

# Land Use Change and Human Health

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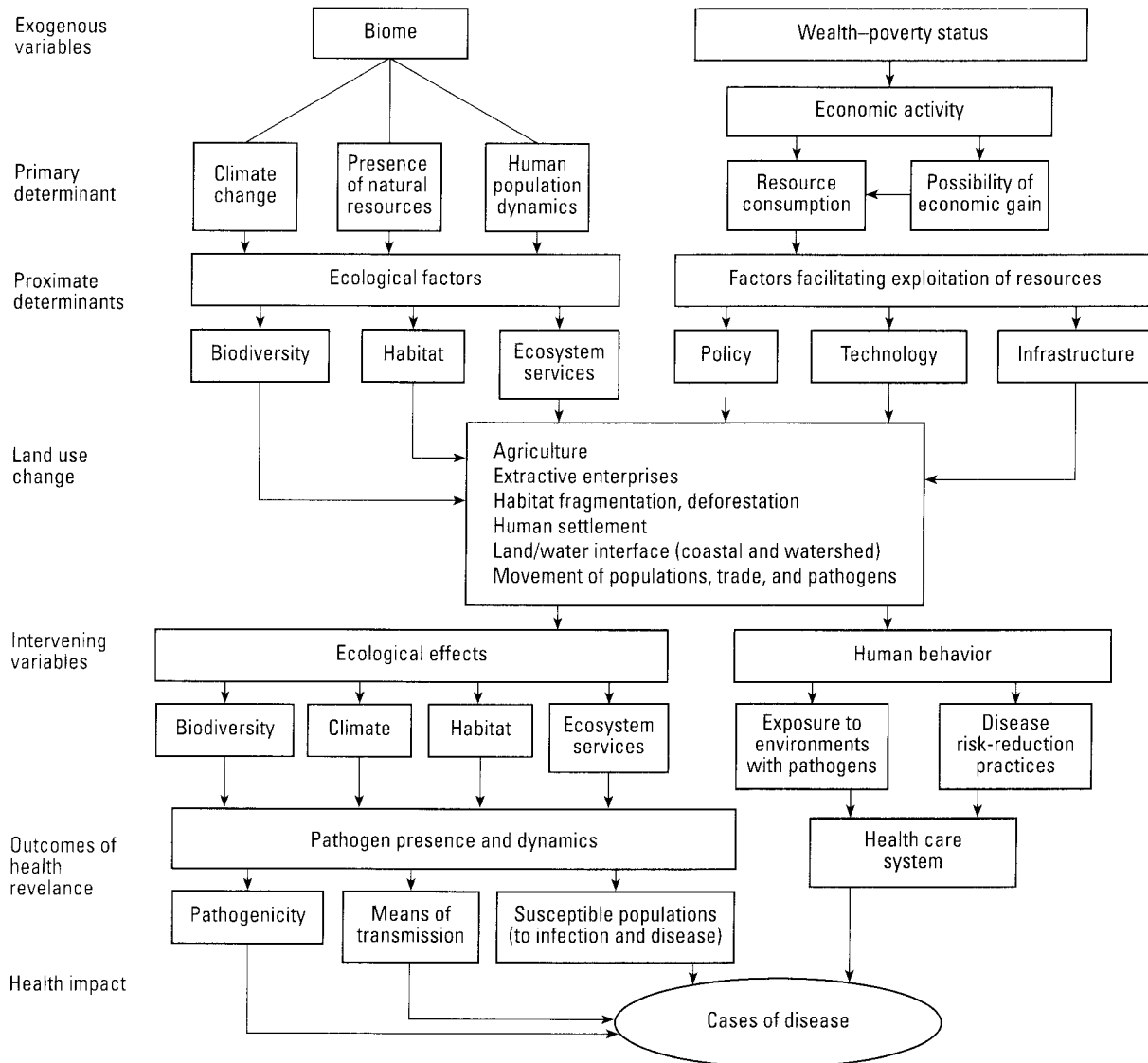
Disease emergence events have been documented following several types of land use change. This chapter reviews several health-relevant land use changes recognized today, including: 1) urbanization and urban sprawl; 2) water projects and agricultural development; 3) road construction and deforestation in the tropics; and 4) regeneration of temperate forests. Because habitat or climatic change substantially affects intermediate invertebrate hosts involved in many prevalent diseases, this chapter provides a basic description of vector-borne disease biology as a foundation for analyzing the effects of land use change. Urban sprawl poses health challenges stemming from heat waves exacerbated by the “urban heat island” effect, as well as from water contamination due to expanses of impervious road and concrete surfaces. Dams, irrigation and agricultural development have long been associated with diseases such as schistosomiasis and filariasis. Better management methods are required to address the trade-offs between expanded food production and altered habitats promoting deadly diseases. Deforestation can increase the nature and number of breeding sites for vector-borne diseases, such as malaria and onchocerciasis. Human host and disease vector interaction further increases risk, as can a change in arthropod-vector species composition.

## 1. INTRODUCTION

As humans change the landscape across the globe, the relationships between disease agents and their host organisms are shifting. Disease outbreaks and new emergence events have

been documented following several types of land use change (Figure 1). These include the concentration or expansion of urban settlements, agricultural development, dam building, irrigation, deforestation, road construction, wetland modification, mining, coastal zone degradation and other activities. Disease emergence from ecosystem change is complex and often poorly understood, yet some case studies have been recognized [Patz *et al.*, 2000; Patz *et al.*, 2004]. Furthermore, landscape integrity is a critical element in the maintenance of ecosystem services of direct economic relevance such as

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**Figure 1.** A systems model of land use change that affects public health. With permission from *Patz et al.*, 2004.

bacterial denitrification, pollination, tourism, fisheries, forest crops and other services [Balmford *et al.*, 2002].

In some cases, there are trade-offs between human health and ecosystem sustainability. For example, draining swamps may reduce vector-borne disease hazards but also destroy the wetland ecosystem and its inherent services. Roads can increase the probability of disease emergence but also provide access to health care and economic development to remote rural regions.

This chapter reviews many health-relevant land use changes recognized today, with a primary focus on infectious diseases. Reviewed are health outcomes related to 1) urbanization and urban sprawl; 2) water projects and agricultural development; 3) road construction and deforestation; and 4) regeneration of temperate forests. A basic description of vector-borne dis-

ease biology is presented as well, as a framework related to the following chapter in this volume by Thomson *et al.*, in which the authors present an in-depth look at how land use change is altering specific diseases in West Africa.

Land use change can change the risk of other health problems as well, that we do not cover in this chapter. These include local and transboundary air pollution, for example, from fires and dust, which are not addressed in this chapter.

## 2. URBANIZATION AND URBAN SPRAWL

On a global basis, the proportion of people living in urban centers will increase to an unprecedented 65% by the year 2030 [Population Reference Bureau, 1998]. Urbanization is

associated with a range of health problems, including vector-borne diseases such as dengue and malaria [Taul, 2001], diarrhoeal diseases [De Souza *et al.*, 2001], and respiratory diseases [D'Amato *et al.*, 2001]. Overcrowding and pollution resulting from inadequate infrastructure can trigger these conditions. At present, there are an estimated 4 billion cases of diarrhoeal disease each year, causing over 2 million deaths. Studies have shown that water sanitation and hygiene interventions can greatly reduce these water-related diseases [Esrey *et al.*, 1991; Taylor *et al.*, 2001].

### 2.1. Urban Heat Island Effect

Mortality due to heat waves is primarily a result of cardiovascular and respiratory disease [Kilbourne, 1997]. The 1995 heat wave in Chicago caused 514 heat-related deaths (12 per 100,000 population) [Whitman, 1997], and over 20,000 died during the 2003 summer heat wave in Europe [Haines and Patz, 2004]. The urban "heat island" effect can amplify general warming trends, whereby urban areas experience higher and nocturnally sustained temperatures due to the concentration of heat-retaining surfaces (e.g., asphalt and tar roofs).

Increased ambient temperature and altered wind and air mass patterns can affect atmospheric chemistry. Subsequently, there is a nonlinear relationship between temperature and the formation of ground level ozone (photochemical urban smog) with a strong positive relationship with temperature above 90° F. Ozone, a potent lung irritant that can heighten the sensitivity of asthmatics to allergens [Koren and Bromberg, 1995; Koren and Utell, 1997], recently has been shown to contribute to the development of asthma in children [McConnell *et al.*, 2002]. Urban sprawl, therefore, has potential to worsen ozone air pollution via the urban heat island effect.

### 2.2. Urban Stormwater Runoff

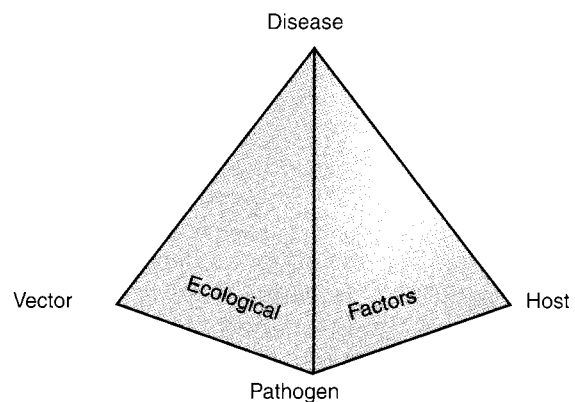
In addition to the "heat island" effect, expanded roadways and parking lots are impervious surfaces to rainwater and snow-melt. Therefore, urban sprawl can exacerbate urban runoff and increase the risk of surface water contamination. One critical, continuing threat to water quality and public health is the use by many communities of combined sewer systems. These systems are vestiges of early sanitation efforts in this country designed to carry both storm water and sanitary wastewater through the same pipe to a sewage treatment plant. During periods of rainfall or snow melt, the volume of water in the system can exceed the capacity of the sewer system or treatment plant; in such a situation, the system is designed to overflow and discharge the excess wastewater directly into surface water bodies. Because such combined sewer overflows (CSOs) contain untreated human and indus-

trial waste, they can carry solids, oxygen-demanding substances, ammonia, other potential toxics, and pathogenic microorganisms (associated with human disease and fecal pollution) to the receiving waters, precipitating beach closings, shellfish restrictions, and other water body impairments [Perciaspe, 1998; and Rose *et al.*, 2001].

## 3. ARTHROPOD-BORNE DISEASE

Arthropod-borne (vector-borne) diseases, such as malaria, West Nile virus, Lyme disease and Rocky Mountain Spotted Fever, for example, are a consequence of the relationships between a given host (e.g., humans), pathogen and vector (Figure 2). Study of these biological relationships has laid the foundation for understanding vector-borne disease transmission, but the reality is that these relationships can be significantly influenced by ecological change and environmental perturbations. Although ecological change may arguably have an effect on such factors as the host immune system or pathogen virulence, the greatest environmental effects on this system are directed towards the vector. From the point of view of remote sensors, geologists, or ecologists, influences may not be very intuitive or obvious. The goal of this section is to describe the potential relationships between environmental and land use changes and vector-borne disease.

Before we can consider the effects of ecology and environment on vector-borne disease transmission, we must have a basic understanding of vector-borne disease transmission cycles. In general, these cycles involve the vector first acquiring the pathogen from a reservoir. This reservoir may be an animal, another human, or the vector population itself. The reservoir may or may not show signs of disease, but plays an integral part in disease transmission—as the source of the pathogen in the disease transmission cycle. After the vector



**Figure 2.** The traditional triads of vector, pathogen and host interact with a myriad of ecological factors (i.e., temperature, humidity, land use change) to culminate in a condition recognized as a "disease."

(i.e., mosquito, tick, black fly) acquires the pathogen from the reservoir, the pathogen may then need to replicate or pass through an obligate developmental step in the vector arthropod. Most viruses, for example, require a few days to replicate to high number before transmission is efficient or even possible. In contrast, other pathogens, such as the malaria parasite, have an obligate developmental step that must occur in a biologically compatible species of mosquito for completion of the life cycle and subsequent transmission. This “time” requirement is recognized as the extrinsic incubation period (EIP). In either scenario, once a transmissible pathogen is ready, the vector must then find a suitable host and transfer the pathogen.

This transmission cycle is bounded by time and space. These components and a number of other biological determinants of vector competence, the biological compatibility of vector and pathogen, are heavily influenced by environmental factors. These influences are best understood by examination of vectorial capacity, the efficiency of vector-borne disease transmission. Vectorial capacity ( $V$ ) as first described by *Macdonald* [1957] and later modified by *Garrett-Jones* [1964] takes into consideration arthropod density in relation to the density of hosts ( $m$ ), the biological compatibility or vector competence of the vector and pathogen ( $b$ ), probabilities that the vector feeds on a host in one day ( $a$ ) and that it survives to the next day ( $p$ ), the time required for the pathogen to become infectious in the vector ( $n = \text{EIP}$ ), and the total lifespan of the vector after becoming infectious ( $1/\ln p$ ) [*Black and Moore*, 1996]. Vectorial capacity is quantified as:

$$V = \frac{mba^2 p^n}{-\ln(p)}$$

There are four major environmental factors that have a broad impact on the vector and vectorial capacity. These are day length, temperature, humidity, and habitat. The first three of these factors are largely determined by climate, latitude, and geography. We are only starting to understand the affects of human activity on temperature and humidity (i.e., global warming, heat islands), but know these factors can be greatly affected by land use (see *Bonan*, this volume, and *Avissar et al.*, this volume), especially at a local level. Day length, on the other hand, is perhaps only minimally affected by human activity, if at all. The fourth factor, habitat, although most often determined or defined by the previously mentioned factors, can be heavily influenced and abruptly altered by human activity, even to the extent of locally overriding the other environmental factors.

Day length is related to seasonality and to vector physiology and activity patterns. In the tropics day length is relatively constant and little seasonality within the vector

population and vector-borne disease is observed as long as humidity and rainfall are relatively constant. Increasingly marked seasonality occurs at higher and lower latitudes with vector populations most abundant during the wet summer (warm) months and least abundant or dormant during the dry winter (cold) months. At these latter latitudes some vector populations can enter diapause, a period of suspended development induced by shortened photoperiod [*Mitchell*, 1988; *Nasci and Miller*, 1996]. Many species of mosquitoes use this mechanism to overwinter as eggs, larvae, or adults. This may also be the mechanism by which mosquito-borne viruses such as West Nile virus and St. Louis encephalitis virus survive from season to season [*Dohm et al.*, 2002b; and *Reisen et al.*, 2002]. Seasonality is also seen in the equatorial zone, but here is associated with hot dry (vector absent) and warm wet (vector present) seasons rather than day length.

Temperature, as an independent factor, can affect the vector-borne disease cycle in a number of ways. Most vectors are small arthropods and lack the ability to regulate body temperature well. They are therefore, at the mercy of their environment in terms of physiological temperature, controlling body temperature by moving into and out of existing temperature zones (i.e., shade and shelter). The replication or development of the pathogen in the vector is often temperature dependent, as is the developmental rate of the arthropod. These rates tend to accelerate with increased temperature and slow with depressed temperature, within particular bounds. Generally higher temperatures affect this system by producing more efficient vectors [*Dohm et al.*, 2002a]. However, temperatures too high or too low can stop development and maturation altogether or even kill the parasite or vector. Therefore, temperature clines determine the geographic distribution of vectors as well as the distribution of vectors in which the pathogen may successfully develop. This is largely due to survivorship of the vector. If the temperature is too low or too high to allow rapid development of the pathogen, the vector may die before the pathogen is transmissible. These effects were clearly illustrated in a study modeling the effects of temperature on the transmission dynamics of lymphatic filariasis in French Polynesia [*Lardeux and Cheffort*, 2001]. On islands at the greatest southerly latitude, transmission is seasonal, with no transmission during the coolest months because the parasite EIP is longer than the lifespan of the mosquito involved in transmission. In contrast, transmission is continuous on islands at the most northerly latitude, due to high temperatures year round and an EIP much shorter than the lifespan of the vector.

When water is present, temperature also has a great effect on humidity. Greater temperatures generally allow for higher humidity. In addition to being very susceptible to environmental temperatures, many arthropods involved in vector-

borne disease transmission are also prone to desiccation at low humidity. Low humidity can effectively shorten the lifespan of the arthropod, having an enormous effect on vectorial capacity even if vectors are locally abundant. Similarly, humidity can be a limiting factor to vector distributions, despite temperatures within acceptable bounds. Several contemporary climate models identify humidity as one of the most important variables for modeling the changing distributions of vector-borne diseases [Randolph and Rogers, 2000; Hales et al., 2002; Minakawa et al., 2002]. In fact, the consideration of changes in local humidity becomes increasingly important as the temperature models alone may predict the expansion of vector-borne diseases into areas that are inhospitable for the vectors.

Habitat is a broad category covering everything from natural land cover to urban landscapes. Habitat is defined by an enormous number of variables. For example, a particular habitat may be defined by the types of plant life that exist on a specific soil mixture, at a given latitude and altitude with a defined amount of rainfall. Similarly, vector species have defined habitats in which they thrive and others in which they simply survive. In addition, many vector species have adapted to man-made habitats, habitats that artificially allow vectors to thrive where they might have never even existed without the inadvertent assistance of habitat alteration. Many mosquito species involved in virus transmission, for example, thrive in discarded tires, containers and clogged gutters in urban settings. These same species might be absent were they to depend on their native tree hole and forest depression habitats that no longer exist in the urban environment. Land use change not only allows for the invasion of vector species into new habitats or extended ranges, but also may alter the composition of vector populations by inducing changes that give a particular

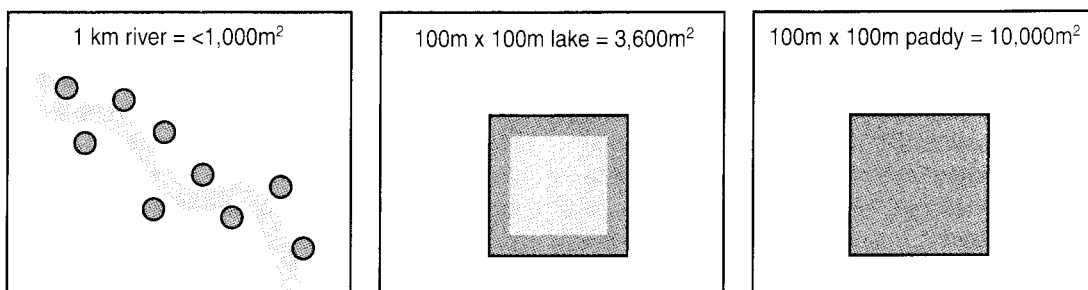
vector species a competitive advantage over other less efficient vectors, or simply enhance vector abundance.

Land use change has a demonstrable effect on vector-borne disease. Slowing water flow and decreasing depth in irrigation canals, lakes behind dams, retention ponds, and rice paddies have a major effect on mosquito habitat [Norris, 2004]. For example, a rice paddy may provide up to ten times more mosquito habitat than a riverine system with equal surface area, simply because the paddy is shallow with little to no water flow (Figure 3). Water management may do much more than simply increase the available vector habitat. Irrigation projects in the arid regions of West Africa have increased mosquito habitat as well as provided water and food for more hosts. This has led to higher transmission rates for lymphatic filariasis by increasing vector abundance and survivorship, effectively creating a larger infective vector population through an extended transmission season [Appawu et al., 2001].

The following sections illustrate the range of effects of land use change on vector-borne diseases.

### 3.1. Water Projects and Agricultural Development

Reservoirs, irrigation canals, and dams are closely associated with parasitic disease. Construction of reservoirs and canals can lead to a shift in vector populations, such as snails and mosquitoes, their larvae and their parasites. Tropical reservoirs and irrigation systems provide ideal aquaria for rapid reproduction and growth of fluke-transmitting aquatic snails. Excavation pits associated with construction of dams and canals provide breeding sites for mosquitoes before the canals and reservoirs behind the new dams are even filled. Additional breeding sites are provided by creation of basin irrigation for rice, by poor drainage, impounded water and seepage.



**Figure 3.** This figure illustrates that the relative contribution to vector habitat of a river, lake and paddy of equal surface area ( $10,000 \text{ m}^2$ ) are inherently different, depending on the specific characteristics of that system. In general, immature mosquitoes prefer relatively shallow, slow-moving water for development. Therefore, streamflow is often too rapid, although overflow pools along a river or stream may serve as an ideal habitat. A 1-km section of river 10 m wide (light grey) may be associated with approximately  $1,000 \text{ m}^2$  of suitable habitat (dark grey) in overflow pools. Similarly, the center of a lake is often too deep and mosquitoes are found to develop predominantly within the first 10 m from shore ( $3,600 \text{ m}^2$  of suitable habitat). Rice paddies, alternatively, are essentially shallow lakes with emergent vegetation and are ideal for the development of many mosquito species ( $10,000 \text{ m}^2$  of suitable habitat). With permission from Norris, 2004.

The larvae, having no air tube, float just beneath the water's surface.

Different mosquito species vary in their habitat requirements, in both the larval and adult stages. Some species prefer sunlit pools with turbid water, with little or no emergent vegetation. Some larvae prefer clear water, inhabiting the edges of clean, clear, gently moving streams or, conversely, others thrive in irrigation and hydroelectric reservoirs with their frequent changes in water level, vertical shorelines, and emergent vegetation without organic material or salinity; others inhabit coastal areas with high salinity. There are species which require extensive vegetation cover and inhabit swamps and relatively permanent waterbodies with organic material [Patz *et al.*, 2000]. Riverine pools, created by diversion of flow out of river beds, provide breeding in shallow, stagnant surface water exposed to sunlight. Deeply shaded pools, seepages in forests, footprints, mining pits and irrigation ditches, and excavated depressions in the open sunlight all provide areas for mosquitoes to deposit their eggs [WHO, 1982]. Unfortunately, the wide variety of conditions under which at least a few species are able to thrive ensures that mosquito-vector parasitic disease is ubiquitous, flourishing throughout many regions of the world, especially in tropical areas.

Agricultural development in many parts of the world has resulted in an increased requirement for crop irrigation, which reduces water availability for other uses and increases breeding sites for disease vectors [Patz *et al.*, 2004]. The Aswan High dam caused soil moisture to increase from the associated irrigation development in the Southern Nile delta. As a consequence, the mosquito, *Culex p. pipiens*, proliferated and increases in Bancroftian filariasis resulted [Harb *et al.*, 1993; Thompson *et al.*, 1996]. Onchocerciasis and trypanosomiasis are further examples of vector-borne parasitic diseases that may be triggered by changing land-use and water management patterns and are further detailed in the next chapter. In addition, large-scale use of pesticides has had deleterious effects on farm workers including hormone disruption and immune suppression [Straube *et al.*, 1999].

Agriculture development also can lead to an increase in diarrhoeal diseases. In intensely stocked farmland, heavy rains can cause contamination of water resources by *Cryptosporidium parvum* oocysts. Intense cattle farming and livestock operations in combination with factors related to watershed management have been implicated in such outbreaks of cryptosporidiosis [MacKenzie *et al.*, 1994; and Graczyk *et al.*, 2000]. A similar mechanism is involved in giardiasis, where a variety of animals may serve as the reservoir of *Giardia lamblia* and contaminate surface water with their excreta. Predicted flooding accompanying climate change could increase the water contamination trends associated with agriculture development.

### 3.2. Road Construction and Deforestation

New roads into pristine areas provide access for new human, livestock, vector, and parasite populations and facilitates acceleration of crop farming, ranching, logging, mining, commercial development, and tourism. Construction of new settlements, building of dams and hydroelectric plants, with all their attendant disruption of the ecological balance, will soon follow.

During construction of roads erosion can be extensive and often creates culverts which may collect rainwater. New water channels and silting block the flow of streams, and ponds are created when the water rises during the rainy season [Kalliola and Flores, 1998]. Increased runoff also accelerates sedimentation, effectively decreasing streamflow [Dian and Changxing, 2001]. Shallow water with little or no streamflow warms quickly, creating ideal breeding habitat for existing or newly invasive mosquitoes [Norris, 2004]. By providing easy access to forested and newly deforested (partially or completely) areas, non-immune, non-protected populations, such as construction workers, loggers, miners, tourists, and conservationists, are exposed to indigenous and newly arrived vectors and their parasites. Further, these visiting human populations bring with them parasitic infections, and new vector species from their far-flung points of origin, that are introduced to existing vectors and settlers along the forested/deforested interface.

When tropical forests are cleared for human activities, they are typically converted into species-poor agricultural and ranching areas. As previously described, this process is usually exacerbated by construction of roads, causing erosion and allowing previously inaccessible areas to become colonized by people [Kalliola and Flores, 1998]. The resulting monocultures in agricultural systems become targets that are taken advantage of by pest species that become so abundant they cannot be effectively controlled, and economic loss ensues. Similarly, ecosystems depleted of animal diversity have also been associated with the exacerbation of tick-borne disease [Schmidt and Ostfeld, 2001], as ectoparasite populations explode, feeding on a monoculture of domestic or peri-domestic animals (discussed in section 3.3).

Deforestation, with subsequent changes in land use and human settlement patterns, has coincided with an upsurge of malaria and/or its vectors in Africa [Coluzzi, 1994], in Asia [Bunnag *et al.*, 1979], and in Latin America [Tadei *et al.*, 1998]. The competence of different anophelines to transmit malaria varies between species. Anopheline species also occupy a variety of ecological niches. *Anopheles darlingi* in South America and *Anopheles gambiae s.l.* in sub-Saharan Africa are the predominant and highly competent vectors in their respective regions. Approximately 50 other anopheline species attain

relative prominence and importance in malaria transmission in macro- and microenvironments they can exploit. Environmental changes allow these and additional anopheline mosquitoes to invade. Cleared lands and culverts that collect rainwater are in some areas far more suitable than forest for malaria-transmitting anopheline breeding [Tyssul Jones, 1951; Marques, 1987; and Charlwood and Alecrim, 1989]. Elsewhere it is the process rather than the final outcome of deforestation that increases malaria. In effect, forest-dwelling *Anopheles* species either adapt to newly changed environmental conditions or disappear from the area, which offers other anophelines (and species that transmit additional mosquito-borne diseases) a new ecological niche [Pova et al., 2003]. Deforestation can also lead to increased levels of contact between wildlife and humans and their domestics, allowing wildlife diseases and ectoparasites to bridge to domestic animals and associated human populations, and vice versa.

In addition to the influence of infectious diseases, deforestation also poses a health risk via contamination of rivers with mercury. In addition to direct mercury pollution from gold mining (described below), the added soil erosion following deforestation adds significant mercury loads (found naturally in rainforest soils) into rivers such that fish in the Amazon have become hazardous to eat [Fostier et al., 2000].

Deforestation has major implications for biodiversity associated with habitat loss from deforestation, particularly in the tropics (see Laurance, this volume). Biodiversity loss can have ramifications for pharmaceuticals and medical research models [Chivian, 2001]. Through evolution, plants have developed biologically active compounds that help protect them against threats from animals, fungi, bacteria, and viruses [Bell, 1993]. Humans have learned to extract these compounds, using them to protect themselves from disease. Traditional remedies are often used as a starting point for researchers in the development of a new drug. In 1785, English physician Dr. William Withering, with the guidance of a local woman, formally discovered that the foxglove plant was an effective treatment for patients suffering from drops. The active agents in foxglove, digitoxin and its less-toxic relative digoxin, are marketed and remain the drug of choice for some heart conditions. Other drugs, such as quinine, atropine, and taxol, were also initially derived from plants. It is estimated that 25% of drugs used in western medicine today contain at least one plant-derived active ingredient [Bell, 1993].

### 3.3. Regenerating Temperate Forests, Biodiversity Loss and Lyme Disease

In temperate developed countries, as land use patterns have shifted away from agriculture and as second-growth forests have become more prevalent, tick populations have changed

[McCarthy et al., 2001]. In the U.S., human infection rates for Lyme disease have risen over time with the increase in contact between humans and ticks [Ostfeld, 1997]. Spatial variation in risk to humans is influenced by the ecology of the mammalian hosts of the tick. White-tailed deer are the primary host for adult ticks and their population size is an important determinant of the abundance of ticks in a particular region [Van Buskirk and Ostfeld, 1995]. While tick population size is an important factor determining the probability of a human being bitten, not all ticks will be infected with the Lyme disease bacterium. The proportion of infected ticks in an area appears to be related to the relative abundance of highly competent small mammal reservoirs for the *B. burgdorferi* bacterium, specifically, white-footed mice [Schmidt and Ostfeld, 2001].

The probability of a human contracting Lyme disease in a given area, therefore, is a function of the density of infected ticks, which in turn depends on the population size of deer and mice. Modeling studies suggest that a complete understanding of the ecology of Lyme disease will need to include other members of the ecological community in addition to these key players. Results suggest the probability of a tick becoming infected depends not simply on the density of white-footed mice, but on the density of mice relative to that of other hosts in the community. Under this scenario, the density effect of white-footed mice, which are efficient reservoirs for Lyme disease, can be "diluted" by an increasing density of alternative hosts, which are less efficient at transmitting Lyme disease. This would suggest that increasing host diversity (species richness) could decrease the risk of disease through a "dilution effect" [Schmidt and Ostfeld, 2001].

Habitat fragmentation (e.g., due to urban sprawl and road building) may exacerbate the loss of species biodiversity, thereby increasing the risk of Lyme disease. When human development fractures forests into smaller and smaller pieces, forest/field edge increases. This "edge" habitat is ideal for rodents and allows the reservoir population to swell. The fragmentation itself may effectively exclude species that require larger, contiguous habitat ranges, additionally constraining species richness. Each of these ecosystem changes has implications for the distribution or exacerbation of microorganisms and the health of human, domestic animal, and wildlife populations.

## 4. CONCLUSION

Land use has modified the distribution and behavior of disease agents, their vectors and/or intermediate host species. The examples cited in this chapter demonstrate the close relationships among land use, climate changes and human disease. Better surveillance and monitoring of land use and

disease occurrence are needed, both for identification of immediately required action and to serve as the basis for developing predictive models.

Further research, particularly across physical, ecological and medical/social scientific disciplines, is essential for more adequate assessment of how changing landscapes may alter the risk of human disease.

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