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EFFECTS OF LAND-USE CHANGES AND AGRICULTURAL PRACTICES ON THE EMERGENCE AND REEMERGENCE OF HUMAN VIRAL DISEASES

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8.1 INTRODUCTION

Land use can be defined as the human management and modification of terrestrial surfaces, including changes to animal and plant populations, soil, surface waters, and topography (Turner et al., 2007). Current levels of land-use changes are unprecedented in history. While humans have transformed and managed natural landscapes since the beginning of agriculture 10 000 years ago, over the past decades changes have been particularly marked as a result of urban expansion, the construction of new roads and waterways, and extensive agricultural development. Today, between half and two-thirds of the earth's ice-free land surface has been transformed (Haberl et al., 2007), with 4.9 billion hectares or close to 38% of the total surface covered by agriculture alone (FAO, 2012).

These developments vary greatly across different regions of the world as land is transformed for agriculture or settled by human populations (Figure 8.1) (Ellis et al., 2010; Lambin et al., 2001). With the global population expected to reach nine billion by 2050, increasing levels of urbanization, higher levels of food consumption, and demand for natural resources and biomass-derived energy, the intensity of land use should be expected to rise as well (Haberl et al., 2007). Rapid transformations in land uses are likely to result in further changes to ecosystems and land fragmentation.

What are the implications of these changes for human health and the environment? On the positive impacts, agricultural development has contributed to socioeconomic growth and food security worldwide by providing cheaper and safer food. Improved agricultural productivity can additionally provide essential ecosystem services such as carbon sequestration, regulation of soil and water, and support of pollinating insects (Power, 2010).

However, concerns have been raised that land-use changes alter the ecological systems leading to environmental damage and contributing to novel hazards to animal and human health. Research has long documented the environmental consequences of land modification and agricultural development, including the depletion of soil nutrients, land degradation, contributions to climate change, disruption of fundamental ecosystems, and loss of biodiversity (Patz et al., 2000; Sherbinin, 2002). In the past few years, increasing attention has been directed to the effects on communicable diseases. Especially after the emergence of highly pathogenic diseases such as severe acute respiratory syndrome (SARS) and Nipah virus in areas undergoing rapid changes in land use, the link between agricultural practices and disease emergence has become more apparent (Patz et al., 2004; Weiss and McMichael, 2004; Wilcox and Gubler, 2005). Given the zoonotic nature of a majority of emerging diseases in humans (Woolhouse et al., 2005), there are concerns that changes in land use may facilitate the emergence of new ecological niches that microbes may exploit and the pathogen exchange between wild animals, livestock, and human populations. It is now

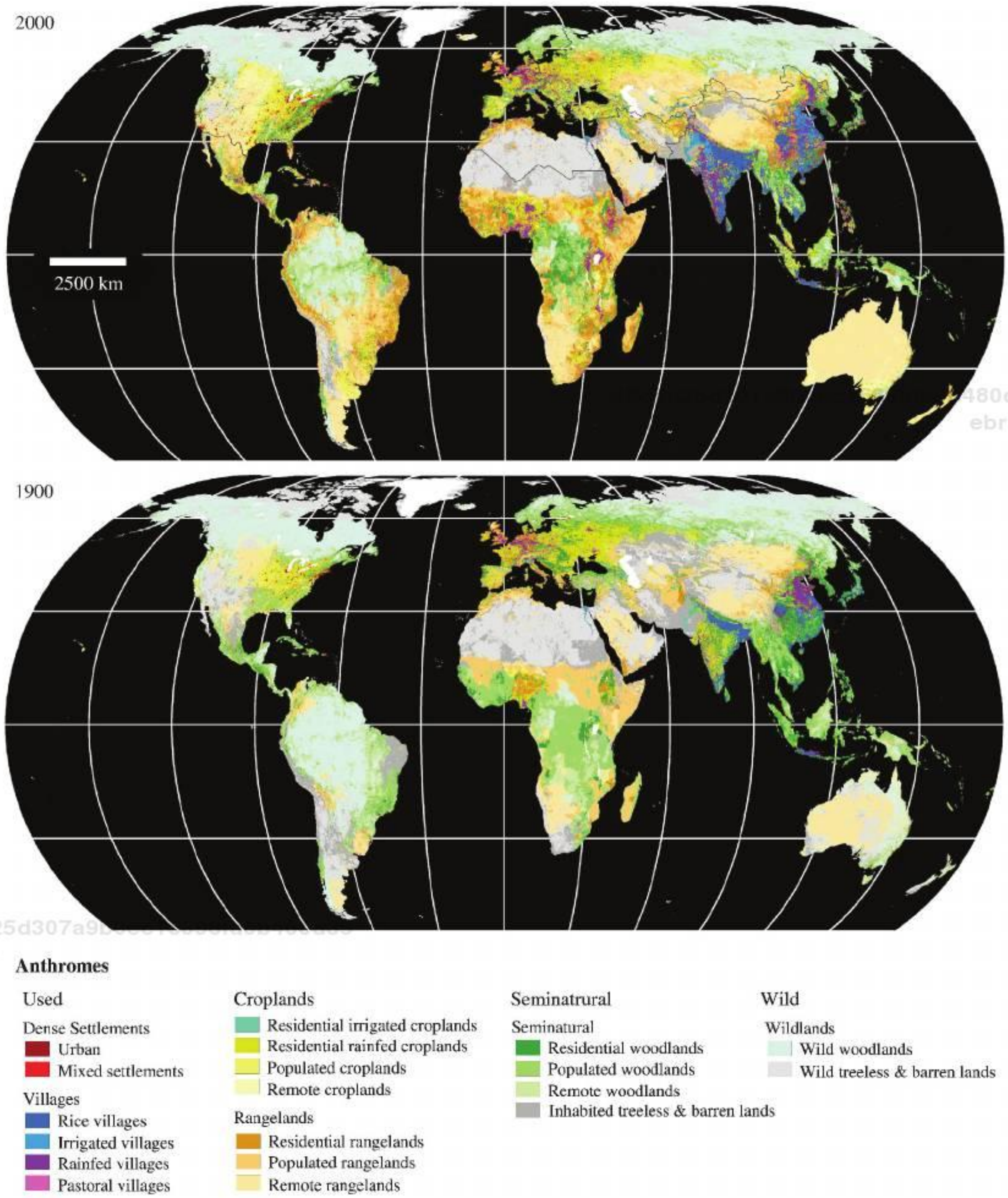

Figure 8.1. Anthropogenic biomes, 1900–2000, reproduced from Ellis et al. (2010). For color detail, please see color plate section.

clear that man-made interventions such as deforestation, urbanization, and agricultural expansion alter fundamental ecosystems and the geographical distribution of human populations and animal species, with mixed effects that may increase or reduce the risk of infectious disease emergence or reemergence. Land use influences in potentially complex ways microbial transmission dynamics, including the likelihood of pathogens progressing through each stage of disease emergence—exposure, infection, and propagation—exerting evolutionary pressures on pathogens, hosts, and ecosystems, while transmission in humans can be amplified by migration, trade, and societal changes.

Despite sustained research, however, the processes that link land-use change with disease emergence are still largely unknown. Disease emergence is not a simple causal effect; it is rather the result of complex multifactorial interactions, requiring pathogens to overcome numerous ecological and evolutionary barriers to switch hosts and establish in human populations. This chapter aims to shed some light on this complexity by reviewing recent scientific works at the interface between ecological and health sciences. Specifically, the chapter examines the role of changes to the environment and ecosystems; agricultural practices, including agricultural expansion and intensification of livestock production; and related demographic changes, including urbanization, trade, and migration.

8.2 ECOLOGICAL AND ENVIRONMENTAL CHANGES

Environmental changes, such as deforestation and habitat fragmentation, disrupt existing ecosystems and change the physical characteristics of landscape. This can affect the size and movement of human, animal, and pathogen populations and can increase the interface between different areas, bringing previously separated populations into contact and changing the population dynamics regulating community structures. Humans entering new environments can be exposed to new pathogens and may spread these pathogens to different communities. Landscape changes additionally modify the physical characteristics of the environment, determining the suitability of different areas as habitats for animal and insect populations and altering migration patterns and connectivity between different areas. These changes can transform the structure of ecological communities, affecting species richness, abundance, and composition.

8.2.1 Deforestation

Deforestation is one of the largest anthropogenic changes to land cover, with 2–3% of planetary forests lost each year (Patz et al., 2004). It is driven by various demands, the extraction of valuable timber, the requirement for extra land for agriculture (see discussion later), and in some cases the need to mark boundaries of nations. Deforestation can be defined as the conversion of forest areas into less diverse biosystems, such as pasture, cropland, plantations, transport routes, or urban areas. The uncontrolled exploitation of forest areas is often associated with large personal gains for individuals or small groups of people with large negative impacts and local disruption for many.

The effects of deforestation on human diseases have been documented in several studies. Deforestation alters habitat structure and species composition and can increase the interface between humans and wildlife. Disruption of forest cover and habitat fragmentation creates ecotones, transition areas between wild and man-made environments with increased contact between hosts, vectors, and pathogens (Lambin et al., 2010). The "edge effect" of these areas was identified as a driver of increased numbers of cases of human tick-borne encephalitis (TBE) cases in Latvia, where human activities and tick habitats overlapped (Vanwambeke et al., 2010). The emergence of yellow fever has also been associated with a forest ecotone. Yellow fever virus is maintained in monkey and mosquito species predominantly living in the deep forest canopy. Encroachment of human settlements and agricultural lands disrupted transmission cycles, infecting domestic *Aedes* mosquito species and exposing humans to the virus (Despommier et al., 2006). Comparable changes from jungle to sylvatic and domestic transmission cycles may have also contributed to the emergence of dengue and other arboviruses in the edges of the Amazon rainforest (Vasconcelos et al., 2001).

Human activity in forests, through logging or other practices associated with deforestation, can further contribute to zoonotic risk. While clear-cutting forests may reduce wildlife diversity, selective extraction of high-value timber species is more likely to sustain biodiversity and thus a wider pool of potential pathogens. Moreover, selective extraction can increase the contact rate between humans and pathogens in the wildlife, as workers venture into forest areas to select and collect wood (Wolfe, 2005). Initial human exposure to Marburg and Ebola viruses most likely occurred through contact with wildlife reservoirs while humans were active in forest environments (Cascio et al., 2011). Additionally, encroachment of agriculture and livestock into forest areas can contribute to disease spread by supporting vector or pathogen populations. Grazing cattle in forest areas in the Mysore State of India provided a source of blood meals for ticks carrying the previously undetected Kyasanur Forest disease virus. The associated increase of tick populations and human exposure led to the emergence of Kyasanur Forest disease in people living around forest areas (Simpson, 1978).

The construction of roads and waterways into forests for the transport of logs and other material can create further opportunities for disease spread into human communities. In some cases, new transport routes may stimulate an increase in deforestation and associated disease risk. Medeiros et al. found a dramatic increase in human hantaviral cases following the completion of a highway through the Brazilian Amazon. Traffic through the region provided new economic opportunities for the inhabitants of the area surrounding the highway, encouraging wood collection, agriculture, and other forest activities (Medeiros et al., 2010).

Deforestation has also been associated with risk resulting from bushmeat consumption; poor individuals living in proximity to wildlife are more likely to hunt and consume bushmeat as an alternative protein source (Brashares et al., 2011). With the identification of chimpanzees as the natural reservoir for HIV, there is strong evidence HIV originated from hunting and butchering nonhuman primates in central Africa (Keele et al., 2006). Bushmeat hunters and other people reporting contact with nonhuman primates have increased exposures to other simian viruses, such as simian foamy virus (Wolfe et al., 2004). Circulation of simian and human T-lymphotropic viruses (HTLV) between African hunters and nonhuman primate populations suggests bushmeat hunting could contribute to HTLV emergence (Wolfe et al., 2005).

8.2.2 Habitat fragmentation

Changes to the environment, whether through deforestation or other changes to land cover, alter the species abundance, distribution, and connectivity of habitats. Habitat fragmentation occurs when the habitat is spatially separated due to land changes, creating a mosaic pattern of different land types. Fragmentation can increase the risk of disease emergence by increasing the area of edges forming ecotones and increasing crowding and contact rates within the habitat. For example, increased density of birds in smaller forest fragments is correlated with higher tick infestation rates, likely due to the increased probability of ticks successfully finding a competent host (Ogrzewalska et al., 2011). However, habitat fragmentation may also decrease risk of disease emergence by isolating susceptible species from disease hosts (Vogeli et al., 2011). Geographical separation of habitats affects host migration patterns and use of land by different species.

The varied effects of habitat fragmentation on hantavirus distribution illustrate the complexity of ecological disturbances on disease dynamics. Phylogenetic studies indicate hantaviruses coevolved with their rodent hosts; geographical distribution of viral strains is determined primarily by the ecological niche and migration patterns of the primary rodent hosts (Wei et al., 2011). Changing land use affects the population size of rodent hosts in different ways depending on the biogeography of the host. In Central America, disturbed areas around the edges of natural habitats were correlated with an increased density of rodent hosts (Suzan et al., 2008). In contrast, land disturbances in North America altered rodent population structures, causing a high turnover, younger ages, and decreased serological evidence of exposure to the Sin Nombre hantavirus (Calisher et al., 2001). Other studies suggest hantavirus infection may not occur below a certain density of rodent hosts (Tersago et al., 2008).

Landscape and habitat fragmentation also affect the connectivity of different habitats. While increased Puumala hantavirus carriage was detected in European bank voles living in large connected habitats, geographically isolated and smaller habitats were associated with decreased hantavirus prevalence (Guivier et al., 2011). Overlapping spatial distribution of different rodent hosts plays a role in virus evolution through host switching and reassortment events. Related hantaviral strains were detected in different rodent species living in humid subtropical forests and temperate grasslands. Although these species live in very different ecological regions, anthropogenic changes to habitats led to transition areas used by previously geographically isolated species (Chu et al., 2006).

8.2.3 Structural changes in ecosystems

Structural changes, such as change in the numbers or behavior of different species, within ecosystems can produce further risk of disease emergence by changing species richness, abundance, and composition. For example, land use may affect the microclimatic variables, changing suitability of areas for vector habitats. Ross River virus emerged in Australia after clearing vegetation decreased transevaporation levels and increased water levels dissolved salt deposits leading to the formation of saline pools. The main vector of Ross River virus, the *Aedes camptorhynchus* mosquito, is salt tolerant and thrives in areas of dryland salinity. Wide-scale habitat changes led to conditions favoring this vector leading to an increase in human cases (Jardine et al., 2008).

In addition, anthropogenic land use and the modification of natural habitats negatively impact biodiversity. The effects of biodiversity on disease emergence and transmission are complex, altering the abundance, behavior, and condition of hosts, vectors, or pathogens (Keesing et al., 2010). In heterogeneous populations, the effect of biodiversity on disease dynamics is primarily determined by structural changes to ecological communities. Whether changing host community structure amplifies or reduces the risk of emergence depends on the disease competence of the new community structure and the transmission mode of the pathogen. Loss of species that are not disease hosts or are suboptimal disease hosts will not affect pathogen transmission rates. Additional species may provide sources of blood meals for vectors, increasing vector population and disease risk. Highly biologically diverse areas can increase the zoonotic pool of pathogens, acting as a source for further disease emergence events (Randolph and Dobson, 2012). If the amplifying host species declines as biodiversity decreases, there will be a reduction in disease risk. Conversely, if the amplifying host thrives as biodiversity decreases, disease risk will increase. Influences on different species and populations in multi-host and vector-borne disease systems may have opposing effects.

Protective effects of biodiversity were initially described for vector-borne parasitic diseases, such as malaria and Lyme disease. Higher densities of noncompetent hosts can either dilute disease effects by "wasting insect bites" or amplify disease risks by supporting populations. Biodiversity decreases risk when vectors preferentially feed on noncompetent hosts; increased species diversity of wild birds, the primary reservoirs of West Nile virus (WNV), was suggested to decrease mosquito infection risk by increasing numbers of suboptimal hosts. Lower virus amplification rates were found in areas with higher non-passerine wild bird species richness (Ezenwa et al., 2006). Further studies found the force of WNV infection, the number of infectious mosquitoes resulting from feeding on a host, depended on changes in host selection and reservoir competence rather than measures of avian diversity (Hamer et al., 2011). Presence of noncompetent hosts can alternatively amplify disease risk by increasing abundance of vector populations. For example, increased deer density is correlated with increased human tick-borne encephalitis virus (TBEV) cases; although deer cannot transmit the virus, increased deer abundance supports tick populations (Rizzoli et al., 2009). Changes in plant diversity can also alter the vector habitats, affecting insect population abundance and feeding behavior (Randolph and Dobson, 2012).

8.3 AGRICULTURAL CHANGE

Agriculture is the largest human use of land and one of the main drivers of ecological changes and environmental modification. Part of the deforestation described earlier relates to a demand for new agricultural land and is not solely about a drive to extract resources. Population growth and changing consumption patterns contribute to rising demand for agricultural products. Global demand for livestock products is predicted to increase from 6 to 23 kg per person per year by 2050, with demand per person more than doubling in sub-Saharan Africa (Thornton and Herrero, 2010). Global biofuel production is also growing rapidly and expected to quadruple in the next 15–20 years due to rising oil prices (Patz et al., 2008). Meeting this demand entails both expanding land use and increasing productivity levels through intensification. Agricultural intensification is characterized by selection for increasingly specialized breeds, mechanization, and use of technology and industrial management. Crop yields have been improved through wide-scale use of irrigation, fertilizers, and pesticides, while livestock industries have adopted higher stocking densities, the use of concentrated feed, and increased pharmaceutical product use. Intensification changes the relative importance and dynamics of diseases, and it can drive disease emergence. In addition by selecting for homogeneous host populations and increasing movement and trade, the impact of disease presence is increased. Biosecurity and husbandry practices affect contact rates with wild populations and management of waste and the environment. Disease control practices may decrease susceptibility to infection or alternatively select for new or more pathogenic infections.

8.3.1 Agricultural expansion

Spatial expansion of agriculture necessitates clearing and modifying lands for crop or livestock production. As discussed earlier, large-scale changes in land surface and deforestation may create favorable ecological conditions to disease emergence and transmission. In some cases, these changes can act in combination with technological interventions to produce high-risk environments. In Bolivia, for example, wide-scale deforestation to create croplands triggered an increase in the wild *Calomys* mouse population, which carried the Machupo virus and fed off agricultural waste. Applications of pesticides such as DDT killed cats, the main predators of mouse populations. As a result, the mouse population further expanded leading to an outbreak of Machupo hemorrhagic fever in humans exposed to mouse excretions. The virus killed close to 15% of the local population (McMichael, 2004; Simpson, 1978).

Expansion of crop production also involves new irrigation systems and construction of waterways, providing further opportunities for transmission of waterborne diseases. Contaminated irrigation water is the suspected source of norovirus, rotavirus, and hepatitis E viruses detected on strawberries grown for human consumption (Brassard et al., 2012). In addition, irrigation modifies surface waters and may create new breeding sites for arthropod vectors of viral diseases; research suggests that water management practices in rice paddies can significantly increase or reduce the number of mosquitoes carrying Japanese encephalitis virus (JEV) (Keiser et al., 2005). Other characteristics of rice paddies, such as the height of plants and dissolved oxygen and nitrogen levels, may also influence their suitability as mosquito-breeding sites (Sunish and Reuben, 2002). JEV is maintained in wild bird populations and can be further amplified by pigs; proximity of rice paddies to wild bird habitats and pig farming influences risk of mosquito viral carriage (Mackenzie and Williams, 2009). Of patients with confirmed cases of JE admitted to a hospital in Assam, India, over 78% reported working in rice cultivation and 55% reported close association with pigs (Phukan et al., 2004).

8.3.2 Intensification of livestock production

The growing intensification of livestock systems further influences the emergence and dynamics of zoonotic diseases. Animal production, particularly for pigs and poultry, has become increasingly intensified in both developed and developing countries. Large numbers of genetically homogeneous animals are kept at high densities in controlled environments under industrial management, frequently integrating all aspects of production from feed production to processing (FAO, 2006). Industrial farms may be easier to regulate, have improved levels of biosecurity, and expose fewer workers to disease risks. Also, industrial farms keep single species in isolation, reducing opportunities for viral recombination between species.

However, large populations of standardized animals can amplify risk of contact and pathogen transmission. Selection for productivity and high throughput also changes the population structure resulting in shorter lifespans and similar ages of farmed animals. Decreased diversity and close contact can facilitate establishment and amplification of pathogens, allowing rapid transmission through the population. Models of avian influenza in Thailand found that both backyard and industrial poultry contributed to outbreaks but industrial production systems were disproportionately more infectious (Walker et al., 2012). When industrial farms have contact with traditional backyard farms, industrial farms may amplify the effects of disease introductions. Additionally, metabolic stress from overcrowding and poor conditions on badly managed commercial farms can weaken animal immune systems, causing increased viral shedding and hyperinfection (Maillard and Sparagano, 2008).

Managing disease risks in industrialized farms necessitates biosecurity and disease control programs. For example, the swine industry has adopted regulations to limit proximity and contact between poultry and pig Concentrated Animal Feeding Operations (CAFOs), which would be difficult to enforce in traditional backyard farming systems (Gilchrist et al., 2007). Risk of epidemic disease outbreaks was speculated to be higher in traditional extensively managed farms due to limited biosecurity measures and frequent contact with wildlife. However, studies of highly pathogenic avian influenza (HPAI) have demonstrated the ability of pathogens to breach biosecurity measures of large commercial farms. In Thailand, for example, large industrial farms had higher relative risks of HPAI infection (Otte et al., 2007). In addition, effective surveillance and disease control programs can limit the spread of disease by isolating or culling infected animals or minimize the consequences of disease introduction, such as through vaccination and treatment. However, antimicrobial resistance due to indiscriminate or excessive agricultural use of antibiotics frequently leads to resistant strains of bacteria. Widespread antibiotic use can alter the host immunity by disrupting normal microbiota and symbiotic bacteria (Dethlefsen et al., 2007). Exposure to pesticides can also negatively affect immunity, increasing susceptibility to infection (Straube et al., 1999). In some cases, vaccination exerts selection pressure for more virulent pathogens, as seen with Marek's disease vaccine selecting for increasingly pathogenic strains of virus (Gimeno, 2008).

Intensified production systems can present additional risks of disease spread due to the need to manage the environment. Large intensive units require ventilation systems to regulate temperature and humidity. Viruses can survive in the dust generated and expelled by ventilation systems, as demonstrated by the airborne spread of influenza between geographically separated poultry units in Canada (Graham et al., 2008). Industrial farming also generates large amounts of animal waste in concentrated geographical areas. In the United States, over half of livestock farms produce more manure than can be feasibly spread on their land (Gollehon et al., 2001). Management of animal waste is poorly regulated in many countries, and its removal and disposal presents further opportunities for disease dissemination. Wastewater lagoons are commonly used to store swine wastes in the United States. Environmental contamination and pathogen spread can result from lagoons breaking or seeping into groundwater. Other methods of managing waste, such as through land application or spray fields, can distribute zoonotic viruses over a larger geographical area (Cole et al., 2000). Increase in zoonotic human hepatitis E cases has been linked to poor sanitation and waste management, as humans are most commonly infected through fecal–oral transmission and contamination of water supplies (Aggarwal and Naik, 2009).

Inputs into livestock systems, such as feed and water, present an additional route of transmission. Feeding livestock animal by-products can expose animals to new pathogens, as demonstrated by the exposure of cattle to contaminated meat and bone meal causing the bovine spongiform encephalopathy epidemic (Imran and Mahmood, 2011). Viruses can also be spread through animal by-products and may not always be activated by feed processing measures (Vinneras et al., 2012). In addition to introducing waterborne diseases, dependence of livestock on groundwater sources can increase risk of exposure to inorganic elements such as fluorine, bromine, arsenic, and lead. Exposure to different constituents can contaminate the food chain and negatively affect livestock susceptibility to disease (Meyer and Casey, 2012).

8.4 DEMOGRAPHIC CHANGES

While population growth and the need for resources drive changes in land use and expansion of agriculture, the availability of food and natural resources also supports population growth and increasing levels of urbanization. Expansion of urban environments can facilitate disease transmission by changing ecosystems, increasing population density, and changing human behavioral patterns. Large urban centers require the development of transport networks, facilitating the movement of people and goods from different areas of the world. Limited resources with high population densities can stress public health measures, further driving disease spread.

8.4.1 Urbanization

By 2050, over six billion people, close to 70% of the global population, are predicted to live in cities (FAO, 2012). Urban development in industrialized countries is usually correlated with improvements in socioeconomic status, and increased levels of urbanization are usually associated with increases in per capita income. In contrast, rapid urbanization in developing countries has caused an expansion of informal settlements leading to health disparities and creation of urban environments favoring disease transmission (Alirol et al., 2011). Of the 100 fastest-growing cities from 1950 to 2000, 75 were in Asia and Africa (Satterthwaite, 2007). In Mumbai, the most densely populated city, populations approach 30 000 people per square kilometer (World Bank, 2012). The overcrowding of people in urban spaces allows rapid transmission of communicable diseases. Human behavioral patterns also affect disease propagation, particularly for sexually transmitted diseases. Changing family structures and behavioral norms and reduced social capital can lead to unsafe sexual behavior and increase in addictions, including intravenous drug use. Analysis of data from national surveys in sub-Saharan Africa found that urban adolescents were more likely to have multiple sexual partners. On the other hand, the same analysis found that urban adolescents were more likely to use condoms due to increased access to sexual education and disease control programs (Doyle et al., 2012). Improvements in sanitation and disease control programs in urban settings can also have positive outcomes, as the reduction of hepatitis A cases in many low- and middle-income countries demonstrated (Tufenkeji, 2000).

In many other contexts, however, urban sanitation systems and infrastructure have struggled to keep up with the rapid population expansion, particularly in slums and low-income areas. The WHO estimates 600 million people in urban settings lack access to adequate sanitation systems and a further 167 million do not have access to safe drinking water (WHO, 2010). Raw sewage collected from cities in the United States, Ethiopia, and Spain was found to harbor over 230 known species of viruses. The high levels of viral diversity present opportunities for recombination and reassortment (Cantalupo et al., 2011). Poor sanitation and water systems increases transmission of waterborne diseases and may create new breeding sites for insect vectors.

The recent resurgence of dengue, a mosquito-borne flavivirus, exemplifies the complex relationships between urban development and infectious diseases. Dengue has reemerged as a major public health problem in urban and peri-urban areas of the tropical and subtropical countries. The spatial distribution of dengue has increased substantially, infecting 50 million people annually. Dengue is associated with urban areas where poor housing and lack of sanitation systems have increased the areas of vector-breeding sites. In the cities of Doula and Yaoundé in Cameroon, urban agricultural practices were shown to influence insecticide resistance levels in mosquitoes (Antonio-Nkondjio et al., 2011). Dengue outbreaks are frequently centered in large cities. For example, the epicenter of the dengue fever outbreak in Brazil was the densely populated city of Rio de Janeiro (Alirol et al., 2011). The level of urbanization was also shown to affect risk of dengue infection in Vientiane, Laos, with infection higher in recently developed urban areas around the periphery of the city center (Vallee et al., 2009). The high mobility of many urban dwellers contributes to the spread of pathogens between different areas of the city and between different cities. Large numbers of migrant workers from dengue-endemic areas have most likely led to the increase of dengue cases in the Middle East (Amarasinghe and Letson, 2012). Local movements and commuting can also introduce diseases into urban environments. Kubiak et al. modeled disease transmission between a small village and city, showing most communities are sufficiently connected to allow disease transmission between commuter towns and urban centers despite spatial segregation (Kubiak et al., 2010).

Urban expansion can also influence animal distribution, creating habitats favorable for some wildlife species. Wildlife such as skunks, foxes, raccoons, and feral dogs inhabit urban areas, scavenging off refuse. Rabies has become a major urban disease problem due to the adaption of mammalian hosts carrying the virus to city environments (Aguirre et al., 2000). Bat populations have also adapted to urban environments; Ebola and Lagos filoviruses were isolated from a bat captured in the urban area of Accra, Ghana, presenting a risk for infection of the human population (Hayman et al., 2010). Urbanization additionally leads to increases in rodent populations, presenting increased opportunities for transmission of rodent-borne diseases. For example, Lassa fever reemerged in West Africa following an explosion of the rodent populations, exacerbated by poor quality housing and hygiene practices allowing infected rodents to access food (Bonner et al., 2007).

Urbanization is also associated with increased levels of stress and pollution, potentially affecting the immunity of both animal reservoirs and people. Physiological responses to stress, such as production of the cortisol hormone, modulate the immune system and can increase susceptibility to disease. Wild birds were found to have higher stress indexes due to interspecific competition in urban environments (Bradley and Altizer, 2007). Exposure to pollutants can produce similar immunosuppressive effects. Ozone, one of the most abundant air pollutants in urban areas, induces oxidative stress, causing airway inflammation and increased susceptibility to respiratory diseases. High levels of ozone pollution were recorded during the emergence of H1N1 pandemic influenza in Mexico City in 2009, and ozone was subsequently shown to increase susceptibility to influenza viruses (Kesic et al., 2012). However, high levels of urban pollution may have opposing effects on some diseases by decreasing survival rates of disease vectors (Awolola et al., 2007).

8.5 LAND USE, DISEASE EMERGENCE, AND MULTIFACTORIAL CAUSATION

As we have seen, changes in land use have important effects on viral diseases, which can be observed throughout the process of disease emergence, from pathogen exposure to the stages of infection and transmission (Figure 8.2). Agricultural development leads to changes to environments and habitats, affecting both the size and contact rates of different populations. Practices such as deforestation and agriculture may create new pathways of pathogen exchange by keeping multiple species in close proximity, increasing opportunities for species jumps from wild and domestic animals to humans, as well as creating additional selection pressures for pathogen evolution. Land use can influence the biological characteristics of viral infections and therefore their chance to survive and spread. Effects of land use can also change the susceptibility of the individuals, such as through weakening immune systems by exposure to pollution or other pathogens. Finally, land uses can facilitate the movement of pathogen, vector, and hosts populations, increasing potential for disease propagation and transmission.

These changes in land use exert complex evolutionary pressures at different levels, from the pathogen and individual host to ecosystem levels. These pressures can both increase and decrease the risk of a disease passing through each stage of emergence and alter the risk of pathogen amplification and spread to new locations. Moreover, land

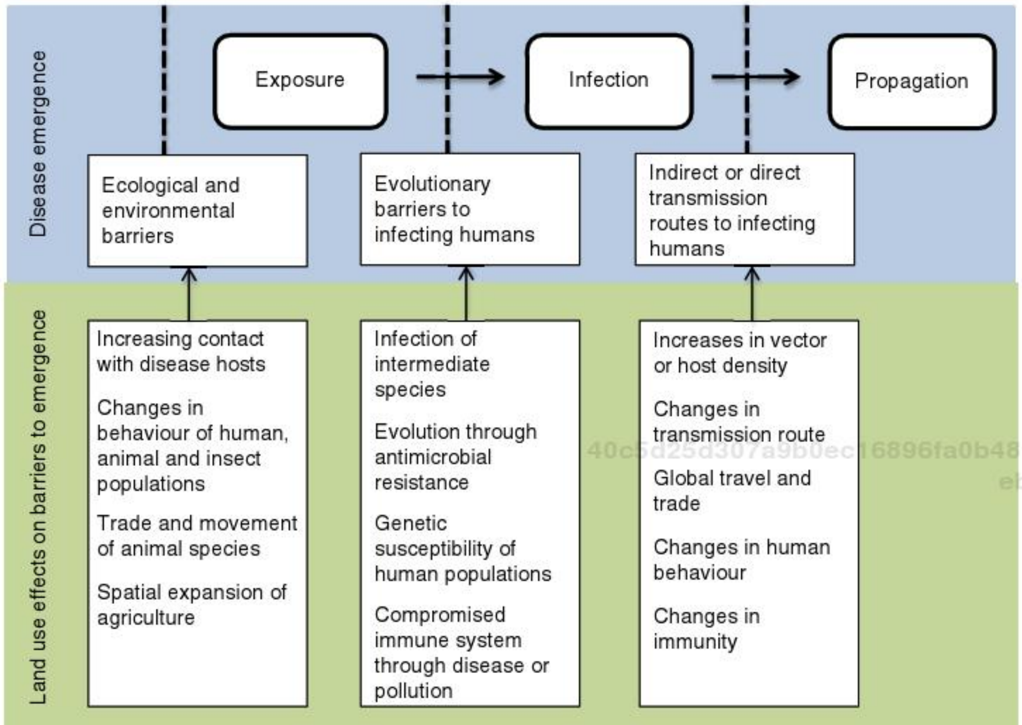

Figure 8.2. Effects of land-use changes on the process of disease emergence. For color detail, please see color plate section.

BOX 8.1 Multiple Effects of Land-Use Changes Causing Nipah Virus Emergence

The case of Nipah virus illustrates how different changes in land use and agricultural systems can interact to cause human disease. The first human outbreaks of Nipah virus, a virus harbored by fruit bats, occurred in Malaysia in late 1998 and 1999, causing over 100 human deaths and costing US$500 million (Field, 2009). The index case for these outbreaks was traced to a 30 000-unit intensive pig farm in the Malaysian forest, where pigs had contact with Nipah-infected fruit bats. The virus was probably passed to the pigs through urine and masticated pellets dropped when the bats fed in overhanging fruit trees. During the initial outbreaks, most human infections resulted from direct contacts with sick pigs or contaminated tissues.

Multiple biological, environmental, and social factors contributed to these complex pathways of disease transmission from wildlife to humans. Increased population growth and demand for food led to spatial expansion and intensification of agriculture, tripling the production of pigs and mangoes within peninsular Malaysia between 1970 and the late 1990s. Increases in pig and mango production were loosely correlated, with mango trees frequently planted in the proximity of commercial pig farms (Pulliam et al. 2012). Mangoes were also used as a food source by wild fruit bats, attracting bat populations to agricultural areas. Wide-scale deforestation and destruction of bat habitats in the region may have additionally contributed to the change in fruit bat migration patterns and their increasing use of lands surrounding mango trees [81]. Finally, high density of homogeneous and susceptible pig populations in intensive production farms facilitated viral persistence and amplification (Chua et al. 2002), while regional trade and pig movements further contributed to transmission to humans.

changes may act synergistically or independently on different disease systems, increasing risks of emergence for one pathogen while simultaneously decreasing risks for another pathogen. There is a need to account for the combined effects of multiple changes on the process of emergence (Box 8.1).

8.6 CONCLUSION

Anthropogenic changes in land use are driven by the need to extract resources and provide food, water, and housing. Rapid population growth combined with income increases also leads to changes in consumption patterns resulting in a rising demand for livestock products and resulting spatial expansion and intensification of agriculture. These positive aspects of land-use change need to be balanced against the environmental changes from both agricultural expansion and natural resource extraction, which if poorly regulated and managed disrupt ecosystems, affecting the composition, density, and behavior of human, animal, and insect populations. Moreover, as populations become increasingly urbanized, this expands trade and travel networks to supply food to cities and allow movement between different areas.

Given the complexity of ecological and evolutionary processes at work, research at the interface between land use, agricultural development, and infectious diseases needs to embrace a holistic approach, able to account for the multiple interactions contributing to produce risk environments in particular ecosystems.

Negative externalities generated from land uses, including disruption of essential ecosystem services and potential for disease emergence and reemergence, need to be valued and where appropriate regulated with a structured decision-making process. At the same time, the policy imperative to manage health hazards needs to be balanced against the many benefits of land uses and agricultural development. Assessing impacts of land-use decisions requires multidisciplinary approaches, evaluating effects on ecosystems, wild, and domestic populations and human health and well-being. Initiatives such as the Millennium Ecosystem Assessment have begun to quantify and value human health costs and benefits from ecosystems and land uses (MEA, 2005). Further research is needed to understand the complex set of drivers influencing each stage of disease emergence and how land planning and management can limit negative consequences of land use in ways appropriate to local contexts, taking into account the local environment, ecology, and socioeconomic development while conforming to international policy, standards and regulations, and surveillance systems.

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