Chapter 11
Land Use Change and Human Health

Samuel S. Myers

Human activity is rapidly transforming our planet. The most pervasive changes to the landscape include deforestation, extension and intensification of agriculture, and livestock management, the construction of dams, irrigation projects, and roads, and rapidly spreading urbanization. In addition to the well-known environmental costs of these changes, each also has important health implications that are often less recognized. However, a growing number of studies that combine ecology and human health are demonstrating how these activities impact the emergence of new infectious diseases and alter the distribution of already recognized diseases.

There are a variety of mechanisms by which land use change may alter exposure to infectious disease (Table 11.1). These mechanisms include the alteration of: (1) biophysical conditions of habitats that can affect the density or presence of disease-related organisms; (2) exposure pathways, or the way organisms (including humans) interact with each other; (3) the genetics of pathogens; (4) the life cycles of pathogens and vectors; and (5) species composition within a community of organisms (Myers and Patz 2009). Infectious diseases which are transmitted by a vector (usually an arthropod), or have a non-human host or reservoir are particularly sensitive to these types of change (Wilson 2001; Eisenberg et al. 2007). Given that such diseases affect over half the human population, particularly the poor, alterations in their transmission rates can have significant impacts on human health and well-being (Lemon et al. 2008).

In this chapter, we discuss the major types of land use change and how these changes are known to impact exposure to infectious disease. In the chapter by Keesing and Ostfeld (this Volume), we discuss disease ecology, which explores how complex changes in whole communities of organisms are likely to alter disease exposure. Both chapters bring into focus the many ways that changing the natural

S.S. Myers (✉)
Department of Environmental Health, Harvard School of Public Health, Harvard Medical School, Landmark Center, 401 Park Drive, Boston, MA 02215, USA
e-mail: Sam_Myers@hms.harvard.edu

J.C. Ingram et al. (eds.), *Integrating Ecology and Poverty Reduction: Ecological Dimensions*, DOI 10.1007/978-1-4419-0633-5_11,

Table 11.1 Mechanisms of altered infectious disease exposure resulting from environmental change

Mechanism by which environmental change alters disease transmission	Examples	Diseases known to be impacted by this mechanism
Changes in density or identity of disease-related organisms	Deforestation or irrigation projects improve breeding habitat and survival of certain anopheline mosquitoes that transmit malaria in Africa, Latin America, and Asia. Deforestation in Cameroon favors one snail species over another, thereby increasing human exposure to pathogenic schistosomes. Sea surface warming and nutrient loading lead to proliferation of *Vibrio cholerae* and disease outbreaks.	Malaria, schistosomiasis, dengue, Japanese encephalitis, filariasis, trypansomiasis, leishmaniasis, cholera, plague, Rift Valley fever, dracunculosis, onchocerciasis, hantavirus, hemorrhagic viruses, Chagas disease, Oropouche/Mayaro virus harmful algal blooms
Changes in Exposure Pathways	Incursions into wildlife habitat can lead t o new exposure to zoonotic disease as seen in Ebola, simian retroviruses, and, possibly, HIV. Dense urban settlements with poor sanitation, waste disposal, or water treatment can lead to increased exposure to many diseases including diarrheal disease, dengue, and leptospirosis.	Malaria, trypanosomiasis, cryptosporidiosis, giardiasis, Ebola, simian retroviruses, HIV, dengue, filariasis, Chagas disease, plague, leptospirosis, typhus, diarrheal disease, food poisoning
Changing the environment in which organisms live creates genetic alterations which can increase disease transmission	Livestock management relying on extensive use of antibiotics in concentrated animal feeding operations (CAFOs) leads to the emergence of pathogens resistant to numerous antibiotics. Confinement of different animal species in wet markets or pig-duck farms can lead to genetic rearrangements resulting in increased virulence or altered infectivity.	Antibiotic resistant bacteria, influenza, SARS

Changes in life-cycle of vectors or pathogens	Deforestation causes increased ambient temperature in homes and breeding sites which shortens gonotrophic cycles, reduces development time, and increases survivalship of anopheline mosquitoes in Kenya.	Malaria
Changes in species composition of communities of organisms	Biodiversity loss in Northeastern forests of the United States increases exposure to Lyme Disease.	Lyme Disease, West Nile virus, malaria, hantavirus, Guanarito, Junin, Machupo, bartonellosis, Nipah virus, St. Louis encephalitis
	Altered species composition of wetlands in Belize in response to nutrient loading creates favorable habitat for a more effective malaria vector.	

world can impact disease exposure in unexpected ways. We hope it will become clear that understanding these ecological relationships is an important element of improving public health globally.

Tropical Deforestation

Widespread deforestation has been one of the most dramatic and biologically profound changes to our global landscape. Over the past 300 years, we have cut down between 7 and 11 million km^2 of forest—an area the size of the continental United States. Approximately, two million km^2 of natural "forest" in temperate and tropical regions are now highly managed plantations with significantly reduced biological diversity (Foley et al. 2005). Deforestation alters biological composition and complexity, soil dynamics, biogeochemical cycles, surface water chemistry, ambient air temperature, exposure to sunlight, and hydrological cycles. It creates forest edges which provides new habitat for a variety of disease vectors and often creates an active interface between human populations and forest-dwelling vectors and host species. Not surprisingly, it has impacts on infectious disease exposure, particularly to vector-borne and zoonotic diseases. Vector-borne diseases are caused by pathogens that are transmitted from one individual to another by an intermediary organism—usually an arthropod such as a mosquito or a tick. Common examples include malaria and Lyme disease. Zoonoses are diseases that exist in both human and non-human vertebrates and, therefore, have natural host reservoirs in the non-human community. Yellow fever (monkeys) and rabies (dogs, raccoons, foxes, skunks, etc.) are common examples. In this section, we focus on tropical deforestation because very little is known about the infectious disease threats of temperate deforestation, with the exception of the well-described relationship between forest fragmentation and increased risk for Lyme disease in temperate forests (LoGiudice et al. 2003).

Because of its global importance, extensive research has been conducted on the relationship between tropical deforestation and malaria. Here, we describe this research in some detail to illustrate the complexity of land use-disease relationships.

Of an estimated three billion people living in malaria-prone areas, approximately 500 million contract the disease annually, and roughly one million people die each year, mostly in Africa (Hay et al. 2005; Snow et al. 2005; Guerra et al. 2006a). Malaria is transmitted by a number of different species of *Anopheles* mosquito. In order to understand the effect of deforestation on malaria, it is necessary to know which species of mosquito are responsible for transmission in a given location and what their breeding habitats and feeding preferences are. Understanding the ecology of the species that transmit malaria locally, including their breeding habitat, feeding preferences, behavior, and environmental niche is essential to understanding the effects of deforestation on the spread of malaria. This, in turn, is essential to predicting the spread of the disease and developing effective control measures.

In the Americas, 4 out of 5 cases of malaria occur in the Amazon. While there are over 50 different *Anopheles* mosquito species in this region, only one appears to be an

important vector of malaria: *Anopheles darlingi* (Guerra et al. 2006b). *A. darlingi* prefers to breed in partly shaded pools with slightly acidic pH. Slash and burn land clearing and road building change the chemical composition of the soil, reduce shading, and often create small pools of water with ideal conditions for *A. darlingi*. As a result, deforestation favors breeding of this vector (Singer and de Castro 2001) and has been shown to increase malaria exposure in the Amazon (Tadei et al. 1998). In the Peruvian Amazon, investigators found that biting rates of *A. darlingi* in deforested areas were 278 times higher than biting rates in forested areas (Vittor et al. 2006). These findings supported earlier work showing strong associations between deforestation and malaria and epidemiological evidence of malaria surges following periods of deforestation.

In Africa, rather than a single malaria vector, there are four primary vectors: *A. gambiae*, *A. funestus*, *A. moucheti*, and *A. nili*. *Anopheles nili* and *A. moucheti* have more localized distributions and are considered "subsidiary" vectors. Of these four, *A. nili* is considered a localized forest species with a relatively limited role in transmission (Carnevale et al. 1992). As in the Amazon, deforestation increases habitat for the primary African malaria vectors; it is, therefore, not surprising that a number of studies have found associations between deforestation and increased malaria exposure in sub-Saharan Africa (Coluzzi et al. 1979; Coluzzi 1984, 1994; Cohuet et al. 2004). A recent review of these and other studies of the relationships between deforestation and malaria concluded that deforestation in sub-Saharan Africa tends to increase malaria transmission (Guerra et al. 2006b). Given the breeding preferences of the primary malaria vectors in Africa, this generalization is likely to hold, although exceptions may well be identified in specialized breeding areas.

Increasing habitat or breeding sites for mosquito species is not the only way that deforestation can increase malaria exposure. An elegant series of investigations by Afrane and colleagues (Afrane et al. 2005, 2006) has gone beyond counting the density of anopheline vectors in forested versus deforested areas. In experiments performed in the western Kenya highlands, they evaluated how deforestation may affect the lifecycle of *Anopheles* species through microclimatic change and showed that, by reducing shading, deforestation raises the average temperature in homes by 1.8°C and in nearby aquatic habitats by 4.8–6.1°C. These ambient temperature changes are associated with much shorter reproductive cycles (nearly 60% shorter), reduced larva-to-adult developmental time, and increased larval and adult survivorship all of which improve the vectorial capacity of the mosquitoes and increase exposure to malaria (Afrane et al. 2005, 2006). For example, as a result of increased ambient temperatures in deforested areas, *A. arabiensis* has a 49–55% higher adult life span and a reproductive rate about twice that in forested areas.

In addition, the alteration of microclimate through deforestation can also increase the geographic range of less abundant vectors. In the case of *A. arabiensis*, deforestation has been shown to facilitate the migration to higher elevations. Afrane and colleagues argue that the combination of deforestation and climate change may facilitate the establishment of *A. arabiensis* as an important malaria vector in the Kenya highlands (Afrane et al. 2007).

In Asia, the relationship between deforestation and malaria appears to be more complex. In part, this is because there are a wider variety of *Anopheles* species that are effective malaria vectors and that have more variable habitat preferences.

Unlike the Americas and Africa where higher rates of transmission occur after deforestation, much of the malaria transmission in Asia occurs in intact forested areas. Almost all of the malaria transmission in Bangladesh in 1989 occurred in forests (Sharma et al. 1991). In India in 1987, 7% of the human population lived in forested areas but contributed to 30% of the malaria cases in the country (Narasimham 1991). In fact, deforestation has driven down the density of important malaria vectors including *A. dirus*, *A. minimus*, and *A. barbirostris* in Thailand, Nepal, India, and Sri Lanka. However, it has also caused an increase in the density of alternative vectors including *A. fluviatilis*, *A. annularis*, *A. jamesii*, *A. nigeririmus*, *A. subpictus*, and *A. peditaeniatus* (Amerasinghe and Ariyasena 1990; Konradsen et al. 1990; Karla 1991; Taylor 1997). In a comprehensive review of over 60 studies of land use change and malaria, Yasuoka and Levins describe the unpredictable nature of the impacts of these complex changes. In Kanchanaburi, Thailand, widespread deforestation from 1986 to 1995 eliminated breeding sites for *A. dirus* and decreased malaria incidence. However, in northeast India, deforestation increased malaria transmission by replacing the historical vector *A. minimus* with *A. fluviatilis* that has since become more abundant (Yasuoka and Levins 2007). In Sri Lanka, deforestation has driven major malaria epidemics (Konradsen et al. 1990). While anopheline ecology is particularly complex in Asia, it conforms, nonetheless, to a general trend throughout the world: deforestation tends to reduce mosquito diversity and the surviving dominant species, for reasons that are not well understood, are almost always more effective vectors for malaria than earlier vectors (Molyneux et al. 2008).

Exposure to another disease, schistosomiasis, also appears to be related to deforestation. Schistosomiasis is a disease caused by parasitic worms (Schisotoma spp.) that spend part of their life cycle in freshwater snails and then leave the snails to penetrate the skin of people who enter contaminated water. The disease can damage liver, lungs, intestines, and bladder and infects roughly 200 million people. Deforestation changes the ecology of freshwater snail populations by increasing sunlight penetration, encouraging growth of vegetation, and changing water levels and flow rates. Many snail species do not survive these changes, but those which do tend to be better hosts for the parasitic worms (schistosomes) that cause this disease (Molyneux et al. 2008). In Cameroon, the upsurge in schistosomiasis following deforestation has been well described. Deforestation there led to the displacement of one type of freshwater snail, *Bulinus forskalii*, by another, *Bulinus truncatus*, better suited to cleared habitats. While *B. forskalii* hosted a type of schistosome that causes little illness in humans, *B. truncatus* is an effective host for *Schistosoma haematobium*, a primary cause of urinary tract Schistosomiasis (Southgate et al. 1976).

The effects of deforestation on other vector-borne diseases have been less well characterized. Deforestation in West Africa has expanded the range of both onchocerciasis (river blindness), which is spread by the bite of the black fly and yellow fever, a mosquito-borne virus (Cordellier 1991; Wilson et al. 2002; Patz and Confalonieri 2005). There is also evidence that deforestation has increased the incidence of cutaneous leishmaniasis (transmitted by the bite of the sandfly) in Latin America (Weigle et al. 1993; Desjeux 2001). However, further research is necessary to understand the full impacts of deforestation on exposure to a variety of vector-borne diseases.

Since nearly half the human population suffers from one or more vector-borne diseases this area represents a rich field for research with important consequences for improving human health (Lemon et al. 2008). Deforestation is one of the most pervasive features of global change driving dramatic biological, geochemical, and hydrological changes that will impact the dynamics of many insect-transmitted diseases. In the majority of diseases that have been studied, impacts associated with deforestation have increased disease exposure. However, regardless of whether deforestation has a positive or negative impact on disease exposure, understanding these relationships is essential in guiding disease surveillance, control, and mitigation efforts. The importance of understanding these relationships has been recently emphasized by the World Health Organization (WHO) as part of their emphasis on integrated vector management (2008b).

Not all health consequences of deforestation are related to infectious diseases however. Over the past decade, investigators have uncovered an important association between deforestation in the Amazon basin and "natural" mercury contamination, particularly of rivers. In watersheds that are hundreds of kilometers removed from the nearest gold mining operations, areas disturbed by deforestation have significantly higher mercury loads than upstream areas that remain intact (Veiga et al. 1994; Fostier et al. 2000). This "natural" contamination is thought to be caused by the release of mercury from burned trees and shrubs and increased soil erosion. The contamination has led to elevated mercury levels in fish and in the local people who eat them. Investigators have demonstrated neurological deficits in Amazonian forest-dwellers even at very low levels of mercury contamination (Lebel et al. 1996, 1998).

There are other, less direct, effects of deforestation on human health. At global scales, deforestation makes a significant contribution to climate change through the release of carbon from soils and forest biomass to the atmosphere and by decreasing carbon uptake from growing trees. Deforestation has contributed roughly one-quarter of the total rise in green house gases (GHG). As we will see in the chapter by Hess and Myers (this volume), the health impacts of climate change are quite significant.

In addition to climate regulation, forests play an important role in maintaining a reliable supply of clean fresh water. Forest litter absorbs water, filters it, and releases it slowly over time. Deforestation causes more rapid runoff, increased sediment loads, and poorer water quality. It can lead to flooding and landslides as well as increased incidence of water-borne disease. In 1998, upstream deforestation played an important role in the Yellow River flood disaster which killed more than 3,500 people, damaged over seven million houses, submerged 25 million hectares of farmlands and caused US$30 billion worth of damage (August 28 2001). In the same year, Hurricane Mitch killed nearly 10,000 people in Central America and left roughly one million people homeless. Areas with deforested hillsides and floodplains suffered disproportionate morbidity and mortality (Environmental Impacts of Hurricane Mitch 1999; Cockburn et al. 1999).

The complex and diverse pathways by which deforestation can impact human health provide an excellent example of the importance of ecology to public health. Without an understanding of ecology, it would be difficult to anticipate that changes in forest cover could have such profound impacts on disease exposure.

Crop Cultivation

Rapid human population growth coupled with economic development and increasing adoption of a western-style diet has driven dramatic increases in global grain and meat production. Since 1960, global food production has risen by roughly two and a half times (2005). Achieving these increases has required bringing more land into cultivation and pasturage. Roughly 40% of the planet's ice-free land surface has been converted to croplands or pasture (Foley et al. 2005). Pressure to increase yields-per-acre has also driven agricultural intensification with industrial fertilizers and pesticides, widespread irrigation, new crop varieties, and mechanization. As with deforestation, there are a wide variety of potential health impacts arising from these practices most of which are not well studied. However, an overview of some of the relationships that have been documented suggests the breadth and variety of health consequences resulting from these types of land use changes.

One way that crop cultivation and livestock management impacts health is by creating new ecological niches that favor disease vectors or hosts. In Trinidad, in the 1940s, the development of cacao plantations caused a major malaria epidemic. The mechanism driving this epidemic was the use of anagroforestry system where species of the *Erythrina* tree were used to provide shade and nitrogen to the cacao. However, the shade provided by the *Erythrina* trees also provided ideal habitat for epiphytic bromeliads which, in turn, created excellent breeding sites for *A. bellator*, the principal local malaria vector. The epidemic was not controlled until the *Erythrina* trees were reduced in number and plantation techniques were changed (Downs and Pittendrigh 1946; Yasuoka and Levins 2007). This example also serves to show the complexity of managing ecological relationships: agroforestry systems, such as the *Erythina* and cacao systems described above, have largely been promoted for their contribution to biodiversity conservation, soil conservation, and carbon sequestration functions amongst others, yet, these case studies demonstrate that there also can be unforeseen health consequences associated with these practices. Thus, conserving and managing a multi-functional landscape will require knowledge of the tradeoffs and synergies among ecosystem services and will require adaptive management to respond to unforeseen consequences resulting from land use decisions.

Numerous other relationships between agricultural cultivation and human health impacts have been documented. In Thailand, both cassava and sugarcane cultivation led to reductions in the density of *A. dirus* but created widespread breeding grounds for *A. minimus* with a resulting surge in malaria (Yasuoka and Levins 2007). In Cote d'Ivoire, cultivation of coffee and cacao plantations has been associated with exposure to African trypanosomiasis (sleeping sickness). The plantations create habitat for the tsetse fly vector and cause exposure by bringing agricultural workers into contact with the vector (Fournet et al. 2000). In a final example, the drainage and cultivation of papyrus swamps in highland Uganda appears to increase the risk of malaria. Households located near drained and cultivated swamps have higher ambient temperatures and more *A. gambiae* mosquitoes per household than households

in villages surrounding undisturbed papyrus swamps (Lindblade et al. 2000). As with deforestation, ecological changes associated with crop cultivation can create new habitat for disease vectors and new modes of human exposure, both of which can increase overall infectious disease exposure.

Agricultural practices can also impact health through contamination of waterways with pathogens and excess nutrients. In 1993, the largest water-borne disease outbreak ever recorded occurred in Milwaukee where over 400,000 people were infected with *Cryptosporidium parvum*, a protozoan that can cause severe diarrhea. The outbreak followed a period of heavy rainfall and runoff that contaminated Milwaukee's water supply despite new filtration and disinfection facilities and killed 54 people (Mac Kenzie et al. 1994). Heavy rainfall and runoff has been associated with other cryptosporidiosis outbreaks. *Cryptosporidium* oocysts are shed in the feces of many animals including ruminants like cows and sheep. They are very small (roughly 3 μm), pervasive, and are not easily filtered from water. Investigators found that 64% of farms in Pennsylvania had at least one cow infected with *Cryptosporidium* and on 44% of the farms, all bovine stool samples were positive. On these farms, the cattle had full access to waterways that could be contaminated by their feces (Graczyk et al. 2000). This combination of land clearing and grazing ruminants with no buffer zones to protect waterways provides the ideal ecological conditions for human infection by this parasite.

In addition to pathogens, agricultural runoff contains high concentrations of nutrients, particularly fixed nitrogen which is naturally limiting in most terrestrial environments. Overall, human activity, adds at least as much fixed nitrogen to the terrestrial environment as all natural sources combined (Vitousek and Mooney 1997). Because fixed nitrogen is a critical and rate-limiting nutrient in many ecosystems, its widespread addition has profound impacts on these systems. In marine and freshwater environments, nutrient enrichment is responsible for a rapidly increasing number of harmful algal blooms. These blooms are caused by a wide variety of algae, causing massive fish kills, shellfish poisonings, disease and death of marine mammals, and human morbidity and mortality. In the United States alone, roughly 60,000 individual cases and clusters of human intoxication caused by algal blooms occur annually. Health impacts range from acute neurotoxic disorders and death to subacute and chronic disease. The cost of HABs in the US over a 15-year period was estimated to exceed $400 million (2001). Nutrient enrichment of coastal waters is also likely to play a role in cholera outbreaks. Plankton blooms stimulated by warm temperatures and increased nutrient levels help to transform the cholera bacteria, *Vibrio cholera*, from a quiescent to an infectious state (Ezzell 1999; Colwell and Huq 2001). Unfortunately, this problem is likely to increase since agricultural ecologists anticipate a global increase in nitrogen and phosphorus application of roughly 250% from current levels in order to meet projected food demand by 2050 (Tilman 2001).

Nutrient enrichment of waterways from agricultural runoff can lead to increased risk of parasitic and infectious diseases through other types of ecological change as well. A recent review of the literature included 34 studies involving 41 different species of pathogens on 6 continents. The authors concluded that in 95% of observations

(51 of 55), nutrient enrichment *increased* exposure to pathogens (McKenzie and Townsend 2007). In India, for example, extensive use of synthetic fertilizers in rice fields has been associated with increased exposure to Japanese encephalitis. Elevated nitrogen in these rice fields is associated with increases in the density of mosquito larvae, presumably because of increased growth of the microorganisms which are the primary food source for these larvae (Victor and Reuben 2000; Sunish and Reuben 2001). Similar associations between nutrient loading of surface water and increased concentrations of mosquito larvae have been shown for malaria vectors in Mexico (Rejmankova et al. 1991), Belize (Rejmankova et al. 2006) and Taiwan (Teng et al. 1998) and for *Culex* and *Aedes* species of mosquito which transmit La Crosse encephalitis, Japanese encephalitis and West Nile virus (Walker et al. 1991; Sunish and Reuben 2001). As with deforestation, this is a rich field for future research given the pervasiveness of nutrient loading, the importance of the diseases likely to be impacted, and the fact that most of these relationships have not yet been described.

A final human health impact of nutrient loading is direct exposure to nitrogenous compounds in air and water. Nitrogen from synthetic fertilizers contributes to the formation of nitrogen oxides, which in turn lead to the production of ground-level ozone (O_3). Nitrogen oxides in the atmosphere are also an important driver of particulate air pollution. Both O_3 and nitrogen oxides contribute to respiratory disease (both chronic and acute) and cardiovascular disease. In addition, agricultural application of nitrogen to land surfaces leads to contamination of groundwater with nitrates. The World Health Organization (WHO) maximum standard of nitrate in safe drinking water is 10 ppm. Globally, this standard is often exceeded. Even in the USA where strict drinking water legislation applies, 10–20% of groundwater sources may exceed 10 ppm. The potential health effects of excess nitrate in drinking water include reproductive problems, methemoglobinemia (blue-baby syndrome), and cancer (Townsend et al. 2003).

Livestock Management

Livestock and wild animal management practices also have a series of important health consequences. In these cases, the mechanism for altered infectious disease exposure is often genetic change in pathogens resulting from a variety of livestock management practices. The widespread use of antibiotics for livestock has contributed to rapidly increasing microbial resistance. Resistant strains of *Campylobacter*, *Salmonella*, and *Escherichia coli* which can cause serious human infections have all been traced to the use of antibiotics in intensive livestock agriculture (Patz and Confalonieri 2005). A variety of other emerging or resurging infectious diseases are associated with livestock management practices as well. Pandemic influenza in humans is thought to result from genetic exchange among the strains of influenza virus in wild and domestic birds, and pigs. Close confinement of these animals in proximity to each other, for example in Asian "wet markets," and in pig-duck farms in China, fosters this type of genetic exchange (Daily and Ehrlich 1996).

The SARS epidemic is likely to have resulted from similar crowding of animals in live-animal markets in China. In this case, the species at the center of the epidemic were horseshoe bats and palm civet cats as amplifying hosts with a possible role for raccoon dogs, and Chinese ferret badgers as well. Most of the early cases of SARS were among people who worked with the sale or handling of these animals (Shi and Hu 2008).

A more complete understanding of the ecology of this type of zoonotic disease transmission could lead to less risky livestock management practices. Most of the infectious diseases that are now endemic in human populations originated in non-human populations. This includes the major killers of humanity—smallpox, tuberculosis, influenza, malaria, measles, cholera, and plague (Diamond 1999). Practices which bring new populations of wild or domestic animals into close proximity with each other or human populations are likely to be a major source of new emerging diseases in the future (Weiss and McMichael 2004).

A final example of livestock management practices leading to resurgence of infectious disease comes from the mountainous regions of Yunnan Province, China. There, an economic development project tried to raise local incomes by giving villagers cows. Cattle are an important reservoir of *Schistosoma japonicum*, the agent responsible for schistosomiasis. As they spread throughout the region, they shed schistosome eggs into waterways where they infect snails that serve as the intermediate host. As a result, schistosomiasis rates surged, infecting up to 30% of some villages particularly impacting those villagers that owned and managed cattle (Jiang et al. 1997).

Dams and Irrigation Projects

Large dams and irrigation projects have become another pervasive feature of the human-dominated landscape. By the end of the twentieth century, there were over 45,000 large dams and more than 800,000 small dams in over 150 countries. About half of these were built exclusively or primarily for irrigation, and about one-third of the world's irrigated cropland relies on dams (World Commission on Dams 2000; Keiser et al. 2005).

There is no doubt that these dams make an essential contribution to global food production, provide reliable water supplies, and are a massive source of clean power generation. Irrigated croplands represent only one-fifth of total agricultural lands, but produce roughly 40% of total global agricultural yield. Dams are estimated to contribute 12–16% of world food production (2000). However, the ecological and health impacts of dams and irrigation can be both devastating and far reaching.

Dams and irrigation projects also change local ecology and create new favorable habitat for the transmission of a variety of vector-borne diseases. As it is with deforestation, the association with malaria is one of the best documented health impacts of dams and irrigation projects. A study in northern Ethiopia has shown a sevenfold increase in malaria in villages within 3 km of microdams compared with control

villages 8–10 km distant (Ghebreyesus et al. 1999). In India, there was a surge of "irrigation malaria" in the 1990s after poorly evaluated irrigation projects improved breeding conditions for the dominant malaria vector, *Anopheles culcifacies*. Malaria became endemic and widespread in a population of roughly 200 million people as a result (Sharma 1996). In general, dams and irrigation projects constructed in endemic areas tend to increase breeding habitat and transmission of malaria (World Commission on Dams 2000; Keiser et al. 2005). Agricultural projects that combine deforestation with dams and irrigation can be a particularly potent combination for increasing malaria exposure given their combined effects on breeding habitat and microclimate.

In the Nile delta area of Egypt, prevalence of lymphatic filariasis (elephantiasis) rose from <1% in 1965 to >20% after construction of the Aswan High Dam and subsequent irrigation projects. This surge resulted from increased surface and subsurface moisture that created improved breeding sites for *Culex pipiens*, the mosquito vector of this disease (Harb et al. 1993; Thompson et al. 1996). In Ghana, a different vector is primarily responsible for transmission of filariasis, *Anopheles gambiae*. However, similar dynamics are observed. Rates of infection, worm load, annual bites per person, and annual transmission rates were all found to be higher in irrigated areas than in communities without irrigation (Appawu et al. 2001).

Filariasis was not the only disease to surge with the construction of the Aswan Dam. Because of the creation of extensive new habitat for *B. truncatus*, a freshwater snail which is an excellent intermediate host for *Schistosoma haematobium*, prevalence of *S. haematobium* infection in Upper and Middle Egypt rose from about 6% before construction of the dam to nearly 20% in the 1980s. In Lower Egypt, intestinal schistosomiasis rose to an even greater extent (Malek 1975; Cline et al. 1989; Molyneux et al. 2008). In the Tana river region of Kenya, the Hola irrigation project led to the introduction of snail vectors where they had never been before. Between 1956, when the project began, and 1966, the prevalence of urinary schistosomiasis in children in the region went from 0 to 70%. By 1982, it was 90% (Mutero 2002). Around the world, the rapid proliferation of dams and irrigation projects has generated new habitat for freshwater snails well adapted to these environments and to hosting schistosomes resulting in a global surge in schistosomiasis infection. Propagation of Rift Valley Fever, leishmaniasis, dracunculosis, onchocerciasis, and Japanese encephalitis has also been associated with these projects (Jobin 1999; Patz and Confalonieri 2005).

Roads

Construction of roads can provide edge habitat for disease vectors as well as create pools of water that can be excellent breeding sites for mosquitoes. Roads built for transportation, access to mines, or construction of pipelines can become entry points for settlers. In these situations, the combination of deforestation and exposure of a non-immune population to local vector-borne or zoonotic disease can lead to new epidemics. With malaria, this phenomenon is referred to as "frontier malaria."

Road building has also been implicated as an important factor in the penetration of human populations into previously undisturbed wildlife habitat. This mixing of previously isolated human and non-human populations can lead to exposure to new zoonoses. The bushmeat trade which leads to handling, slaughtering, and consuming wild animal species, particularly in Africa and Asia, further increases the risk of human exposure to new pathogens.

In particular, bushmeat hunting may provide opportunities for exchange of pathogens between humans and non-human primates. In Central Africa, 1–3.4 million tons of bushmeat are harvested annually (Fa and Peres 2001). In West Africa, a large share of protein in the diet comes from bushmeat. In West Africa, the bushmeat harvest includes a large numbers of primates, facilitating interspecies disease transfer. The "Taxonomic Transmission Rule" states that the probability of successful cross-species infection increases the more closely hosts are genetically related, since related hosts are more likely to share susceptibility to the same range of potential pathogens (Wolfe et al. 2000).

Recently, infection with simian foamy virus, a retrovirus that is endemic in most Old World primates, was demonstrated in hunters who reported direct contact with blood or body fluid of non-human primates. This finding provides additional support for already compelling hypothesis that the retrovirus causing HIV/AIDS was likely a mutated simian virus contracted through bushmeat hunting (Hahn et al. 2000; Wolfe et al. 2004). It is likely that Ebola virus infection in human populations also had its origin in bushmeat hunting.

Of the wave of emerging infectious diseases over the past several decades, three quarters are zoonotic (Taylor et al. 2001). This fact is consistent with the theory that new incursions of human populations into previously isolated wildlife habitat, and animal markets that bring live wildlife into close proximity with other human and non-human species may represent important sources of new human infectious diseases.

Urbanization

A final pervasive form of land use change has been the rapid, global development and expansion of cities. Over the past two centuries, the proportion of people living in cities or large towns has grown from approximately 5–50% and continues to climb (Global Environmental Change and Human Health 2007). Between 1960 and 1980, the urban population in developing countries more than doubled. By 2025, urban population in developing countries is expected to account for well over half the global population (Knudsen and Slooff 1992).

Widespread urbanization can result in numerous direct ecological consequences that affect human health. As with deforestation, agriculture, dams and irrigation systems, urbanization creates new habitat for disease hosts and vectors while changing human exposure patterns. Much of the rapid urbanization occurring today is

taking place in urban or periurban slums with few services for clean water provision, sewage disposal, solid waste management, or quality housing. Pools of contaminated water, water containers kept in homes, tires and other refuse capable of holding water, and piles of municipal waste all create excellent habitat for a variety of rodent hosts and arthropod vectors, particularly those which transmit dengue, malaria, filariasis, Chagas disease, plague, and typhus. In addition, rural to urban migration brings people from different disease endemic regions together in high density providing a source for new infection as well as non-immune hosts. A final contributing factor to the spread of vector-borne disease in slums is the poor quality housing which does not provide an effective barrier to mosquitoes, rodents, or fleas.

Dengue fever provides a good example of a vector that is well adapted to urban habitat. Over recent decades there has been a tremendous surge in dengue cases as it has spread out of South-east Asia and the Pacific and become endemic throughout the tropics. Dengue is the most common mosquito-borne viral disease in the world with roughly 50 million cases in over 100 countries each year (2008a). It is transmitted by the bite of infected *Aedes* mosquitoes, primarily *Aedes aegypti*. These mosquitoes prefer to feed on humans over other animals and usually live close to human dwellings. They breed in man-made containers like earthenware jugs, tires, metal drums, discarded plastic food containers, and other items that collect rainwater. These characteristics make them highly effective at adapting to urban areas (Daily and Ehrlich 1996; Mackenzie et al. 2004). Relatively simple, ecologically based solutions, such as eliminating rainwater holding containers that serve as the breeding habitat for this species have proven effective control measures.

A second consequence of rapid urbanization in slums and squatter settlements is lack of access to safe drinking water or sanitation. Current projections are that, if efforts to provide water and sanitation to the underserved continue at the current rate, more than 692 million people will live without basic sanitation and 240 million without improved sources of drinking water, in urban areas in 2015 (Norstrom 2007). Particularly in crowded urban conditions, the exposure to infectious disease resulting from both contaminated drinking water and inadequate sanitation is significant. Diarrhea alone, caused primarily by contaminated drinking water, causes around 2.2 million deaths each year, about 4% of all global mortality. Poor sanitation can also lead to outbreaks of leptospirosis as was observed in San Salvador between March and November of 1996, where investigators identified over 300 cases of leptospirosis—a disease that is transmitted by direct exposure to rodents or an environment contaminated by their urine. They found the highest incidence rates among the urban poor where exposure to rats and to flood waters contaminated by rat urine was likely to be highest. The disease had a 15% case fatality rate. Investigators noted an epidemiological shift in leptospirosis transmission from rural to urban areas and postulated that urban slums with large rodent populations create an environment in which heavy rains, which can drive rodents out of burrows and lead to contaminated flood waters, will trigger these epidemics (Ko et al. 1999).

Finally, rapidly growing urban areas cast a footprint far beyond their local boundaries. Wastewater from cities is often poorly treated and can expose downstream

communities to infectious disease. It can also lead to outbreaks of harmful algal blooms and shellfish poisoning as it contaminates coastal marine environments (Rose et al. 2001). Urban emissions contribute to local air pollution but also have impacts regionally (acid rain, for example) and globally (climate change). Air pollution, primarily from urban centers in Asia, is causing the formation of atmospheric brown clouds which have become so extensive that they are impacting regional weather patterns, reducing agricultural productivity, and increasing glacial melting in addition to causing extensive deaths from cardiorespiratory disease (Ramanathan et al. 2008). Urban demand for food, building materials, fiber, and other ecosystem services drives many of the land use trends discussed earlier. Whether these demands are reduced by concentrating people in urban environments and creating efficiencies of scale or increased because of transportation requirements and waste has not been well studied.

Conclusion

Human activity has changed the face of the global landscape. These changes in land use and cover have, in turn, altered the dynamics of infectious disease transmission in numerous ways. They have created new habitat and breeding sites for disease vectors which, in many cases, favor disease transmission. They have altered exposure pathways by changing the way organisms (including humans) interact with each other. They have led to changes in the genetics, and thereby, the virulence, and infectiousness of pathogens. They have also changed the lifecycle of pathogens and vectors and the species composition of whole communities of disease-relevant organisms. Not surprisingly, these changes have occurred coincident with a rise in new or reemerging infectious diseases. These are not merely academic concerns; the diseases impacted by these changes represent a large percentage of the total global disease burden.

For practitioners working to improve human wellbeing, understanding how large-scale changes in the landscape can create new dynamics in disease transmission is important. This type of understanding can help us anticipate that, despite their potential economic benefits, there may be significant health effects from environmental change such as dam building, irrigation projects, the use of fertilizers, and the clearing of forests. Knowledge of these interactions can and should help guide surveillance and mitigation efforts. The goal of the practitioner must be to maximize the benefits of these projects while minimizing the negative health impacts. Accomplishing that goal requires an understanding of the underlying ecological relationships between the disease, its vectors, and the environmental niches of both. It also requires active use of health impact assessments (HIAs) as discussed in our chapter on climate change and health.

Finally, it is important, wherever possible, to develop a fine-grained understanding of local field conditions and of the complex relationships between communities of organisms, (including pathogens), habitat, and human populations.

Ecological understanding of the community interactions between diseases, hosts, vectors and the afflicted not only serve to help us understand how our alteration of the environment impacts the spread of these diseases, but can be a powerful tool in preventing, immobilizing, and controlling these diseases as will be discussed in Chap. 13 (this volume) on disease ecology.

References

1999. Environmental Impacts of Hurricane Mitch. Environ Health Perspect.

2000. Dams and Development: A New Framework for Decision-Making. World Commission on Dams, London.

2001. Oceans and Human Health Roundtable Report. National Institute of Environmental Health Sciences and National Science Foundation, Research Triangle Park, North Carolina.

Ecosystems and Human Well-being: Synthesis. Millenium Ecosystem Assessment. Island Press, Washington, DC.

2007. Global Environmental Change and Human Health (2007) Science Plan and Implementation Strategy. Earth System Science Partnership (Diversitas, IGBP, IHDP, and WCRP) Report No.4; Global Environmental Change and Human Health Report No.1.

2008a. Fact Sheet No. 117: Dengue and dengue hemorhagic fever. Page WHO Fact Sheet *in* W. H. O. M. Centre, editor. Fact Sheet No. 117. World Health Organization, Geneva.

2008b. Malaria control: the power of integrated action. Health and Environment Linkages Policy Series. World Health Organization, Geneva, Switzerland.

August 28, 2001. Asian Development Bank News Release 091/01: Novel Approach To Managing Floods On People's Republic Of China's Yellow River.*in* A. D. Bank, editor. New Release 091/01.

Afrane, Y. A., B. W. Lawson, A. K. Githeko, and G. Yan. 2005. Effects of microclimatic changes caused by land use and land cover on duration of gonotrophic cycles of Anopheles gambiae (Diptera: Culicidae) in western Kenya highlands. J Med Entomol **42**:974–980.

Afrane, Y. A., G. Zhou, B. W. Lawson, A. K. Githeko, and G. Yan. 2006. Effects of microclimatic changes caused by deforestation on the survivorship and reproductive fitness of Anopheles gambiae in western Kenya highlands. Am J Trop Med Hyg **74**:772–778.

Afrane, Y. A., G. Zhou, B. W. Lawson, A. K. Githeko, and G. Yan. 2007. Life-table analysis of Anopheles arabiensis in western Kenya highlands: effects of land covers on larval and adult survivorship. Am J Trop Med Hyg **77**:660–666.

Amerasinghe, F. P. and T. G. Ariyasena. 1990. Larval survey of surface water-breeding mosquitoes during irrigation development in the Mahaweli Project, Sri Lanka. J Med Entomol **27**:789–802.

Appawu, M. A., S. K. Dadzie, A. Baffoe-Wilmot, and M. D. Wilson. 2001. Lymphatic filariasis in Ghana: entomological investigation of transmission dynamics and intensity in communities served by irrigation systems in the Upper East Region of Ghana. Tropical Medicine & International Health **6**:511–516.

Carnevale, P., G. Le Goff, J. C. Toto, and V. Robert. 1992. Anopheles nili as the main vector of human malaria in villages of southern Cameroon. Med Vet Entomol **6**:135–138.

Cline, B. L., F. O. Richards, M. A. el Alamy, S. el Hak, E. Ruiz-Tiben, J. M. Hughes, and D. F. McNeeley. 1989. 1983 Nile Delta schistosomiasis survey: 48 years after Scott. Am J Trop Med Hyg **41**:56–62.

Cockburn, A., J. St Clair, and K. Silverstein. 1999. The Politics of "Natural" Disaster: Who Made Mitch So Bad? International Journal of Health Services **29**:459–462.

Cohuet, A., F. Simard, C. S. Wondji, C. Antonio-Nkondjio, P. Awono-Ambene, and D. Fontenille. 2004. High malaria transmission intensity due to Anopheles funestus (Diptera: Culicidae) in a village of savannah-forest transition area in Cameroon. J Med Entomol **41**:901–905.

Coluzzi, M. 1984. Heterogeneities of the malaria vectorial system in tropical Africa and their significance in malaria epidemiology and control. Bull World Health Organ **62 Suppl**:107–113.
Coluzzi, M. 1994. Malaria and the Afrotropical ecosystems: impact of man-made environmental changes. Parassitologia **36**:223–227.
Coluzzi, M., A. Sabatini, V. Petrarca, and M. A. Di Deco. 1979. Chromosomal differentiation and adaptation to human environments in the Anopheles gambiae complex. Trans R Soc Trop Med Hyg **73**:483–497.
Colwell, R. and A. Huq. 2001. Marine Ecosystems and Cholera. Hydrobiologia **460**:141–145.
Cordellier, R. 1991. [The epidemiology of yellow fever in Western Africa]. Bull World Health Organ **69**:73–84.
Daily, G., C. and P. Ehrlich, R. 1996. Global Change and Human Susceptibility to Disease. Annual Review of Energy and the Environment **21**:125–144.
Desjeux, P. 2001. The increase in risk factors for leishmaniasis worldwide. Trans R Soc Trop Med Hyg **95**:239–243.
Diamond, J. 1999. Guns, Germs, and Steel. W.W.Norton & Co., New York, NY.
Downs, W., G. and C. Pittendrigh, S. 1946. Bromeliad malaria in Trinidad, British West Indies. American Journal of Tropical Medicine **26**:47–66.
Eisenberg, J. N., M. A. Desai, K. Levy, S. J. Bates, S. Liang, K. Naumoff, and J. C. Scott. 2007. Environmental determinants of infectious disease: a framework for tracking causal links and guiding public health research. Environ Health Perspect **115**:1216–1223.
Ezzell, C. 1999. It Came from the Deep. Scientific American **280**:22.
Fa, J., E and C. Peres, A. 2001. Game vertebrate extraction in African and Neotropical forests: an intercontinental comparison. Pages 203–241 *in* J. Reynolds, D, G. Mace, M, K. Redford, H, and J. Robinson, G, editors. Conservation of exploited species. Cambridge University Press, Cambridge, U.K.
Foley, J. A., R. Defries, G. P. Asner, C. Barford, G. Bonan, S. R. Carpenter, F. S. Chapin, M. T. Coe, G. C. Daily, H. K. Gibbs, J. H. Helkowski, T. Holloway, E. A. Howard, C. J. Kucharik, C. Monfreda, J. A. Patz, I. C. Prentice, N. Ramankutty, and P. K. Snyder. 2005. Global consequences of land use. Science **309**:570–574.
Fostier, A. H., M. C. Forti, J. R. Guimaraes, A. J. Melfi, R. Boulet, C. M. Espirito Santo, and F. J. Krug. 2000. Mercury fluxes in a natural forested Amazonian catchment (Serra do Navio, Amapa State, Brazil). Sci Total Environ **260**:201–211.
Fournet, F., S. Traore, A. Prost, E. Cadot, and J. P. Hervouet. 2000. Impact of the development of agricultural land on the transmission of sleeping sickness in Daloa, Cote d'Ivoire. Ann Trop Med Parasitol **94**:113–121.
Ghebreyesus, T. A., M. Haile, K. H. Witten, A. Getachew, A. M. Yohannes, M. Yohannes, H. D. Teklehaimanot, S. W. Lindsay, and P. Byass. 1999. Incidence of malaria among children living near dams in northern Ethiopia: community based incidence survey. BMJ **319**:663–666.
Graczyk, T. K., B. M. Evans, C. J. Shiff, H. J. Karreman, and J. A. Patz. 2000. Environmental and Geographical Factors Contributing to Watershed Contamination with Cryptosporidium parvum Oocysts. Environmental Research **82**:263–271.
Guerra, C. A., R. W. Snow, and S. I. Hay. 2006a. Defining the global spatial limits of malaria transmission in 2005. Adv Parasitol **62**:157–179.
Guerra, C. A., R. W. Snow, and S. I. Hay. 2006b. A global assessment of closed forests, deforestation and malaria risk. Ann Trop Med Parasitol **100**:189–204.
Hahn, B. H., G. M. Shaw, K. M. De Cock, and P. M. Sharp. 2000. AIDS as a zoonosis: scientific and public health implications. Science **287**:607–614.
Harb, M., R. Faris, A. M. Gad, O. N. Hafez, R. Ramzy, and A. A. Buck. 1993. The resurgence of lymphatic filariasis in the Nile delta. Bull World Health Organ **71**:49–54.
Hay, S. I., C. A. Guerra, A. J. Tatem, P. M. Atkinson, and R. W. Snow. 2005. Urbanization, malaria transmission and disease burden in Africa. Nat Rev Microbiol **3**:81–90.
Jiang, Z., Q. S. Zheng, X. F. Wang, and Z. H. Hua. 1997. Influence of livestock husbandry on schistosomiasis transmission in mountainous regions of Yunnan Province. Southeast Asian J Trop Med Public Health **28**:291–295.

Jobin, W. 1999. Dams and Disease: Ecological Design and Health Impacts of Large Dams, Canals, and Irrigation Systems. E&FN Spon, London.

Karla, N., L. 1991. Forest Malaria Vectors in India: Ecological Charcteristics and Epidemiological Implications. Pages 93–114 *in* V. Sharma, P. and A. Kondrashin, V, editors. Forest Malaria in Southeast Asia. World Health Organization/Medical Research Council, New Delhi, India.

Keiser, J., M. C. De Castro, M. F. Maltese, R. Bos, M. Tanner, B. H. Singer, and J. Utzinger. 2005. Effect of irrigation and large dams on the burden of malaria on a global and regional scale. Am J Trop Med Hyg **72**:392–406.

Knudsen, A. B. and R. Slooff. 1992. Vector-borne disease problems in rapid urbanization: new approaches to vector control. Bull World Health Organ **70**:1–6.

Ko, A. I., M. G. Reis, C. M. R. Dourado, J. W. D. Johnson, and L. W. Riley. 1999. Urban epidemic of severe leptospirosis in Brazil. The Lancet **354**:820–825.

Konradsen, F., F. Amerasinghe, P., W. van der Hoek, and P. Amerasinghe, H. 1990. Malaria in Sri Lanka, Current Knowledge on Transmission and Control. International Water Management Institute, Battaramulla, Sri Lanka.

Lebel, J., D. Mergler, F. Branches, M. Lucotte, M. Amorim, F. Larribe, and J. Dolbec. 1998. Neurotoxic effects of low-level methylmercury contamination in the Amazonian Basin. Environ Res **79**:20–32.

Lebel, J., D. Mergler, M. Lucotte, M. Amorim, J. Dolbec, D. Miranda, G. Arantes, I. Rheault, and P. Pichet. 1996. Evidence of early nervous system dysfunction in Amazonian populations exposed to low-levels of methylmercury. Neurotoxicology **17**:157–167.

Lemon, S., M, F. Sparling, P, M. Hamburg, A, D. Relman, A, E. Choffnes, R, and A. Mack, editors. 2008. Vector-Borne Diseases: Understanding the Environmental, Human Health, and Ecological Connections. National Academies Press, Washington, DC.

Lindblade, K. A., E. D. Walker, A. W. Onapa, J. Katungu, and M. L. Wilson. 2000. Land use change alters malaria transmission parameters by modifying temperature in a highland area of Uganda. Trop Med Int Health **5**:263–274.

LoGiudice, K., R. S. Ostfeld, K. A. Schmidt, and F. Keesing. 2003. The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. Proc Natl Acad Sci USA **100**:567–571.

Mac Kenzie, W. R., N. J. Hoxie, M. E. Proctor, M. S. Gradus, K. A. Blair, D. E. Peterson, J. J. Kazmierczak, D. G. Addiss, K. R. Fox, J. B. Rose, and et al. 1994. A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. N Engl J Med **331**:161–167.

Mackenzie, J. S., D. J. Gubler, and L. R. Petersen. 2004. Emerging flaviviruses: the spread and resurgence of Japanese encephalitis, West Nile and dengue viruses. Nat Med **10**:S98-109.

Malek, E., A. 1975. Effect of Aswan High Dam on prevalence of schistosomiasis in Egypt Tropical and Geographical Medicine **27**:359–364.

McKenzie, V., J and A. Townsend, R. 2007. Parasitic and Infectious Disease Responses to Changing Global Nutrient Cycles. EcoHealth **4**:384–396.

Molyneux, D., R. S. Ostfeld, A. Bernstein, and E. Chivian. 2008. Ecosystem Disturbance, Biodiversity Loss, and Human Infectious Disease. Pages 287–323 *in* E. Chivian and A. Bernstein, editors. Sustaining Life: How Human Health Depends on Biodiversity. Oxford University Press, Oxford; New York.

Mutero, C. M. 2002. Health impact assessment of increased irrigation in the Tana River Basin, Kenya.*in* I. W. M. Institute, editor. The Changing Face of Irrigation in Kenya: Opportunities for anticipating change in Eastern and Southern Africa. International Water Management Institute, Colombo.

Myers, S. S. and J. Patz. 2009. Emerging threats to human health from global environmental change. Annual Review of Environment & Resources **34**:223–252.

Narasimham, M., V,V,L. 1991. Perspective of forest malaria in India. World Health Organization, New Delhi, India.

Norstrom, A. 2007. Planning for drinking water and sanitation in peri-urban areas. 21.

Patz, J. A. and U. E. C. Confalonieri. 2005. Human Health: Ecosystem Regulation of Infectious Diseases. Pages 391–415 *in* R. M. Hassan, R. Scholes, and N. Ash, editors. Millennium

Ecosystem Assessment (Program). Condition and Trends Working Group. Ecosystems and human well-being : current state and trends : findings of the Condition and Trends Working Group of the Millennium Ecosystem Assessment. Island Press, Washington, DC.

Ramanathan, V., M., H. Agrawal, M. Akimoto, S. Aufhammer, and L. Devotta. 2008. Atmospheric Brown Clouds: Regional Assessment Report with Focus on Asia., United Nations Environment Program, Nairobi, Kenya.

Rejmankova, E., J. Grieco, N. Achee, P. Masuoka, K. Pope, D. Roberts, and R. Higashi. 2006. Freshwater community interactions and malaria. Page 227 *in* S. Collinge, K and C. Ray, editors. Disease ecology. Oxford University Press, Oxford.

Rejmankova, E., H. M. Savage, M. Rejmanek, J. I. Arredondo-Jimenez, and D. R. Roberts. 1991. MULTIVARIATE ANALYSIS OF RELATIONSHIPS BETWEEN HABITATS, ENVIRONMENTAL FACTORS AND OCURENCE OF ANOPHELINE MOSQUITO LARVAE ANOPHELES ALBIMANUS AND A. PSEUDOPUNCTIPENNIS IN SOUTHERN CHIAPAS, MEXICO. Journal of Applied Ecology **28**:827–841.

Rose, J. B., P. R. Epstein, E. K. Lipp, B. H. Sherman, S. M. Bernard, and J. A. Patz. 2001. Climate variability and change in the United States: potential impacts on water- and foodborne diseases caused by microbiologic agents. Environ Health Perspect **109 Suppl 2**:211–221.

Sharma, V., P., C. Prasittisuk, and A. Kondrashin, V. 1991. Magnitude of forest related malaria in the WHO Southeast Asia region., World Health Organization, New Delhi, India.

Sharma, V. P. 1996. Re-emergence of malaria in India. Indian J Med Res **103**:26–45.

Shi, Z. and Z. Hu. 2008. A review of studies on animal reservoirs of the SARS coronavirus. Virus Research **133**:74–87.

Singer, B. H. and M. C. de Castro. 2001. Agricultural colonization and malaria on the Amazon frontier. Ann N Y Acad Sci **954**:184–222.

Snow, R. W., C. A. Guerra, A. M. Noor, H. Y. Myint, and S. I. Hay. 2005. The global distribution of clinical episodes of Plasmodium falciparum malaria. Nature **434**:214–217.

Southgate, V., H. Wijk, and C. Wright. 1976. Schistosomiasis in Loum, Cameroun: Schistosoma haematobium, S. intercalatum, and their natural hybrid. Zeitschrift fur Parasitenkund **49**:149–159.

Sunish, I. P. and R. Reuben. 2001. Factors influencing the abundance of Japanese encephalitis vectors in ricefields in India - I. Abiotic. Medical and Veterinary Entomology **15**:381–392.

Tadei, W. P., B. D. Thatcher, J. M. Santos, V. M. Scarpassa, I. B. Rodrigues, and M. S. Rafael. 1998. Ecologic observations on anopheline vectors of malaria in the Brazilian Amazon. Am J Trop Med Hyg **59**:325–335.

Taylor, D. 1997. Seeing the forests for the more than the trees. Environ Health Perspect **105**:1186–1191.

Taylor, L. H., S. M. Latham, and M. E. Woolhouse. 2001. Risk factors for human disease emergence. Philos Trans R Soc Lond B Biol Sci **356**:983–989.

Teng, H., J, Y. Wu, L, S. Wang, J, and C. Lin. 1998. Effects of environmental factors on abundance of Anopheles minimus larvae and their seasonal fluctuations in Taiwan. Environmental Entomology **27**:324–328.

Thompson, D. F., J. B. Malone, M. Harb, R. Faris, O. K. Huh, A. A. Buck, and B. L. Cline. 1996. Bancroftian filariasis distribution and diurnal temperature differences in the southern Nile delta. Emerg Infect Dis **2**:234–235.

Tilman, D. 2001. Forecasting Agriculturally Driven Global Environmental Change. Science **292**:281–284.

Townsend, A., R, R. Howarth, W, F. Bazzaz, A, M. Booth, S, C. Cleveland, C, S. Collinge, K, A. P. Dobson, P. Epstein, R, E. Holland, A, D. Keeney, R, M. Mallin, A, C. Rogers, A, P. Wayne, and A. Wolfe, H. 2003. Human health effects of a changing global nitrogen cycle. Frontiers in Ecology and the Environment **1**:240–246.

Veiga, M. M., J. A. Meech, and N. Onate. 1994. Mercury pollution from deforestation. Nature **368**:816–817.

Victor, T. J. and R. Reuben. 2000. Effects of organic and inorganic fertilisers on mosquito populations in rice fields of southern India. Medical and Veterinary Entomology **14**:361–368.

Vitousek, P. M. and H. A. Mooney. 1997. Human domination of Earth's ecosystems. Page 494 Science.

Vittor, A. Y., R. H. Gilman, J. Tielsch, G. Glass, T. Shields, W. S. Lozano, V. Pinedo-Cancino, and J. A. Patz. 2006. The effect of deforestation on the human-biting rate of Anopheles darlingi, the primary vector of Falciparum malaria in the Peruvian Amazon. Am J Trop Med Hyg **74**:3–11.

Walker, E. D., D. Lawson, L, and R. Merritt, W. 1991. Nutrient dynamics, bacterial populations and mosquito productivity in tree-hole ecosystems and microcosms. Ecology **72**:1529–1546.

Weigle, K. A., C. Santrich, F. Martinez, L. Valderrama, and N. G. Saravia. 1993. Epidemiology of cutaneous leishmaniasis in Colombia: environmental and behavioral risk factors for infection, clinical manifestations, and pathogenicity. J Infect Dis **168**:709–714.

Weiss, R. A. and A. J. McMichael. 2004. Social and environmental risk factors in the emergence of infectious diseases. Nat Med **10**:S70–76.

Wilson, M. D., R. A. Cheke, S. P. J. Fiasse, S. Grist, M. Y. Osei-Ateweneboana, A. Tetteh-Kumah, G. K. Fiasorgbor, F. R. Jolliffe, D. A. Boakye, J. M. Hougard, L. Yameogo, and R. J. Post. 2002. Deforestation and the spatio-temporal distribution of savannah and forest members of the Simulium damnosum complex in southern Ghana and south-western Togo. Transactions of the Royal Society of Tropical Medicine and Hygiene **96**:632–639.

Wilson, M. L. 2001. Ecology and Infectious Disease. Pages 283–324 *in* J. L. Aron and J. Patz, editors. Ecosystem Change and Public Health. Johns Hopkins University Press, Baltimore, MD.

Wolfe, N. D., M. Eitel, N, J. Gockowski, P. Muchaal, K, C. Nolte, A. Prosser, T, J. Torimiro, N, S. Weise, F, and D. Burke, S. 2000. Deforestation, hunting, and the ecology of microbial emergence. Global Change and Human Health **1**:10–25.

Wolfe, N. D., W. M. Switzer, J. K. Carr, V. B. Bhullar, V. Shanmugam, U. Tamoufe, A. T. Prosser, J. N. Torimiro, A. Wright, E. Mpoudi-Ngole, F. E. McCutchan, D. L. Birx, T. M. Folks, D. S. Burke, and W. Heneine. 2004. Naturally acquired simian retrovirus infections in central African hunters. Lancet **363**:932–937.

Yasuoka, J. and R. Levins. 2007. Impact of deforestation and agricultural development on anopheline ecology and malaria epidemiology. Am J Trop Med Hyg **76**:450–460.