The Journal of Experimental Biology 213, 955-960

doi:10.1242/jeb.037663

Deforestation and avian infectious diseases

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Accepted 23 December 2009

Summary

In this time of unprecedented global change, infectious diseases will impact humans and wildlife in novel and unknown ways. Climate change, the introduction of invasive species, urbanization, agricultural practices and the loss of biodiversity have all been implicated in increasing the spread of infectious pathogens. In many regards, deforestation supersedes these other global events in terms of its immediate potential global effects in both tropical and temperate regions. The effects of deforestation on the spread of pathogens in birds are largely unknown. Birds harbor many of the same types of pathogens as humans and in addition can spread infectious agents to humans and other wildlife. It is thought that avifauna have gone extinct due to infectious diseases and many are presently threatened, especially endemic island birds. It is clear that habitat degradation can pose a direct threat to many bird species but it is uncertain how these alterations will affect disease transmission and susceptibility to disease. The migration and dispersal of birds can also change with habitat degradation, and thus expose populations to novel pathogens. Some recent work has shown that the results of landscape transformation can have confounding effects on avian malaria, other haemosporidian parasites and viruses. Now with advances in many technologies, including mathematical and computer modeling, genomics and satellite tracking, scientists have tools to further research the disease ecology of deforestation. This research will be imperative to help predict and prevent outbreaks that could affect avifauna, humans and other wildlife worldwide.

Key words: deforestation, birds, infectious diseases, disease ecology, global change.

Introduction

The number of catalogued emerging infectious diseases (EIDs), and especially those that originated in wildlife, has increased in the last several decades (Jones et al., 2008). Factors contributing to the emergence of infectious diseases include urbanization, human population growth, agricultural development, wildlife trade, the loss of biodiversity and pathogen pollution through invasive species (Aguirre and Tabor, 2008; Daszak et al., 2000; Dobson and Foufopoulos, 2001; Molyneux, 2003; Patz et al., 2008; Vora, 2008). In addition, it is likely that climate change will contribute to novel occurrences of disease transmission (Jones et al., 2008; Lafferty, 2009; Patz et al., 2008; Rogers and Randolph, 2006). The possible results of climate change on disease emergence include direct effects of temperature, rainfall and sea level on pathogens, hosts and vectors (Patz et al., 2000). However, whether the effects of climate change will lead to a net increase in EIDs or rather a shift in their distributions is still under debate (Lafferty, 2009). Perhaps even more immediate than these other factors are the implications of large-scale deforestation on disease emergence. The global rate of tropical deforestation appears to be increasing readily; between 2000 and 2005, more than 2.3% of the tropical humid forests were cleared (Hansen et al., 2008). Brazil and other nations of the Americas account for the majority of these losses (Hansen et al., 2008). Rates of deforestation in parts of Africa are near 1% per year (FAO, 2009). Deforestation is also increasing in temperate regions, due to logging in North America, Russia and Europe (FAO, 2009). Deforestation can transform whole ecosystems and thus affect disease transmission (Taylor, 1997). The field of deforestation disease ecology is in its infancy, with relatively few scientific studies published relative to such a globally pertinent threat. This review will highlight some recent work exploring both the direct and possible indirect effects of deforestation on infectious diseases of birds.

Deforestation can be defined as the conversion of forested areas to non-forest land through cutting, clearing and removal of rainforest or related ecosystems into less biodiverse ecosystems such as pasture, cropland, plantation, urban use, logged area or wasteland (Kricher, 1997). Presently, human activities such as logging, road construction, mining and agricultural development cause the majority of deforestation (FAO, 2009). Deforestation is associated with a loss of biodiversity (Gardner et al., 2009; Vieira et al., 2008). One particularly compelling example showed that the creation of small, isolated, forest fragments in Brazil resulted in local extirpation of bird species and overall reduced avian biodiversity (Ferraz et al., 2007). Direct effects of deforestation include the alteration of many fundamental aspects of ecosystems, including soil and water conditions, microclimates and the local biota (Taylor, 1997; Yasuoka and Levins, 2007).

There have been several examples of the direct effects of deforestation on human diseases. It is clear that habitat alteration can affect the prevalence and incidence of human malaria. The effects of deforestation on anopheline mosquitoes, the vectors of human malaria, are complex, and can be influenced by the nature of agricultural development and specific local ecological characteristics (Yasuoka and Levins, 2007). However, increased levels of sunlight, as associated with open spaces, have been correlated with increased anopheline mosquito densities (Yasuoka and Levins, 2007). In addition, studies have shown that forest clearing increased the observed human feeding rate of *Anopheles darlingi*, the major vector of *Plasodium falciparum* in Peru (Vittor et al., 2006; Vittor et al., 2009). Other human vector-borne diseases are also affected by deforestation (Walsh et al., 1993). For example, the blackflies that transmit severe onchocerciasis increase in density with deforestation (Wilson et al., 2002), Lyme disease has increased with forest fragmentation in North America (Allan et al., 2003;

Brownstein et al., 2005; Killilea et al., 2008), and cutaneous leishmaniasis is increasing with deforestation in Costa Rica (Chaves et al., 2008). Deforestation has also been associated with the increased emergence of viral pathogens, such as SARS, Ebola and other viruses of bats (Field, 2009; Leroy et al., 2005; Looi and Chua, 2007). In addition, deforestation can indirectly result in increased human contact with wildlife, increasing the exposure to zoonoses (Wolfe et al., 2005; Wolfe et al., 2007). For example, it is now clear that human/chimpanzee contacts led to the HIV pandemic (Keele et al., 2006; Keele et al., 2009), and may have provided the first transmission of Falciparum malaria (Rich et al., 2009).

Birds are affected by viral, bacterial, parasitic and fungal infectious pathogens, and can also act as reservoirs of numerous zoonotic pathogens (Davis, 1971; Hubalek, 2004; Thomas and Hunter, 2007). It is likely that the direct and indirect effects of deforestation could affect the transmission of all the four major classes of avian pathogens.

Viruses

Avian influenza

The most topical virus of birds is the highly pathogenic avian influenza H5N1. Avian influenza viruses reside in wild aquatic birds; they are the natural reservoir for all influenza A viruses (Olsen et al., 2006; Stallknecht et al., 2008; Yee et al., 2009). They are also found in many passerine birds and birds of other orders (Kelly et al., 2008; Olsen et al., 2006; Stallknecht st al., 2008). Avian influenza is generally spread through the fecal–oral route, can survive for weeks or months in cold water, or years in ice, and has been documented to spread through bird migration, and also through the legal and illegal transport of birds (Gilbert et al., 2008; Kilpatrick et al., 2006; Stallknecht and Brown, 2009; van den Berg, 2009; Webster and Govorkova, 2006; Webster et al., 2006). Because influenza virus has a single-stranded, segmented RNA-based genome, it has a high potential for adaptive change as a human pathogen, and thus is of worldwide concern (Skeik and Jabr, 2008; Stallknecht et al., 2008).

Global change will affect the spread of avian influenza viruses through several mechanisms. With global warming, bird distributions will shift northwards in the northern hemisphere (and southwards in the southern hemisphere), and there is already evidence for changes in migration timing (Carey, 2009; Hitch and Leberg, 2007; La Sorte and Thompson, 2007; Louchart, 2008; Parmesan and Yohe, 2003). Climate change will result in changes in local species compositions, and these changes may cause the redistribution of avian influenza into different age classes, species and flyways. However, to date, it is unknown whether climate change has contributed to the emergence of highly pathogenic H5N1 avian influenza as a global disease (Gilbert et al., 2008). Rather, the more immediate effects of the international poultry trade, in combination with avian migration, appear to have contributed to the long-distance spread of the virus (Kilpatrick et al., 2006; van den Berg, 2009; Yee et al., 2009). In addition, the agricultural claim of wetlands, the breeding/feeding grounds for aquatic birds, will lead to higher contacts between wild and domestic avifauna (Gilbert et al., 2008). Presently, there are no studies directly linking deforestation with the spread of avian influenza; however, it is possible to speculate that habitat transformation could contribute to altered flyways, and increased contact among different avian populations. In addition, it is known that terrestrial birds can harbor avian influenza (Kalthoff et al., 2009; Kelly et al., 2008) but it is unclear how landscape transformation may affect disease transmission in this avifauna.

West Nile virus and arboviruses

West Nile virus (WNV) is a flavivirus spread by mosquitoes; it causes significant mortality in birds, humans and other vertebrates and appears to have caused large-scale declines in North American bird populations (Kramer et al., 2007; LaDeau et al., 2007). The virus is commonly found in bird species that predominate in habitats of low biodiversity, i.e. the American crow, house sparrow, American robin and house finch. In less-disturbed habitats, these reservoir species are less common or absent (Ostfeld, 2009). The dilution effect hypothesis predicts that mosquitoes occurring in areas of low avian diversity should have a high probability of finding a suitable reservoir host for WNV, and that areas of high biodiversity should maintain lower prevalences of WNV (Allan et al., 2009; Ostfeld, 2009). Indeed, a recent study reported a lower incidence of human WNV in counties of the United States that have greater avian (viral host) diversity (Swaddle and Calos, 2008). Similarly, infection rates of mosquitoes with WNV have been shown to be inversely correlated with the species richness of non-passerine birds and wetland cover (Ezenwa et al., 2006; Ezenwa et al., 2007). However, another study in the Chicago area suggests that other factors such as variation in mosquito host preference, reservoir host competence, temperature and precipitation, may be more important than species richness in predicting the spread of WNV (Loss et al., 2009). The effects of landscape transformation on the spread of WNV are thus not entirely clear but evidence suggests that deforestation, accompanied by the subsequent loss of biodiversity and altered microclimates, may lead to altered patterns of viral transmission.

Birds are also the reservoir for another vector-borne flavivirus, Japanese encephalitis, common to Asia (van den Hurk et al., 2009). With more than 10,000 human deaths yearly, this virus appears to be increasing in prevalence. Ardeid birds, for example black-crowned night herons, little egrets and plumed egrets, are the maintenance hosts of this virus, which is often transmitted to pigs and then humans through its major mosquito vector *Culex tritaeniorhynchus* (Mackenzie and Williams, 2009). Much of its spread in Asia is believed to be due to deforestation for increased agriculture, especially for the irrigation of rice fields (Mackenzie and Williams, 2009). Here the intensification of human land use, and the subsequent loss of forested areas, has resulted in increased breeding and foraging opportunities for the avian hosts, along with increased contact with humans and domestic mammals. Although the virus does not significantly impact birds, the results are increased incidences of disease in humans. Besides these examples, there are many other avian viruses that can be spread with migratory birds (Hubalek, 2004), where transmission could be similarly affected through the indirect effects of deforestation.

Bacteria and fungi

Birds carry many bacterial and fungal pathogens. Several are potential zoonotics, among them *Mycobacterium avium*, which causes avian tuberculosis, *Vibrio cholerae*, the cause of cholera, and *Borrelia burgdorferi*, the agent of Lyme disease (Hubalek, 2004). *Mycoplasma gallisepticum*, the cause of several outbreaks of eye lesions in house finches (*Carpodacus mexicanus*), has been widely studied since its initial outbreak in 1993 in the eastern United States (Dhondt et al., 2005; Hawley et al., 2006; Hochachka and Dhondt, 2000; Hosseini et al., 2006). Fungal pathogens include the yeast-like fungi *Candida albicans* and *Candida tropicalis* and the agent of aspergillosis *Aspergillus fumigatus* (Hubalek, 2004). However, the deforestion ecology of these pathogens is understudied. Studies on mammals can inform research on avian pathogens. One study in primates has shown that forest fragmentation in Uganda is associated with an increased similarity of *Escherichia coli* bacteria between humans and red-tailed guenons, suggesting increased transmission between the two populations (Goldberg et al., 2008). Another study has shown that an increase in the prevalence of *Mycobacterium ulcerans*, a pathogen that causes Buruli ulcer in humans, appears to be correlated with deforestation in West Africa (Walsh et al., 2008). It is likely that forest fragmentation is having similar effects on avian bacterial and fungal pathogens. The spread of pathogens will probably go unnoticed in most cases because transmission will be among unstudied bird species. However, the results may be a loss of more endemic species, and thus an overall loss in biodiversity (Pedersen et al., 2007; Williams et al., 2002).

Avian malaria and other haemosporidians

Haemosporidian parasites (Haemosporida) of the genera *Plasmodium*, *Haemoproteus* and *Leucocytozoon* are transmitted by diverse and widely distributed blood-sucking dipteran insects (Valkiūnas, 2005). The vectors of avian malaria parasites (*Plasmodium* spp.) are Culicidae mosquitoes belonging to eight genera; most commonly species of *Culex* and *Culiseta* (Valkiūnas, 2005). Recently mosquitoes of the genus *Coquelletidia* have also been described as vectors (Njabo et al., 2009). *Haemoproteus* spp. of songbirds are transmitted by blood-sucking biting midges of the *Culicoides*, and *Leucocytozoon* spp. are transmitted by blackflies belonging to the Simuliidae (Valkiūnas, 2005). These parasites are cosmopolitan and found worldwide on all the continents except Antarctica.

Several studies have begun to address the relationship between avian haemosporidian parasites and various ecological determinants (Valkiūnas, 2005). For example, one study showed marked and complex associations between avian malaria infections and landscape on a local scale, within a single population of birds (Wood et al., 2007). There has been extensive work conducted to determine the prevalence and diversity of these parasites in rainforest birds in West-Central Africa (Beadell et al., 2009; Sehgal et al., 2005; Waldenström et al., 2002). This region of Africa is highly relevant to the investigation of effects of habitat modification due to continued high rates of deforestation and forest fragmentation (FAO, 2009). Some recent work has focused on the interplay between the distribution of haemosporidian infections and deforestation. One examined the prevalence of infection in both pristine and disturbed forests from birds over a broad geographical range in Cameroon (Bonneaud et al., 2009). In contrast to results found with human malaria (Vittor et al., 2006), the authors found a higher prevalence of *Plasmodium* lineages in pristine as compared with disturbed forest sites (Bonneaud et al., 2009). The authors suggested five non-mutually exclusive hypotheses to explain these findings: (1) that disturbed forest sites may impart a fitness cost on host species, and thus the birds most weakened by the parasites were not sampled; (2) pristine forests may offer better breeding grounds for vectors than disturbed forest sites; (3) increased competition among insect vectors at disturbed sites may cause the decline in numbers of mosquitoes susceptible to avian *Plasmodium*, resulting in lower prevalence of infection in birds; (4) vectors may have altered feeding preferences in disturbed forests, and feed on non-avian species; and (5) there may be an increase in the density of alternative hosts which may be less susceptible to infection; thus, resulting in a decrease in parasite prevalence through the dilution effect (Bonneaud et al., 2009).

To further investigate these hypotheses, a recent study conducted a large-scale survey in Cameroon sampling birds at deforested and intact sites (Chasar et al., 2009). The sites were chosen in pairs, one deforested within a few kilometers of a pristine site; thus, eliminating confounding abiotic variables such as temperature and rainfall from the analyses. They studied the diversity, prevalence and distribution of avian malaria parasites (*Plasmodium* spp.) and other related haemosporidians (species of *Haemoproteus* and *Leucocytozoon*) from these sites in two widespread species of African rainforest birds, the yellow-whiskered greenbul (*Andropadus latirostris*) and the olive sunbird (*Cyanomitra olivacea*). Twenty-six mitochondrial cytochrome *b* lineages were identified: 20 *Plasmodium* lineages and six *Haemoproteus* lineages. These lineages showed no geographical specificity or significant differences in lineage diversity between habitat types. However, akin to the study by Bonneaud et al. (Bonneaud et al., 2009), they found that the prevalence of *Leucocytozoon* and *Haemoproteus* infections were significantly higher in undisturbed than in deforested habitats (*Leucocytozoon* spp. 50.3% *vs* 35.8%, *Haemoproteus* spp. 16.3% *vs* 10.8%). They also found higher prevalence for all haemosporidian parasites in *C. olivacea* than in *A. latirostris* species (70.2% *vs* 58.2%).

In contrast to findings by Bonneaud et al. (Bonneaud et al., 2009), the study by Chasar et al. found the effects of deforestation on *Plasmodium* parasite prevalence to be quite complex (Chasar et al., 2009). One morphospecies of *Plasmodium* in the olive sunbird, as represented by a clade of related lineages, showed increased prevalence at disturbed sites, while another showed a decrease (Fig. 1). More specifically, it was found that lineages representing *P. megaglobularis*, exhibited a higher prevalence in undisturbed habitats, and lineages of *P. lucens* exhibited higher prevalence in

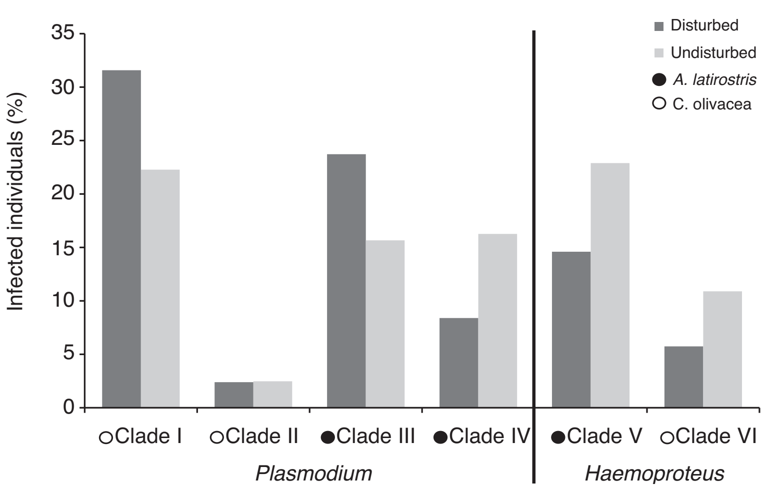

Fig. 1. Effects of deforestation on the prevalence of *Plasmodium* and *Haemoproteus* species in two common African rainforest birds. The prevalence of different *Plasmodium* species differs between disturbed and undisturbed sites. The figure depicts haemosporidian parasite prevalence of different clades of mitochondrial cytochrome *b* lineages; each clade probably representing a distinct morphospecies. Percentage prevalence for each clade recorded from all disturbed (gray bar) and undisturbed (light gray bar) study sites. The *y*-axis is the prevalence of infection as a percentage. *Andropadus latirostris*=yellow-whiskered greenbul, *Cyanomitra olivacea*=olive sunbird. Clade I=*Plasmodium globularis*, clade II=*P. multivacuolaris*, clade III=*Plasmodium lucens*, clade IV=*Plasmodium megaglobularis*, clade V=*Haemoproteus* sp., clade VI=*Haemoproteus vacuolatus* (modifed from Chasar et al., 2009).

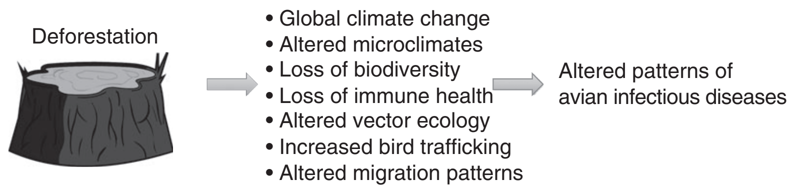

Fig. 2. A summary of potential effects of deforestation on avian infectious diseases.

disturbed habitats. These results help elucidate the complex distribution of parasites, and stress the importance of studying groups of related lineages of parasites representing separate morphospecies, as verified by microscopy. This study also emphasizes that summarizing distribution data of all lineages belonging to one haemosporidian genus might mask some patterns of infection distribution. It is known that different species of avian malaria can have different pathological effects on different bird species (Palinauskas et al., 2008; Valkiūnas, 2005). Thus, grouping data together at the generic level should be avoided in studies of ecological and evolutionary biology.

Now with these data, it will be crucial to do further research to better understand the underlying causes of these patterns of parasite prevalence at degraded forest sites. As mentioned above, there are several hypotheses that can account for these contrasting effects of habitat on parasite prevalence. The data from Chasar et al. (Chasar et al., 2009) may be a clear example of a parasite–host association being driven by vector specificity, with the individual species of vectors competing with variable success in deforested areas. The study suggests that distinct parasite morphospecies may be transmitted by species of mosquitoes that are affected differently by changes in habitat. This is not surprising because habitat degradation has been shown to have variable impacts on the diversity of mosquito species (Vittor et al., 2006; Vittor et al., 2009; Yasuoka and Levins, 2007). It will be essential to do a thorough investigation into the vector ecology at forested *vs* deforested sites. Alternatively, it is known that environmental factors can affect host immunity (Harvell et al., 2009). The composition of the parasite community could be determined by the variation in selection pressures imposed by the host's individual immunity on different parasite strains. In any case, the data illustrate that transmission patterns, even among closely related lineages of avian malaria, can differ in relation to deforestation, and that anthropogenic habitat change can affect the prevalence of haemosporidian parasites in wild bird populations.

This work has implications for endemic populations of birds that lack a robust immune response to avian malaria, such as the Hawaiian honeycreepers (Atkinson et al., 2000; van Riper et al., 1986; LaPointe et al., 2009; Reiter and LaPointe, 2007). The disease is thought to have contributed to the decline of many native bird species in Hawaii, and deforestation could further alter the patterns of malaria transmission (Atkinson and LaPointe, 2009; van Riper et al., 1986). For example, it has been suggested that deforestation of the Alaka`i Wilderness Preserve on Kaua`i Island could alter the pattern of seasonal transmission of avian malaria to a pattern of continuous yearly transmission (Atkinson and LaPointe, 2009).

In continental North America, the spotted owl (*Strix occidentalis*) is one of the most publicized bird species threatened by deforestation (Hanson et al., 2009; Noon and Blakesley, 2006; Stokstad, 2008). They are also threatened by the range expansion of the barred owl (*Strix varia*), which appears capable of utilizing habitats in both disturbed and undisturbed conditions (Dark et al., 1998). In a study by Ishak et al., many lineages of haemosporidian parasites were found in the spotted owl (Ishak et al., 2008). The authors found the highest lineage diversity of the parasite *Leucocytozoon* in spotted owls, as compared with 12 other owl species. In addition, the authors found 12 lineages that were unique, found only in spotted owl hosts (Ishak et al., 2008). In addition, the study reported the first known infection of spotted owls with avian malaria (*Plasmodium* species). Although this parasite may have already been present at low levels in local bird populations, this *Plasmodium* parasite lineage differed by only one base change in the mitochondrial cytochrome *b* sequence from a parasite found in the invasive barred owl (Ishak et al., 2008). Thus, the effects of the range expansion of the barred owl, due to its better success in more open habitats, may be an example of 'pathogen pollution' that arrives with an invasive species. Future work should focus on the combination of blood parasite analyses along with immunity tests and estimated annual survival and reproductive rates for infected spotted owls.

General conclusions and future directions

The impacts of deforestation on avian infectious diseases will be diverse and in many cases go undetected. Effects can range in scale from local changes in microclimate to large-scale global climate change and loss of biodiversity (see Fig. 2). The direct effects of logging and clearing for agriculture can be increased occurrences of standing water that could provide suitable breeding grounds for insect vectors. The species composition of vector communities can thus be rapidly altered with clear-cutting. Similarly, subsequent to forest clearing follows an immediate redistribution of remaining bird populations; thus, potentially bringing birds into contact with species that they normally do not encounter. Greater interactions among bird species can result in increased transmission of infectious diseases.

In addition, loss of suitable habitats could alter migration patterns, with results similar to what is expected with global climate change (Gilbert et al., 2008). Altered flyways can result in species redistributions, again leading to increased inter-species contacts and transmission of pathogens. Large-scale deforestation will indeed contribute to global climate change, and it is difficult to separate the effects of the two phenomena on disease transmission (Cramer et al., 2004; Shukla et al., 1990), although the effects of deforestation will probably be more immediate and perceptible.

Deforestation could have subtle indirect effects on the fitness of avian populations. Theory predicts that habitat fragmentation can contribute a decrease in genetic diversity. A study of blue manakins (*Chiroxiphia caudata*) revealed significant genetic structure among populations, and suggested that future forest fragmentation could result in inbreeding (Francisco et al., 2007). Decreased genetic diversity can contribute to a decreased immune health of threatened bird species, so as to make them more susceptible to invasive pathogens (Tompkins et al., 2006). In addition, the clearing of forests can lead to an increase in bird trafficking, as humans gain access to more forest tracts, again leading to an increase in EIDs (Wolfe et al., 2005). Overall biodiversity decreases with deforestation, and this can lead to an increase in EIDs in birds. Thus, in conservation biology, it is imperative to begin large-scale studies on the impacts of deforestation on infectious diseases.

Future studies of the disease ecology of deforestation will take advantage of several technologies that will accelerate the research and increase scientific understanding. First, genomics is revolutionizing all aspects of biology, and will play an important role in the conservation of wildlife (Romanov et al., 2009). Soon it may be possible to perform metagenomic analyses of whole forest ecosystems, to assess the effects of deforestation on the spread of EIDs. For example, the diversity of viruses found in vectors in deforested areas could be compared with those from intact areas. Similarly proteomics and microarrays will play important roles in assessing the relationships of pathogens in avian hosts. These technologies will help elucidate the effects of habitat loss on immune health, and also allow the detection of undescribed pathogens. Second, remote sensing is providing predictive models regarding the spread of important pathogens of humans (Ageep et al., 2009; Brooker, 2007; Hay and Lennon, 1999; Kalluri et al., 2007), and can also be used to model EIDs in birds. Third, computer and mathematical modeling of deforestation will allow theoretical studies where empirical studies on deforestation prove unfeasible or unethical (Manson and Evans, 2007). Finally, satellite tracking and other monitoring technologies have become increasingly feasible with birds and will greatly aid in understanding the movements of migratory birds (Nowak and Berthold, 1991; Stutchbury et al., 2009), and thus the spread of infectious diseases.

Birds are important model organisms for the study of the evolution and ecology of disease. Given the socioeconomic patterns of disease in humans, the use of birds for the study of infectious diseases is advantageous because they represent natural populations that can be studied in both human-impacted environments and in pristine unaltered forests. Studies on the disease ecology of deforestation could potentially avert human pandemics and ultimately provide data for policy decisions that could lead to the conservation of the planet's forests.

Acknowledgements

The author thanks The Company of Biologists for the invitation to write this review and to the symposium 'Survival in a Changing World'. The author also is grateful to two anonymous reviewers for helpful suggestions regarding the manuscript. R.N.M.S.'s research program was funded by the joint NSF–NIH Ecology of Infectious Diseases Program award EF-0430146. Deposited in PMC for release after 12 months.

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