

Chapter 4

Human-Wildlife Contact and Emerging Infectious Diseases

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Abstract The majority of all emerging pathogens in humans are zoonotic (nonhuman animal) in origin. Population, ecological, and behavioral changes that increase contact with wildlife exacerbate emergence of these pathogens. Anthropogenic modification of the physical environment has altered not only our risk of zoonotic infection from wildlife but also the likelihood of pathogen transmission from human to nonhuman animal populations. This is particularly the case for primates that share a number of common infections with humans. In this chapter, I use a series of case studies involving SARS, HIV, Nipah virus, Lyme disease, malaria, and Ebola to exemplify how various anthropogenic factors have facilitated pathogen transmission between human and nonhuman animal populations. The costs and benefits of primate-based ecotourism are also reviewed to better illustrate how human-wildlife contact can affect both populations. Responsible health monitoring of human-wildlife interactions is a necessary prerequisite for prevention of the transmission of future emerging infectious diseases.

4.1 Emerging Infectious Diseases

The patterns of morbidity and mortality in human populations have changed significantly over the past few thousand years. In most populations to date, pestilence and famine decreased as the result of advances in sanitation, medicine, and nutrition. Increases in life expectancy paralleled socioeconomic development and technological changes, and pandemics were replaced with chronic degenerative disorders. Of course, different populations progress through such epidemiologic transitions at different rates and in different times. To use the United States of America as an

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example, rapid development throughout the agricultural and industrial revolutions followed by advances in science and public health drove the US Surgeon General William H. Stewart to reportedly tell the Congress in 1969 that it was “time to close the book on infectious diseases, declare the war against pestilence won, and shift national resources to such chronic problems as cancer and heart disease.” He did not foresee the massive impact that emerging and reemerging infectious diseases would have on human health and economic prosperity worldwide.

Emerging infectious diseases (EIDs) are those that have recently increased in incidence (number of new cases in a given period of time), expanded in geographic range, moved into a new host population, or are caused by newly evolved or discovered pathogens (Lederberg et al. 1992; Morens et al. 2004; Weiss and McMichael 2004). Examples include dengue hemorrhagic fever, West Nile virus, and H5N1 avian influenza. The primary causes of EIDs in human populations have been the anthropogenic modifications of physical and social environments (Cohen 2000; Daily and Ehrlich 1996; McMichael 2004; Morens et al. 2004; Patz et al. 2000, 2004; Weiss and McMichael 2004). Human and livestock populations continue to grow rapidly, increasing the number of hosts potentially susceptible to novel infections. Mass transportation of people, products, livestock, and vectors of disease brings each of these closer to one another, and more quickly (Kimball et al. 2005; Wilson 2003). Population movements due to war, social disruption, and rural-to-urban migration, in addition to general urbanization, increase the densities of nonimmune human hosts and pose significant sanitation problems. Natural disasters and bioterrorism may destroy public health infrastructure and other resources (Watson et al. 2007). Changes in water usage, such as during the construction of dams, culverts, and irrigation systems, can increase the potential breeding sites of vector species like mosquitoes and snails (Keiser et al. 2005; Steinmann et al. 2006). Sex tourism, intravenous drug abuse, reuse of injectable medical equipment, and improper disinfection or ineffective protective measures in hospitals all contribute to the rapid evolution of resistant and deadly pathogens. The misgovernance of epidemics with decentralized testing, misdiagnoses, and underreporting of cases has resulted in slow public health responses to several pandemics (Cyranoski 2005; Normile 2005). Interactions between human and wildlife populations have also been a source of EIDs.

4.2 Wildlife and Emerging Infectious Diseases

Over half of all human infections are zoonotic (nonhuman animal) in origin (Cleaveland et al. 2001; Woolhouse and Gaunt 2007), and the majority of all emerging pathogens in humans are zoonotic in origin (Jones et al. 2008; Taylor et al. 2001). Population, ecological, and behavioral changes that increase contact with wildlife exacerbate emergence of these pathogens (Daszak et al. 2000). Human

encroachment into previously undisturbed areas increases remote area accessibility and introduces more vectors and reservoirs of infection to new hosts. Encroachment, extensification of agricultural land, and urban sprawl all alter population densities and distributions of wildlife, which change disease dynamics (Patz et al. 2004). Forest fragmentation can produce an “edge effect,” increasing the flow of organisms across ecotones, novel species contact, and the likelihood of infection transmission between populations.

Wildlife populations can serve as sinks for human pathogens. That is, increased biodiversity can function as a buffer of hosts in an environment, decreasing the likelihood that vectors of infection would feed off of humans. Areas with increased host diversity could be characterized by a reduced probability of pathogen transmission to vectors as well as a reduced encounter rate between vectors and hosts. “Zooprophylaxis,” or the purposeful use of wildlife and livestock to protect against zoonotic infection, has been utilized to try to guard humans against a number of infections, including encephalitis and malaria (Hess and Hayes 1970). Biodiversity loss due to global climate change, deforestation, the spread of invasive species, overexploitation, and other causes decreases this important buffer, increasing the likelihood of cross species transmission (Maillard and Gonzalez 2006; Pongsiri et al. 2009). Increased inbreeding and decreased genetic diversity in remaining wildlife populations could even facilitate further outbreaks due to impaired immune functions in host animals (ibid.).

Considering all wildlife as a source of human infections would obviously be a grave mistake; protecting wildlife actually serves a selfish purpose of protecting our own health. For example, species-poor areas in the northeastern United States are characterized as having more white-footed mice (*Peromyscus leucopus*), and thus increased incidence of the Lyme spirochete *Borrelia burgdorferi* (LoGiudice et al. 2003). Squirrels and other “dilution hosts” can protect areas from Lyme disease (ibid.). With more hosts in general, and less competent hosts (those that do not support optimal growth and transmission of the pathogen) in particular, *Ixodes* ticks are less likely to feed on humans and successfully transmit Lyme disease. Predatory vertebrates also help protect humans from Lyme disease and many other infections by keeping the abundance of rodent hosts at bay (Ostfeld and Holt 2004).

Maintaining preferential hosts in an environment places a barrier against vector-borne infections. For example, counties in the eastern United States with greater avian diversity exhibit lower incidence of human West Nile virus cases (Swaddle and Calos 2008), thanks in part to the ornithophilic nature of *Culex* and other mosquito species. Similarly, increased small-mammal diversity protects sympatric rodent populations from the spread of Hantavirus infection (Suzán et al. 2009). Such protection represents a largely overlooked ecosystem service. Twenty-five percent (25%) of all extant mammals are now endangered, and approximately 30% of these will become extinct by the year 2050 (Schipper et al. 2008). Overexploitation of animals and environments has and will continue to produce “spillover” of zoonotic infections into human populations.

4.3 Consumption of Wildlife

Responsible human-wildlife contact requires that we carefully consider how to manage wildlife as sources and sinks of human infections. One way in which wildlife serves as a source of infection is via bushmeat, or the use of wild animals for food, medicinal, or traditional cultural purposes, usually involving ingestion. Bushmeat is an important source of protein and income for millions of people, and the illegal bushmeat trade has been facilitated by the use of modern weapons and communication, logging operations that provide access to forests and transportation of products, lack of economic alternatives, and minimal capacity to enforce laws (Karesh and Noble 2009). Current usage rates of bushmeat are unsustainable, well exceeding 100 kg/km²/year in many locations (Robinson and Bennett 2000).

Bushmeat hunting, preparation, and consumption are linked with several pandemics and epidemics, most notably human immunodeficiency virus (HIV), Ebola, and severe acute respiratory syndrome (SARS). The current HIV pandemic, with an estimated global prevalence of 33 million people (UNAIDS 2008), appears to have originated from nonhuman primate simian immunodeficiency viruses (SIV). Through the hunting and butchering of nonhuman primates (resulting in cutaneous or mucous membrane exposure) in West Africa, SIV likely entered into the human population several times and became established as HIV around 1900 in what is now the Democratic Republic of Congo (Worobey et al. 2008). Based on overwhelming genetic similarities (genome structure and protein homology), HIV-1 groups M and N likely originated from chimpanzees (*Pan troglodytes troglodytes*) in Cameroon, group O from western lowland gorillas (*Gorilla gorilla gorilla*) in Cameroon, and HIV-2 from sooty mangabeys (*Cercocebus atys*) in Côte d'Ivoire (Apetrei et al. 2005; Bailes et al. 2003; Gao et al. 1999; Hahn et al. 2000; Keele et al. 2006; Peeters et al. 2002; Santiago et al. 2005; Van Heuverswyn et al. 2006). Direct exposure to nonhuman primate blood through hunting and butchering is common in several populations in West Africa (Wolfe et al. 2004), and SIV has been identified in nonhuman primate bushmeat, pet animals, and bushmeat hunters in West Africa (Apetrei et al. 2005; Kalish et al. 2005; Peeters et al. 2002).

In contrast to HIV, *Ebola virus* (subtypes Zaire, Sudan, Côte d'Ivoire, Bundibugyo, and Reston) is an RNA filovirus that has wiped out several nonhuman primate populations over the past 20 years (Bermejo et al. 2006; Leroy et al. 2004; Walsh et al. 2003). The virus appears to be restricted to the rainforests of central and western Africa and Southeast Asia (Monath 1999; Peterson et al. 2004). Marburg, a related virus, appears to be restricted to dry, open areas of central and eastern Africa, and the distributions of Ebola and Marburg likely reflect natural host distribution (ibid.). Nearly all cases of Ebola in humans can be traced back to the handling or consumption of infected wildlife carcasses, particularly that of apes (Leroy et al. 2004; Pourrut et al. 2005). Although not definitively known at this time, several fruit bat species are suspected to be the natural reservoir host for Ebola (subtype Zaire) and possibly other subtypes (Biek et al. 2006; Leroy et al. 2005).

Bats (genus *Rhinolophus*) may also be the natural reservoir for SARS (Lau et al. 2005; Li et al. 2005; Vijaykrishna et al. 2007; Wang et al. 2006). SARS is a coronavirus (Drosten et al. 2003; Rota et al. 2003) that produced a human epidemic between 2002 and 2004, claiming almost 775 lives (Hughes 2004). The epidemic originated in the Foshan Municipality of the Guangdong Province of China, with the earliest infections confirmed in animal traders (Yu et al. 2003). Several species of animals in the wet market tested positive for SARS, including Himalayan palm civets (*Paguna larvata*), raccoon dogs (*Nyctereutes procyonoides*), and Chinese ferret badgers (*Melogale moschata*) (Guan et al. 2003). Horseshoe bats were likely fed to farmed civets or came into contact with civet farms, and these infected civets were later sold in wet markets, introducing SARS into the general human population. The main February 2003 outbreak can be traced to a single individual who stayed at a hotel in Hong Kong where the pathogen was then spread via aerosolization to ten hotel guests who then traveled to seven different countries, subsequently spreading the pathogen to many individuals, including hundreds of healthcare workers (Hughes 2004). Fear of decreased tourism, travel, and trade was the primary reason for Chinese government officials' underreporting the actual number of casualties, delayed reporting to the World Health Organization, and initial noncooperation with the US Centers for Disease Control and Prevention (Barry 2004; Bloom 2003; Heymann 2004; Kahn 2003; Parry 2003).

4.4 Land-Use/Land-Cover Change and Climate Change

Consumption of wildlife is obviously not the only mechanism by which zoonoses have been introduced into human populations. Vector-borne and waterborne EIDs have been linked in complex ways with climate change (Epstein 2001; Patz et al. 2008). Examples include outbreaks of hantavirus pulmonary syndrome in the southwest United States (Engelthaler et al. 1999), West Nile virus in the United States (Epstein 2001), Rift Valley fever in Kenya (Linthicum et al. 1999), and cholera in Bangladesh (Colwell 1996). Degradation and fragmentation of habitats also forces the overlap of wildlife, domestic animal, and human ecologies. In fact, an important, but often overlooked externality of land-use/land-cover change is the spread of infection. Examples of EIDs linked with climate and land-use/land-cover changes include Nipah virus, Lyme disease, Chikungunya virus, and malaria.

Nipah virus is a single-stranded RNA virus of the family Paramyxoviridae that causes severe acute febrile encephalitis in humans. Like SARS and many other viruses (Calisher et al. 2006; Halpin et al. 2007; Wong et al. 2007), bats (flying fox, genus *Pteropus*) are the natural reservoir of Nipah virus. Nipah virus resulted in over 100 human fatalities in the Kinta district of northern peninsular Malaysia in 1998–1999. The cause of the outbreak is now attributed to a complex interaction of human-induced environmental changes. Fire-mediated deforestation for the expansion of oil palm plantations produced significant respirable suspended-particulate matter in the region. Combined with a drought produced by the El Niño

Southern Oscillation, the availability of flowering and fruiting forest trees was reduced (Chua et al. 2002). Malayan flying foxes (*Pteropus vampyrus*) and island flying foxes (*Pteropus hypomelanus*) began feeding in durian (*Durio zibethinus*) and rambutan (*Nephelium lappaceum*) orchards planted near pig farms. Bat urine as well as partially eaten fruit entered the pigsties, and the virus amplified within the swine. Despite producing severe respiratory disease, the infected pigs were distributed throughout Malaysia. Transmission between bats, pigs, and humans probably happened several times, beginning even before the El Niño event of 1998. It may also have simply been the case that a rapidly expanding pig population increased the probability of contact between bats and pig farms, or that bats were forced to feed in these human orchards because of hunting in other areas (Field 2009). Habitat loss and climate change may also have facilitated viral transmission via nutritional stress, which alters bat physiology, increasing viral seroprevalence and persistence in these animals, increasing the likelihood of horizontal transmission between the flying foxes, and possibly direct transmission between flying foxes and humans (Plowright et al. 2008). Subsequent human outbreaks have been reported in Bangladesh, with transmission largely attributed to *Pteropus giganteus* (Epstein et al. 2006).

Climate is an important determinant of the distribution of arthropod vectors of disease. This is particularly the case of the deer tick, *Ixodes scapularis*, the primary vector for Lyme disease (Brownstein et al. 2003). Suitable habitat for various tick species is expected to increase dramatically in North America and Europe with global warming (Brownstein et al. 2005; Lindgren et al. 2000; Ogden et al. 2005). Already the *Aedes aegypti* mosquito has expanded its geographic range in response to increased global temperatures, resulting in increased risk of Dengue virus in subtropical and temperate climates (Hales et al. 2002). Similarly, the outbreak of hantavirus pulmonary syndrome in the southwestern United States in the early 1990s was linked to heavy summer rains associated with the El Niño Southern Oscillation effect and the subsequent proliferation of pine nuts and deer mice (*Peromyscus maniculatus*), the natural reservoir of the Hantaan virus (family Bunyaviridae) (Engelthaler et al. 1999).

Land-use and climate changes are associated in complicated ways with altered rates of malaria incidence. Malaria is a mosquito-borne disease caused by protozoa of the genus *Plasmodium* (phylum Apicomplexa, order Haemosporidia, family Plasmodiidae), with 172 named species that parasitize reptiles, birds, and mammals (Coatney et al. 1971; Garnham 1966; Levine 1988). Of these, only 4 usually infect humans (*P. falciparum*, *vivax*, *ovale*, and *malariae*), 19 infect nonhuman primates, and 19 infect various other mammals. The parasites are transmitted by over 50 species of female *Anopheles* mosquitoes (Kiszewski et al. 2004). Members of the genus *Plasmodium* likely diverged from the order Haemosporidia around 500 million years ago, perhaps around the time of the Cambrian explosion (Escalante and Ayala 1994). Worldwide climatic changes throughout the last glaciation and the advent of agriculture would have facilitated the spread of the *Anopheles* vector (Coluzzi 1999). During the agrarian revolution, higher-density, sedentary human populations provided mosquitoes with necessary bloodmeals and potential breeding

sites (Hartl 2004; Livingstone 1958). Malaria is now endemic in most tropical regions of the world (Guerra et al. 2008). The global incidence of human malaria is estimated to be more than 300 million new clinical cases each year (Trigg and Kondrachine 1998; WHO 1999). The economic and social impacts of this disease are enormous (Gallup and Sachs 2001).

Deforestation and increased global temperature have led to alterations in mosquito breeding habitats. Forest habitat alterations can create new breeding sites by changing surface-water availability (J. Walsh et al. 1993) and increasing water temperatures (Tuno et al. 2005). The combined effects include more mosquitoes with shortened parasite development time (Afrane et al. 2005, 2006) and increased vectorial capacity (number of infective bites a host receives in a given time period) (Afrane et al. 2008; Vittor et al. 2006). Cultivated areas with elevated water temperatures are also characterized by higher mosquito production rates and increased malaria transmission risk compared to undisturbed areas (Lindblade et al. 2000; Munga et al. 2006). Further shifting of geographic and temporal patterns of incidence of malaria is expected in response to continued climate change (Bhattacharya et al. 2006). This will likely include increased duration of transmission windows, increased mosquito abundance (Pascual et al. 2006), and increased altitudinal distribution of mosquitoes (Ebi et al. 2005; Martens et al. 1995).

The incidence of zoonotic malaria is also expected to increase with continued encroachment of humans into forested areas in Southeast Asia. Zoonotic malaria has probably infected humans for thousands of years, but not until recently has *Plasmodium knowlesi* become recognized as a potential major cause of human morbidity and mortality. *Plasmodium knowlesi* commonly infects long-tailed (*Macaca fascicularis*) and pig-tailed (*Macaca nemestrina*) macaques and is typically transmitted by forest-dwelling *Anopheles hackeri* and *Anopheles latens* in Sarawak, Malaysia, and *Anopheles cracens* in peninsular Malaysia (Vythilingam et al. 2008), and other *Anopheles* mosquitoes of the leucosphyrus group in other countries (Galinski and Barnwell 2009). Many cases of fatal malaria once attributed to *P. malariae* were only recently recognized to have been caused by *P. knowlesi* (Jongwutiwes et al. 2004; Singh et al. 2004). Encroachment into forested areas will likely facilitate continued host switching of malarial parasites (Cox-Singh and Singh 2008). Conflict and natural disasters combined with normal rural-to-urban migration will result in population movements that increase the likelihood of malaria transmission (Martens and Hall 2000).

Other malaria parasites, like *P. cynomolgi* and *P. inui*, that naturally infect some nonhuman primates in Asia may also pose a future zoonotic threat to humans (Galinski and Barnwell 2009). The same could be said for Chikungunya virus, an *Alphavirus* transmitted by *Aedes* mosquitoes and maintained naturally in nonhuman primate and other wildlife populations (Gould and Higgs 2009; Powers and Logue 2007). Infection causes severe joint pain in humans. However, humans will not need to enter forests inhabited by monkeys in order to contract this disease. Rather, this virus is already rapidly spreading to urban areas as well as higher latitudes (ibid.).

Thanks in part to global climate change, EIDs are now affecting residents of even very high latitudes (Greer et al. 2008; Parkinson and Evengård 2009).

Anthropogenic ecosystem disruptions that increase contact between humans, wildlife, and vectors of disease increase the likelihood of not only zoonoses but also anthroozoonoses (pathogens transmitted from human to nonhuman animal populations). Nonhuman animals are very susceptible to our pathogens (Epstein and Price 2009); infectious diseases can cause high levels of morbidity and mortality with subsequent population declines in wildlife (Pedersen et al. 2007; Smith et al. 2009). We must be conscious about how our behaviors, including habitat alteration, meat consumption, and even recreational use of natural areas, may inadvertently affect the risks of zoonotic and anthroozoonotic pathogen transmission.

4.5 Primate-Based Ecotourism

One overlooked aspect of human-wildlife contact (and conflict) includes nature-based tourism. Ecotourism accounts for a significant proportion of all international tourism and contributes billions of dollars to the national income of various countries (Filion et al. 1994). Such revenue can enhance economic opportunities for local residents, support environmental education, and protect the natural and cultural heritage of the area, including the conservation of biodiversity and improvement of local facilities. Wildlife is certainly more valuable alive than dead (Thresher 1981). Unfortunately, rapid, unmonitored development of ecotourism projects can lead to degradation of habitats and deleterious effects on animal well-being (Kuss et al. 1990; Speight 1973). Habituation of animals to human presence can increase the likelihood that animals will actively seek out contact with humans, particularly in the form of crop raiding and invasion of garbage pits and latrines. Habituation also makes wildlife more vulnerable to poaching because of their loss of fear of humans. Other risks associated with ecotourism in protected areas include habitat degradation, pollution, crowding, introduction of invasive species, increased tax burdens for local communities, inappropriate development of infrastructure, and disruption of community activities, particularly when communities do not have adequate control over their involvement in tourism.

Increased human-wildlife contact through ecotourism can contribute to the transmission of bacteria, protozoa, viruses, and helminths through direct and indirect infection routes. Zoonotic and anthroozoonotic infection transmission are of vital consideration given the increasing demand from tourists to experience direct encounters with wildlife. This is particularly the case for primates that are genetically closely related to humans (and thus share susceptibility to many common pathogens) and are threatened by habitat destruction, exploitation and fragmentation, hunting and bushmeat consumption, illegal pet trade, and infectious diseases. Transmission of infections between human and nonhuman primate populations is exacerbated by expansion of local communities, refugee populations, park personnel, researchers, filmmakers and photographers, and tourists. This is despite

the fact that primate ecotourism is increasingly perceived as a venue for promoting awareness about conservation issues. Over the past few decades, primate ecotourism activities have been flourishing throughout Asia, Middle East, Africa, and Central and South Americas.

Wild primates function as reservoirs for a number of human infections, including filariasis, yellow fever, and Chikungunya virus (Mak et al. 1982; McIntosh 1970; Monath 2001). More important, though, nonhuman primates are very susceptible to many human infections (Brack 1987), and transmission of these anthroponoses poses a significant threat to wildlife (Daszak et al. 2000). These animals are usually immunologically naïve to our pathogens, and ape populations can be quickly decimated because of their slow reproductive rates. To date, several infection transmission events from human to nonhuman primate populations have been either suspected or confirmed, including human respiratory syncytial virus and metapneumovirus in chimpanzees in Côte d'Ivoire (Köndgen et al. 2008) and intestinal pathogens *Giardia* and *E. coli* in mountain gorillas and chimpanzees in western Uganda (Goldberg et al. 2007; Graczyk et al. 2002; Rwego et al. 2008). Although local populations or researchers have been the sources of these infections, tourists pose an uncalculated risk to wildlife, which in turn has the potential of producing devastating health and economic outcomes (Muehlenbein and Ancrenaz 2009).

To better understand the risks of anthroponotic infection between tourists and wild nonhuman primates, our research team has surveyed ecotourists at the Sepilok Orangutan Rehabilitation Centre in Sabah, Malaysia. Sepilok is one of the largest primate-based ecotourism destinations in the world, averaging over 100,000 visitors annually. Of 633 individuals surveyed in 2007, over half reported being currently vaccinated against tuberculosis, hepatitis A, hepatitis B, polio, and measles. Fewer participants reported current vaccination status for influenza, rabies, and chicken pox. Despite the fact that the majority of visitors to Sepilok are from temperate regions where influenza is relatively more prevalent, 67.1% of those surveyed with medical-related occupations reported not being currently vaccinated for influenza (Muehlenbein et al. 2008). Respiratory infections, like influenza, would arguably be the easiest to transmit while visiting these primates (primarily orangutan and macaques) at Sepilok. Visitors to other wildlife sanctuaries are likely undervaccinated as well.

Fifteen percent of the 633 tourists self-reported at least one of the following current symptoms: cough, sore throat, congestion, fever, diarrhea, and vomiting (Muehlenbein et al. 2010). Of the sample of tourists, 10.8% self-reported at least one symptom associated with respiratory tract infection (cough, sore throat, or congestion). The participants with recent animal contact (e.g., livestock, wildlife at other sanctuaries, unfamiliar domestic pets) were more likely to report current respiratory symptoms compared to individuals with no such animal contact. Similarly, participants with a medical-related occupation were more likely to report current respiratory symptoms while at Sepilok compared to participants with nonmedical occupations. That is, currently ill and potentially infectious tourists were still visiting a wildlife sanctuary to view endangered species, despite having

at least some basic knowledge about infection transmission (i.e., medical-related occupation), or having had animal contact immediately prior to arriving. While participants in nature-based tourism are generally concerned about environmental protection, analyses suggest that a significant proportion of ecotourists are either uninformed of the risks they may pose to nonhuman animal health or chose to ignore such risks. These tourists underestimate their own risk of infection as well as their potential contribution to the spread of diseases.

Despite a plethora of best-practice guidelines produced by various tourism and conservation organizations (Ceballos-Lascuráin 1996; Christ et al. 2003; Eagles et al. 2002; Higginbottom 2004; UNEP/WTO 2005; Wood 2002), tourists are simply not being adequately informed about the risks of zoonotic and anthroozoonotic diseases. Tourist ignorance over infection risks, particularly the risks they pose to the very same wildlife they are interested in protecting, cannot be justified, regardless of the large sums of money people spend to visit these exotic destinations. It is therefore the combined responsibility of the tourism and medical communities to more accurately communicate the risks of zoonotic and anthroozoonotic infections in ways that best support the needs of humans and wildlife alike. The global management of zoonotic and anthroozoonotic epidemics is an obligation that transcends any one discipline. Unregulated ecotourism, climate and land-use/land-cover changes, and bushmeat consumption can all exacerbate human-wildlife contact and the subsequent emergence and reemergence of infectious diseases. Yet arguably these human-wildlife interactions are manageable to some degree, given the right resolve and resources. Responsible health monitoring of human-wildlife interactions is a necessary prerequisite for prevention of future emerging infectious diseases.

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