike symptoms may include fever, chills, muscle aches, headache, and nausea. In the most severe cases, parasites travel to vital organs such as the brain, which can swell, leading to seizures, coma, and, in 20% to 50% of such cases, death (Gilles and Warrell 1993).

Changes in plant diversity—particularly through habitat alteration, fragmentation, and deforestation—can increase the risk of malaria transmission through effects on mosquito survival, density, and distribution (Yasuoka and Levins 2007). Deforestation can also increase transmission by raising surface-water availability and creating new breeding sites for some *Anopheles* mosquitoes (Walsh et al. 1993). It can also affect microclimates: Existing larval habitats may be exposed to more sunlight, raising water temperatures and changing community dynamics, which can increase the survival of larval mosquitoes (Tuno et al. 2005). A warmer microclimate can cause mosquitoes to digest blood meals more quickly, leading them to feed and lay eggs more often, resulting in higher rates of vector development and reproduction (Afrane et al. 2006). Higher temperatures also affect the malaria parasite itself, reducing its development time and making mosquitoes infectious more quickly (Gilles 1999). Land-use changes can have multiple impacts on disease transmission, as the risk of malaria can depend on the arrival of opportunistic vectors, adaptation of vectors to newly created niches, and migration of nonimmune people (Walsh et al. 1993).

Vittor and colleagues (2006) were the first to demonstrate the adverse impacts of deforestation through changes in

Table 1. Mechanisms linking biodiversity change and human health at different levels.

Level of diversity	Aspect of biodiversity undergoing change	Possible mechanism leading to human health effect
Genetic	Gene frequencies within populations of pathogens or hosts	Change in pathogen virulence or host resistance
Microbial	Composition of microbial communities in the external environment or within the host	Change in pathogen exposure or virulence; change in host immune response and allergic sensitization; expansion of range through anthropogenic transport
Vector species	Abundance, diversity, composition, and geographic range of vectors	Change in host-vector contact rates; change in contact between infected vectors and humans; expansion of range through anthropogenic movement
Host species	Diversity, composition, and range of host species	Change in host-pathogen contact rates; change in competent host-vector contact rates; change in pathogen prevalence; expansion of range through anthropogenic transport
Community (interacting species including predators, competitors, etc.)	Host density and contact with pathogen; host susceptibility to infection	Change in pathogen prevalence; change in human-pathogen contact rates
Habitat structure	Structure, complexity, and diversity of vegetation	Change in vector abundance and composition; change in host composition and distribution; change in host-pathogen contact rates; change in vector-host contact rates; change in infected vector-human contact rates; change in host-human contact rates

potential malaria transmission in Amazonian Peru. Compared with forested areas, deforested sites had higher densities of *Anopheles darlingi,* the most efficient vector of malaria in the region, and higher rates at which humans are bitten. This relationship held true regardless of human population density. The effects of landscape change on mosquito distribution may be complex, involving changes in species composition, abundance, and distribution. Such changes can be influenced by habitat availability and changing microclimates. Human factors, such as immune status, migration patterns, and treatment of disease, also play important roles in malaria incidence and continued transmission (Molyneux 1998). Furthermore, there is a need to consider the effects on disease transmission of human behaviors that contribute to ecological change and behavioral responses to disease risk (Pattanayak and Yasuoka 2008). An interdisciplinary approach to studying the relationships among structural diversity, habitat availability, vector ecology, epidemiology, and human behavior is critical in reducing the risk of malaria.

Schistosomiasis

The loss of predators can cause dramatic changes in ecosystem processes and functioning. Recent studies have shown that such declines can also affect the transmission of parasitic illnesses. Evidence from Lake Malawi, for example, indicates that overfishing of mollusk-eating fish has resulted in a greater number of *Bulinus* gastropods and the subsequent spread of schistosomiasis.

Schistosomiasis is a parasitic disease that infects humans through skin contact with the free-swimming larval stage (cercariae) of trematodes that develop in freshwater snails. Approximately 200 million people are infected with the disease worldwide, which can be chronic, causing liver and intestinal damage. In Malawi, overfishing and the increased use of fine-mesh beach seines have caused a decrease in density of the snail-eating cichlid *Trematocranus placodon* (Stauffer et al. 2006). The decline of these predators in Lake Malawi appears to have been responsible for the rise in transmission of the schistosome parasites in the lake since the 1980s (Evers et al. 2006). In Kenya and Cameroon, the introduction of cichlids has been shown to be relatively ineffective in eliminating or reducing schistosomiasis infections (Slootweg et al. 1994), but there may be a role for other natural predators in integrated control efforts. For example, Stauffer and colleagues (2006) contend that a higher density of molluscivorous fishes will reduce cercariae production in the open waters of Lake Malawi and function as a biological control of schistosomes and their hosts. Research into the relationship between snail species richness and schistosomiasis transmission is of interest, since it may reveal other opportunities for controlling transmission through protection or manipulation of the snail community.

Lyme disease

Recent studies have demonstrated that variation in host species diversity has a strong effect on the level of human risk from Lyme disease, the most frequently reported vector-borne disease in the United States. The causative agent of this disease, the spirochete bacterium *Borrelia burgdorferi,* is transmitted among hosts, including humans, by blood-feeding ticks, such as *Ixodes scapularis* in eastern and central North America and *Ixodes pacificus* in the western United States.

Ticks feed readily from dozens of species of vertebrates, but host species differ dramatically in their probability of transmitting the infection (LoGiudice et al. 2003). Several studies have documented that white-footed mice (*Peromyscus leucopus*) feed and infect large numbers of ticks, whereas most other hosts serve a protective role by feeding but not infecting the vector (e.g., LoGiudice et al. 2003, Brisson and Dykuizen 2006). In areas dominated by white-footed mice, ticks have a high probability of becoming infected and dangerous to people. The species composition and relative abundance of a host community strongly influence key metrics of disease risk to humans (Ostfeld and LoGiudice 2003). Because white-footed mice tend to reach higher abundances in species-poor communities (Nupp and Swihart 1998), host community diversity also influences Lyme disease risk. Empirical studies confirm that disease risk is significantly higher in areas of low vertebrate diversity, such as small forests (less than 2 hectares [ha]; Allan et al. 2003) and highly fragmented landscapes (Brownstein et al. 2005).

Hantavirus pulmonary syndrome

A similar protective effect of high vertebrate diversity seems to occur for hantavirus pulmonary syndrome (HPS) in Panama, where the rice rat (*Oligoryzomys fulvescens costaricensis*) is the primary natural reservoir of the causative agent, Choclo virus. Hantaviruses are found in Asia, Europe, and the Americas, where particular virus species are associated with specific rodent hosts. Hantaviruses are transmitted among rodents through biting and scratching. Transmission to humans can occur when aerosolized rodent feces or urine is inhaled. In the Americas, several hantaviruses cause severe pulmonary disease, with death rates as high as 37% (CDC 2004). Ruedas and colleagues (2004) surveyed small-mammal communities in southeastern Panama, where an outbreak of HPS occurred in 1999 and 2000. On the basis of an analysis of 13 locations near human-case clusters and a control site, they suggested that human activities that decrease biodiversity, and during periods with unusually heavy precipitation, can increase the probability of HPS outbreaks. An experimental reduction in small-mammal species diversity in Panama's Azuero Peninsula confirmed that high rodent diversity reduces the density of infected reservoir hosts, a key risk factor for humans (Suzán et al. 2009). Rodents' life history patterns may be another mechanism by which biodiversity decline increases the human risk of infectious disease (Mills 2006). Rodents that host hemorrhagic fever viruses can adapt relatively easily to changing environmental conditions, and their fecundity and high reproduction rate

allow them to reach high population densities, which facilitate pathogen transmission.

Peixoto and Abramson (2006) built a mathematical model to predict the effects of variation in biodiversity on HPS dynamics in North America, where the deer mouse (*Peromyscus maniculatus*) is the primary reservoir. They compared the transmission of HPS in a community that consisted only of *P. maniculatus* to more diverse communities that included other competing mouse species. The model assumed that the competitor mice do not become infected with HPS, an assumption supported by field data. Consistent with the empirical observations of Suzán and colleagues (2009), the model indicated that hantavirus infection in deer mice should decrease or even disappear in the presence of competitors.

For both Lyme disease and HPS, the mechanisms that underlie the negative correlation between diversity and disease risk or incidence require clarification. In the case of Lyme disease, these include interactions between hosts and ticks, between hosts and pathogen, and among different host species: For example, an abundance of hosts other than mice appears to reduce tick burdens on *Peromyscus* (Brunner and Ostfeld. 2008). For HPS, which does not involve a vector, only the host-pathogen and host-host mechanisms are possible. Lower population density of reservoir hosts and a lower proportion of individuals that are infected with the pathogen under higher diversity conditions suggest that both mechanisms operate (Suzán et al. 2009). Higher diversity of small mammals appears to regulate populations of reservoir hosts through competition or predation. High small-mammal diversity might also inhibit intraspecific aggressive encounters between reservoir hosts that result in hantavirus transmission (Suzán et al. 2009). A major research frontier is to determine the relative importance of these potential mechanisms of the dilution effect (Keesing et al. 2006) and assess their generality among various zoonoses. In principle, experimental manipulations of host communities combined with measurements of host burdens, abundances of unmanipulated species, and infection prevalence or reservoir competence would reveal the strength of each mechanism.

West Nile virus

Wild birds probably serve as the primary reservoir and amplification hosts of WNV (Marra et al. 2004). The first known case of this mosquito-borne zoonotic disease occurred in Uganda in 1937. It is now found in Africa, Europe, Asia, and North America. West Nile virus appeared in the United States in 1999, when encephalitis was reported in humans and horses (Hayes et al. 2005). Although it is unknown how this recent invasion began, the spread of WNV has caused the decline of bird populations throughout North America (LaDeau et al. 2007). In humans, only about 20% of WNV infections are symptomatic, and the virus has a broad spectrum of clinical manifestations, ranging from febrile illness to neuroinvasive disease (Hayes et al. 2005). The most important mosquito vectors are thought to be *Culex* species (Andreadis et al. 2001). Most mammals appear to be incompetent or dead-end hosts for the virus, but among birds, competence varies widely: American robins (*Turdus migratorius*) and American crows (*Corvus brachyrhynchos*) are highly efficient virus transmitters; rock doves (*Columba livia*) and Canada geese (*Branta canadensis*) and other wading birds are very poor hosts (Komar et al. 2003, Reisen et al. 2005).

The ecological complexity associated with WNV transmission, including the virus's broad host range, wide variability in host competence, and the catholic feeding habits of associated vectors, make it a strong candidate as a disease system where transmission dynamics may be linked to patterns of host and vector diversity. In the simplest case, a dilution effect in highly diverse host communities could result from a reduction in the encounter rate between vectors and the most competent hosts (Keesing et al. 2006). Recent work provides evidence that host diversity is linked to WNV ecology in the United States. For example, Ezenwa and colleagues (2006) documented a negative association between the number of nonpasserine bird species (mainly wading birds) and WNV infection prevalence in *Culex* mosquitoes across a series of field sites in a single Louisiana county; the same trend was also apparent on a larger scale for human disease incidence across the state. Similarly, Allan and colleagues (2009) found that bird diversity was negatively correlated with WNV infection in vectors at a regional scale in St. Louis, Missouri, and with human disease incidence at a national level across the United States.

The mechanisms underlying potential dilution effects in WNV transmission remain to be clarified. The relative role of species richness versus species composition is particularly important in the face of increasing habitat loss and land-use change, since these forces contribute not only to a reduction in numbers of species (Foley et al. 2005) but also potentially favor generalist species that act as amplification hosts. High transmission among birds in an urban environment could be the result of a decline in overall bird diversity or greater densities of highly efficient WNV hosts in peridomestic settings. Thus, in low-diversity communities, species identity is likely to play a key role in disease transmission.

The role of land-use change in altering WNV transmission may go beyond the effects of urbanization. One study showed that as wetlands decline in the Gulf Coast of Louisiana, the prevalence of WNV in mosquitoes grew (Ezenwa et al. 2007). Of the factors potentially explaining this pattern, the relative abundance of passerine or perching birds to nonpasserine birds was the most important explanatory variable. This suggests that the effects of wetland loss on WNV in this region are very likely mediated by changes in bird species composition. Future research on the complex causal links between land-use change, species diversity and composition, and host-vector interactions will be essential for understanding associations between biodiversity and disease risk. Indeed, a recent study of WNV ecology in the Chicago area found no evidence of an association between avian diversity and WNV prevalence in either mosquito vectors or birds (Loss et al. 2009), suggesting that interactions among anthropogenic,

biotic, and abiotic factors may drive regional variability in the dynamics of this disease.

Forest fragmentation, human encroachment, and pathogen exchange

Anthropogenic changes to forest habitats can reduce biodiversity and bring people into closer contact with wildlife, increasing the risk of zoonotic disease transmission. Nonhuman primates have emerged as important disease reservoirs in this regard, not only because of their physiological similarity to people but also because of their ecological responsiveness to habitat disturbance (Chapman et al. 2005).

Recent studies in western Uganda show that forest disturbances can affect infectious disease dynamics at local scales. Forest fragmentation can alter disease dynamics in primates, and human encroachment strongly affects cross-species infection rates (Goldberg et al. 2008a). Goldberg and colleagues (2007) found that rates of interspecific bacterial exchange are elevated between people and nonhuman primates even where direct contact does not typically occur: Humans frequenting chimpanzee habitats tend to share genetically similar gastrointestinal bacteria with the apes, and the chimpanzees themselves harbored bacteria resistant to multiple antibiotics, even though they had never been administered to local wildlife. People and livestock living near forest fragments with monkeys exchange gastrointestinal bacteria at accelerated rates (Goldberg et al. 2008b). The greater the disturbance of a forest fragment, the greater the rate of bacterial transmission. Biodiversity loss in these fragments may increase zoonotic and anthroponotic pathogen exchange by forcing species into atypical ecological interactions that facilitate transmission.

The study of wild primates may help reveal the relationship between biodiversity and infectious disease. Large-bodied, conspicuous animals with complex social systems and diverse habitat requirements, primates play key ecological roles in forests as seed dispersers, competitors, predators, and prey (Onderdonk and Chapman 2000). The transmission dynamics of pathogens between humans and nonhuman primates may be especially sensitive to changing patterns of biological diversity.

Improving biodiversity and human health policies

Links between biodiversity and human health are often complex, with the interplay of regional and global drivers, such as human migration and climate change, acting over relatively long periods of time. The mechanistic pathways have been identified in the case studies discussed above and in other health outcomes that include viral, bacterial, protozoan, and metazoan agents (summarized in table 2). We have focused on human disease as an end point, but there are other non-disease health consequences of biodiversity loss, such as psychological well-being, that have been investigated (deVries et al. 2003, Fuller et al. 2007). At the level of microbial diversity, reduced exposure to natural microbial diversity has been linked to decreased immune tolerance to allergens (box 1). On the landscape scale, biodiversity conservation in the

Table 2. Summary of case studies linking biodiversity change to health effects in humans.

Disease system	Component of biodiversity change	Location	Effect on human health	Proposed mechanism	Observations
Lyme disease (*Borrelia burgdorferi*)	Host diversity	Northeastern United States	Changes in *Peromyscus leucopus* mouse abundance influences risk of Lyme disease transmission to humans	Relative abundance of competent reservoirs (small mammals) affects vector (black-legged tick) infection rates	Many hosts vary in reservoir competence and may contribute to disease risk
Hantavirus pulmonary syndrome (HPS)	Host diversity	Panama	HPS outbreaks associated with less diverse rodent assemblies	Absence of nonreservoir species is associated with higher abundance and infection prevalence in competent reservoir species	Lower diversity increases encounter rates between infected and susceptible hosts
West Nile virus	Host diversity	Louisiana, United States	High avian diversity associated with lower mosquito infection rates and human disease incidence	Presence of alternative host species diverts mosquito blood meals away from more competent hosts	Feeding preferences of mosquito species remain unknown
Schistosomiasis (*Schistosoma haematobium*)	Predator diversity	Lake Malawi, Africa	Human prevalence of schistosomiasis rises as populations of snail-eating fish decline	Fish predation on intermediate hosts reduces *Schistosoma* larval production	
Enteric disease	Structural diversity	Uganda	Human activity and forest fragmentation elevate human-primate microbial exchange	Processes underlying forest fragmentation drive increased contact between humans and sources of infection	Habitat types may influence microbial abundance and community composition
Malaria	Structural diversity; vector diversity	Peru	Deforested sites have greater densities of vectors and higher human biting rates, leading to increased risk of human infection	Change in canopy structure influences density and diversity of malaria-transmitting vectors	Aquaculture activities in newly settled areas may contribute to vector breeding
Asthma	Microbial diversity	Urban areas in the United States and Western Europe	Lack of contact with diverse bacteria is associated with some allergic and inflammatory diseases	Changes in microbial diversity lead to imbalanced immune system responses	Diet, lifestyle, and genetics may also play a role in causing these chronic diseases (see box 1)

Box 1. Microbial diversity and human disease.

Microbial ecology, the study of the interrelationships between microorganisms and their living and nonliving environments, is an active area of research that has great potential to inform our understanding of the relationships between biodiversity and health. In humans, certain bacteria are vital to the proper development of the immune system, protection against microorganisms that could cause disease, and the digestion and absorption of food and nutrients. The ecological balance of microbes in and on a person's body can have major impacts on that person's health and well-being, with the best-known examples being allergic diseases such as asthma, food allergies, and inflammatory bowel disease (IBD). These immune disorders appear to result from changes in exposure to microbial populations (mainly bacteria). Natural exposure to microbes through mucosal surfaces may be critical for the development of the immune system and of clinical tolerance to allergens, and microbial diversity may offer a front line of defense against these common diseases (Strober et al. 2002).

Policy implications: Traditional medical paradigms have stressed antibiotics, or pre- and probiotics, as therapeutic strategies for modulating specific constituents of microbial communities. The recognition that maintaining a balanced and diverse microbial population in various physiological environments is important for health presents new opportunities for ecologically informed public health interventions. For example, insufficient exposure to a diverse population of microbes (especially to gram-negative bacteria) may result in an immune system imbalance that favors allergic responses, such as asthma. Public health recommendations could target at-risk populations in Western cities known to have inadequate exposure to endotoxins. Controlled clinical exposure to certain microbes or microbial factors can stimulate host immune system defenses. Probiotics also could help correct microbial ecological imbalances, treating conditions such as food allergies (Iweala and Nagler 2006), IBD, and vaginal infections (Zhou et al. 2007). Indeed, probiotics are already available commercially as dietary supplements intended to restore the physiological homeostasis of microbial communities. Further research at the interface of microbial ecology and the health sciences can lead to unique insights into the etiology of diseases linked to microbial exposure, and to new strategies for disease prevention and treatment.

Amazon has been linked to the mitigation of vectorborne diseases and the resulting impact on economic well-being, measured by macroeconomic indicators such as gross domestic product, exports, imports, and national investments (Pattanayak et al. 2007).

For infectious diseases in which high species diversity has been demonstrated to reduce disease risk or incidence, environmental policies that maintain or enhance diversity should be supported. For Lyme disease and WNV, high risk or incidence is associated with the loss of vertebrate diversity, which in turn is associated with certain types of habitat destruction and fragmentation (Allan et al. 2003, LoGiudice et al. 2003, Ezenwa et al. 2006). Protecting large forested areas in the vicinity of residential areas may reduce the risk of Lyme disease. Land-use zoning policies that promote the spatial aggregation of deforestation could help maintain contiguous forested areas at the same time. For example, designing housing developments in which deforested areas, or lawns, are clustered and forests are left unfragmented might reduce the disease risk associated with small and scattered forest patches.

For Lyme disease and HPS, small mammals such as mice and shrews are the most important natural reservoirs of the pathogen. Consequently, direct control of these reservoirs—for example, by kill trapping or poisoning—represents an alternative to broader-based environmental management of land use. However, interventions to reduce populations of small rodents over epidemiologically meaningful scales may be impractical, and could cause indirect negative effects on the environment. For example, white-footed mice regulate populations of gypsy moths (*Lymantria dispar*), a destructive, exotic pest that causes enormous economic damage by killing trees (Jones et al. 1998). Mice also serve as a key food supply for carnivorous mammals and birds, and their removal or reduction could indirectly result in wider-scale biodiversity loss. Targeting reservoir hosts for vaccination might become a feasible strategy for reducing disease risk (Tsao et al. 2004), although practical methods for delivering vaccine to sufficient numbers of mice or other wildlife remain elusive. Further research on the role of biodiversity in WNV transmission may inform land-use planning that preserves habitat favoring populations and species that moderate disease transmission. For example, natural wetlands protection or restoration in the Gulf Coast region could be geographically targeted to decrease mosquito infection prevalence.

The role of predators and predator loss in infectious disease transmission is an emerging area of study. For prey populations, predator removal is likely to be harmful when parasites are highly virulent, macroparasites are aggregated in their prey, hosts are long-lived, and predators select infected prey (Packer et al. 2003). For zoonotic diseases, the connections are less clear. The role of terrestrial vertebrates in controlling rodent reservoirs of human disease depends on the importance of rodent population density to the disease incidence, the roles of predators in determining population dynamics and rodent density, and the effects of human-caused environmental change on predators and prey (Ostfeld and Holt 2004).

Stauffer and colleagues' (2006) studies of Lake Malawi indicate that overfishing has direct effects on human health. The management and restoration of snail-eating cichlids such as *Trematocranus placodon* could reduce schistosomiasis transmission in the lake. The return of this important food fish also would make an important resource available to local populations. Yet biological control by a single species will

not completely eliminate the disease (Slootweg et al. 1994, Stauffer et al. 2006). Integrated management and ecological restoration may be necessary to rid schistosomiasis from infected areas.

Predatory fish have been successfully exploited as biological controls against dengue-transmitting *Aedes aegypti* mosquitoes in Australian wells and around gold mines (Russell et al. 1996). In some cases, mosquito control can be better achieved by combining biocontrols with environmental modification (Bence 1982). Application of the bacteria *Bacillus thuringiensis* and *Bacillus sphaericus* has been effective in reducing malaria in Nicaragua, China, and Cameroon (Sutherst 2004). Biological controls can be sustainable and cost effective against some disease-carrying vectors, and a combination of strategies that are locally appropriate may be required.

Understanding how habitat alteration and human settlement affect the abundance and distribution of important vector species can help identify landscape features and human behaviors, such as patterns of outdoor recreation and natural resource extraction, that may be useful in predicting disease risk. Land-use policies could be modified to help minimize or prevent potential adverse health effects from a greater abundance of important malaria-transmitting vectors and contact with humans. At the same time, in addition to traditional malaria control measures, communities might rely more on environmentally based strategies to target vectors, including building closed wells, applying biocontrols, and regularly clearing or removing accessible vector breeding sites. A better understanding of how people use natural areas and thus, possibly inadvertently, increase their risk of disease can help guide public policies that focus on behavioral change. Several management strategies may be beneficial in cases where changes in structural diversity are accompanied by human encroachment, including discouraging destruction of wildlife habitats by restricting access, providing alternative sources of income or substitutes for natural products such as fuel wood and bushmeat, and modifying crop-planting practices. For example, fruit trees favorable to animals such as primates and bats could be maintained in core areas of forest fragments in order to reduce human-wildlife conflict, such as crop raiding, at the edges of disturbed habitats.

Ultimately, people's disease-prevention behaviors, including changes to ecosystems, respond to disease levels in their communities, suggesting a dynamic feedback between exposure and control (Pattanayak and Yasuoka 2008). Data from Brazil and Indonesia show that the deforestation-related risks of malaria are three times greater than predicted by a simple model lacking the potential feedback of malaria-stricken communities that clear more land because of the poverty associated with the disease. The incorporation of human behavior marks a departure from a purely biophysical approach, one that can easily overlook social, cultural, and economic drivers crucial to understanding anthropogenic ecosystem disruptions and their health impacts (Parkes et al. 2003). Traditional ecological research and interdisciplinary studies by health and social scientists can help account for the interactions and feedbacks between ecological and human behavioral factors (box 2).

Box 2. Decisionmaking tools.

The lack of sufficient and reliable information prevents the full and careful consideration of environmentally based policy strategies for improving human health. Cost-effectiveness analysis (CEA) and benefit-cost analysis (BCA) are two prominent decision-support tools. The quantification of effects is necessary for applying either CEA or BCA. To apply CEA or BCA to a broader range of human health protection measures—including those based on ecosystem protections or other environmentally based measures suggested by the case studies reviewed above—additional research should also focus on quantifying the health and ecological effects of alternative management approaches and estimating the values of changes in relevant health and ecological end points. Anticipating the potential effects of an intervention can help avoid unintended consequences if, for instance, an action reduces the incentives to use personal preventative measures or increases incentives for land-use changes that will expose more people to disease-transmitting vectors. Valuing changes in ecological and health end points is a necessary input for BCA and can be useful in its own right for setting priorities by improving our understanding of which health and ecological risks people consider to be most important. This may require novel applications of economic valuation methods that typically have been used by environmental economists to value nonmarket goods and services, such as improvements in recreation opportunities and air and water quality (Freeman 2003). Human health scientists and ecologists have a role to play here as well, in helping economists to design environmental valuation studies to properly account for the joint production of ecological and human health benefits.

The value of information: Incomplete knowledge, combined with the irreversible nature of some forms of biodiversity loss, such as species extinctions, implies a type of premium for biodiversity protection called quasi-option value. This is the value of forestalling an irreversible change with uncertain consequences based on the possibility of learning more in the interim. The value of new research in biodiversity and human health will depend on its potential for discovering ways to enhance the effectiveness of current public health measures or for discovering new, more cost-effective measures. Quasi-option value provides a conceptual tool for targeting new research such that a balance is achieved between varying probabilities of success and potential payoffs.

Conclusion

Along with increasing mobility, demographic change, and anthropogenic disturbance, we appear to be undergoing a distinct change in global disease ecology. Recent studies at the interface of biodiversity and health are helping to elucidate how changes in biological diversity affect health-related outcomes, but policies that are derived from basic research still need to be designed and implemented.

Clearly, there is a need for improved health monitoring of human populations in at-risk areas where population growth is high, ecologically disruptive development is under way, and human and wildlife overlap considerably. Jones and colleagues (2008) noted that global resources to combat the risk of emerging infectious diseases are poorly allocated: The risks of emergence are greatest in lower-latitude developing countries in the tropics, particularly for zoonotic diseases of wildlife origin. Effectively addressing emerging diseases requires adequate health infrastructure that recognizes the environmental, epidemiological, and social drivers of disease transmission.

The links between changes in ecosystems, biodiversity, and infectious diseases are complex. These links may involve other social and global environmental changes, such as climate change, migration, and population growth, which occur over different scales of space and time, so there may be many ways in which these factors interact to affect human disease. The complexity of disease life cycles and the ecology of disease, including pathogen genetics and evolution and species interactions, need to be better appreciated and understood. The case studies described in this article have clear implications for how we make decisions related to pest management of vectors, hosts, and pathogens; land use and development; and wildlife conservation. Research programs and approaches to policy evaluation that integrate biodiversity and human health can help unite public health and conservation goals. The relationship clearly requires more research, but these studies indicate that biodiversity protection may be as important to people on a local scale in their everyday lives as it is in remote protected ecosystems.

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