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Forests and emerging infectious diseases: unleashing the beast within

Jean-François Guégan^{1,2,3} , Ahidjo Ayouba⁴ , Julien Cappelle^{3,5} and Benoît de Thoisy⁶

¹ UMR MIVEGEC, IRD, CNRS, Université de Montpellier, Centre IRD de Montpellier, B.P. 64501, 34394, Montpellier Cedex 5, France

² Comité national français sur les changements globaux (CNFCG), Paris, France

³ UMR ASTRE, INRAE, CIRAD, Université de Montpellier, 34398, Montpellier Cedex 5, France

⁴ UMI TransVIHMI, IRD, INSERM, Université de Montpellier, centre IRD de Montpellier, B.P. 64501, 34394, Montpellier Cedex 5, France

⁵ UMR EpiA, INRAE, VetAgro Sup, 69280, Marcy-l'Etoile, France

⁶ Laboratoire des interactions Virus-Hôtes, Institut Pasteur de la Guyane, 23 Avenue Pasteur, BP 6010, 97306, Cayenne Cedex, French Guiana

E-mail: jean-francois.guegan@inrae.fr

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Abstract

Deforestation and associated changing landscapes are major components of environmental changes, with important implications for ecosystem functioning and biodiversity conservation. Tropical forests are hot spots of biodiversity and provide multiple goods and ecosystem services which benefit *people* in many ways. Forests also play an important role in health-related legends, myths, and fairy tales from all over the world, and are important sources of new potential emerging microbial threats to humans. Although plausibly numerous abundant microbial forms with a forest origin may exist, our systematic literature review shows that forest-derived infection studies are relatively unexplored, and both taxonomically and geographically biased. Since biodiversity has been associated with emergence of novel infectious diseases at macro-scale, we describe the main biogeographical patterns in the emerging infection-biodiversity-forest loss nexus. Then, we illustrate four fine-scale case studies to decipher the underlying processes of increased infection risk in changing forest clearing landscapes. Finally, we identify scientific challenges and regional management measures required to mitigate these important new emerging threats.

1. Introduction

In a general perspective, all people have some dependence on forests. Forests vividly affected the imagination of preliterate societies, and over time, they have come to represent different concepts in the imaginations of populations living in various geographical locations. In addition to these positive views, woodlands are also believed to be inhabited by mythical beings, trying to harm humans, or to be a great danger and inhospitable areas of evil and darkness [1], and of mysterious diseases [2–4]. In modern times, many people rely directly on forests for their livelihoods; woodlands provide sustenance, building materials and medicinal plants. People who live inside forests are often hunter-gatherers or shifting cultivators, and people living near forest are usually involved in agriculture outside the forest, and regularly use forest product partly for their own subsistence purposes. Lastly, people living outside forest, including

urban dwellers, are engaged in commercial activities and forest industries, and use, at least, timber products and paper. Although paucity of data makes it difficult to draw definite conclusions about the state of many tropical forest ecosystems, precluding a global understanding of impacts of current disturbances on animal vital rates [1, 5], many forest ecosystem services could be lost forever, as highlighted for instance by Amazon rainforest decimated by widespread fires and logging [2, 6, 7]. As Earth's forested areas and their native biodiversity could become progressively scarce and remain under increasing pressures for resource extraction and land conversion this new anatomy could create novel conditions for pervasive risks to emerge and interact in the longer term [8] (see table 1 and figure 1).

These continued environmental changes interacting with human and animal demographics on Earth might reasonably be shown to have a greater immediate impact on current and future zoonotic and

Table 1. List of forested regions in the world surrounded by densely populated areas, and having large number of exposed people below poverty line.

The forest region in Nepal. See subfigure A on figure 1. Between 1990 and 2005, Nepal lost 24.5% of its forest cover (*c.a.* 1181 000 hectares). Measuring the total rate of habitat conversion (defined as change in forest area plus change in woodland area minus net plantation expansion) for the 1990–2005 interval, Nepal lost 7.9% of its forest and woodland habitat.

The forest region in Bangladesh. See subfigure A on figure 1. By 1980 only about 16% of the land was forested, and forests had all but disappeared from the densely populated and intensively cultivated deltaic plain. The largest areas of forest are in the Chittagong Hills (more than 4600 square kilometers) and the Sundarbans (nearly 6000 square kilometers).

The forest regions north of Goma and Kinshasa (Democratic Republic of Congo). See subfigure B on figure 1. Since 1990 till 2015, the rate of deforestation in the DRC has remained constant at 0.20%, which equates to the loss of 311 000 hectares annually. From 2015 to 2019 the rate of deforestation in the Democratic Republic of Congo has doubled.

The forest region around Douala (Cameroon). See subfigure C on figure 1. Forest land cover in Cameroon has declined for the last 25 years (1990–2015) with a loss of around 1.0% forest cover per year, which is one of the highest deforestation rates in the Congo Basin.

The forest regions around Pointe-Noire and Loango (Congo). See subfigure D on figure 1. About 65% of the Republic of Congo is covered in dense rainforest. Deforestation rates are relatively low, compared to many other tropical forest countries; the annual rate of deforestation for the period 2010–15 was 0.1%.

The forest regions west of Mejicanos and San Salvador (El Salvador). See subfigure E on figure 1. El Salvador is the second most deforested country in Latin America after Haiti. Almost 85% of its forested cover has disappeared since the 1960 s, leaving about 5% of the land area forested. Less than 6000 hectares are classified as primary forest.

The forest region around Mixco (Guatemala). See subfigure E on figure 1. Deforestation is rife in Guatemala, which lost 17% of its forest between 1990 and 2005.

The southern forest region in Nigeria. See subfigure F on figure 1. As of 2005, Nigeria has the highest rate of deforestation in the world. Between 1990 and 2005, in total Nigeria lost 35.7% of its forest cover, or around 6145 000 hectares, and between 2000 and 2005 the country lost 55.7% of its primary forests, and the rate of forest change strongly increased up to 31.2% per annum.

The northern forest near Bo, the southern and eastern forests around Freetown (Sierra Leone). See subfigure F on figure 1. Forest loss in Sierra Leone is rapid, drastic and recent. Forest lands seem to have been preserved more along the western and southern edge of the country, but important development and deforestation are evident all along the coast.

The forest region around Abidjan (Côte d’Ivoire). See subfigure G on figure 1. From 2001 to 2018, Côte d’Ivoire lost 2,78 million hectares, which is equivalent to a deforestation rate of 19% since 2000 on average.

The Atlantic forest (Brazil). See subfigure H on figure 1. The Atlantic Forest was one of the largest rainforests of the Americas, originally covering around 150 million ha, with only 7% remaining, and inhabited by 130 millions of people. Causes of deforestation include agriculture, urban sprawl, cattle ranching, eucalyptus plantations. Forest loss is slowing down, thanks to important conservation initiatives.

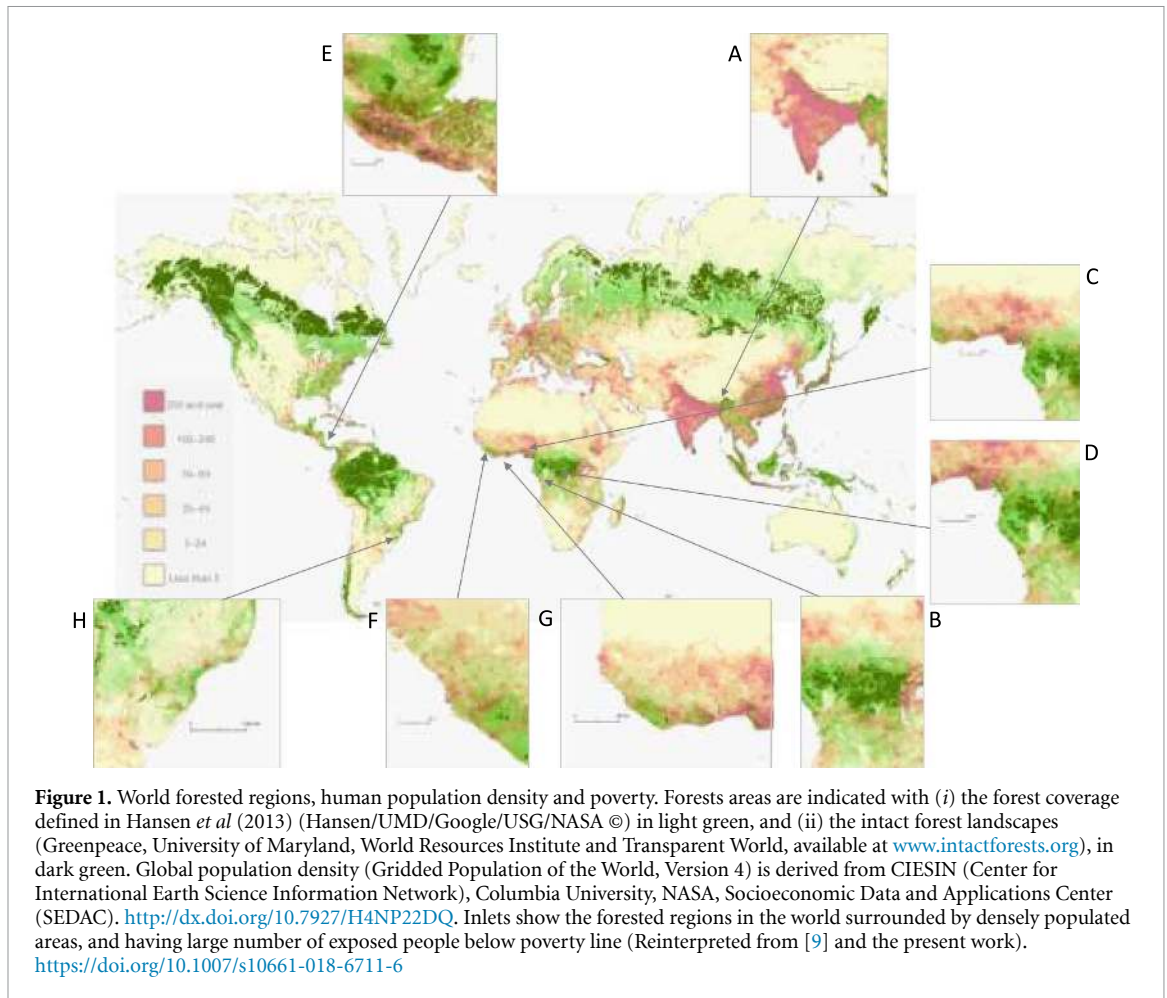
Adapted from [9] and the present work.

vector-borne emerging infections [10, 11]. In this context, previous research has strongly focused on the expected effects of climate change on spatial and temporal disease risk, and notably for infectious diseases transmitted by arthropod vectors [12]. Research to date has paid less attention to other factors that are already known to interact with emerging infections, such as land-use changes, massive deforestation of tropical rainforests and fragmentation of forest areas. In this review, we assess past and current knowledge on the relative effects of deforestation and forest alteration processes on emerging infectious diseases risk and examine how these have been incorporated into available analyses. We argue that the contact between wild animals, wild habitats, domestic animals and people in regions undergoing deforestation is a key driving process that induces the emergence of new infectious diseases. Although major focus has been given on the impact of climate change on emerging disease risks, we suggest, far more importantly that global climate changes, land-use transformation and

particularly deforestation in the most speciose areas of the planet, and locally at-risk nature resources harvest behaviors, is associated with increased risk of several major pathogens in human. We discuss how contemporary studies have focused on understanding unilateral aspects of forest-derived infections and propose adopting a system-based dynamics approach to understanding forest-borne disease risk [13], and recommend future directions for research to guide interventions and contribute to planetary health efforts.

2. Materials and methods. A systematic literature review

In April 2018, we searched the Web of Science Clarivate Analytics, InCites, JCR, ESI, EndNote, Publons, Medline and Google Scholar databases using a combination of search terms related to forests (undisturbed, pristine, perturbed forests), deforestation,



and emerging infectious diseases that were cross-checked to identify all original research articles published from January 1953 to mid-April 2018. Emerging infectious diseases are caused by newly identified species or strains of pathogens and whose incidence has increased in the past 40 years and could increase in the near future. Many emerging diseases in human are zoonotic (originated from an animal reservoir) or sapronotic (from soil, water or plant rhizosphere). The retained search terms were: tropical forest*; equatorial forest*; forest*; rainforest*; pristine forest*; undisturbed (non-disturbed) forest*; edge; edge effect*; deforestation; forest fragmentation; forest fragment*; deforested habitat*; deforested landscape*; (human) emerging infectious disease*; (human) emerging infection*; human health; zoonotic infection*; zoonotic infectious disease*; vector-borne disease*; and vector-borne infection*. We identified 565 article items (mainly scientific publications) in which one or several of these search terms were present in the article title, summary, or introduction section only. However, in most of these citations, the article core did not concern exactly the topic of this review, but mainly molecular biology and phylogenetic analyses. We excluded these articles from the final database.

We also excluded articles with redundant information based on the same empirical work. As our review focuses on emerging human infectious diseases, we also discarded articles on emerging infections in wildlife in relation to forests. For instance, we excluded studies that were identified with the search terms forest succession and edge effect because it concerned only wildlife health. By reviewing the abstracts, titles, keywords and by reading the articles, we eliminated many other publications due to their lack of direct relevance. Finally, we retained 165 articles on temperate and tropical/equatorial forests, their environmental changes, and emerging human infections. The initial and final lists of scientific articles are available upon request.

From our systematic literature review, it is observed a general increase in the number of scientific productions on forests and emerging infectious diseases from 1953 to mid-April 2018 (end of our literature review), with an important rise of this scientific works from 2000–2004 (figure 2(A)). Distinction between different categories of research works on this topic reveals that empirical studies also tend to increase during this period, whereas descriptive and systematic reviews, modeling studies and syntheses show a flat trend (figure 2(A)). The scientific

production was clearly dominated by studies from three main large areas, i.e. North America, South and Meso America, and Sub-Saharan Africa notably for its central and eastern regions, then followed by Europe (figure 2(B)). For North America and Europe, a large proportion of studies concerned Lyme disease, and for South and Meso America leishmaniasis, two disease systems that predominantly feed the topic. Classifying scientific papers based on the focus systems indicates that the number of studies asking unique and partitioning research questions, e.g. research done on insect vectors, or animal reservoirs or pathogens separately, clearly increase after 2006 (figure 2(C)). A similar trend is observed for publications addressing host (or vector)-pathogen interactions, which undoubtedly increases biological representation in those disease transmission studies (figure 2(C)). However, research studies integrating hosts and/or vectors, zoonotic pathogens and human cases do not show any particular trend, and such integrative studies on forest-derived diseases are still very rare. Indeed, research work on arthropod vectors only (without any analysis of pathogens) strongly increased after 2006 (figure 2(D)). Conversely, after 2006, although many studies still focused only on the hosts (e.g. mammals, bats), without any consideration for their status of infection, research on animal reservoirs/hosts started to take into account also the disease prevalence or incidence in animal reservoirs or in humans.

Overall, our systematic review clearly shows that the topic on forest-emerging infections is not strongly supported by many empirical evidences, and that trends though time in taxonomic and geographical research options may definitely bias the conclusions claimed by the different authors. Notably, we would have expected more general studies on forest-derived infections and the different mechanisms responsible for disease spillover and emergence rather than on identification of specific pathogens associated with forest habitats and associated hosts.

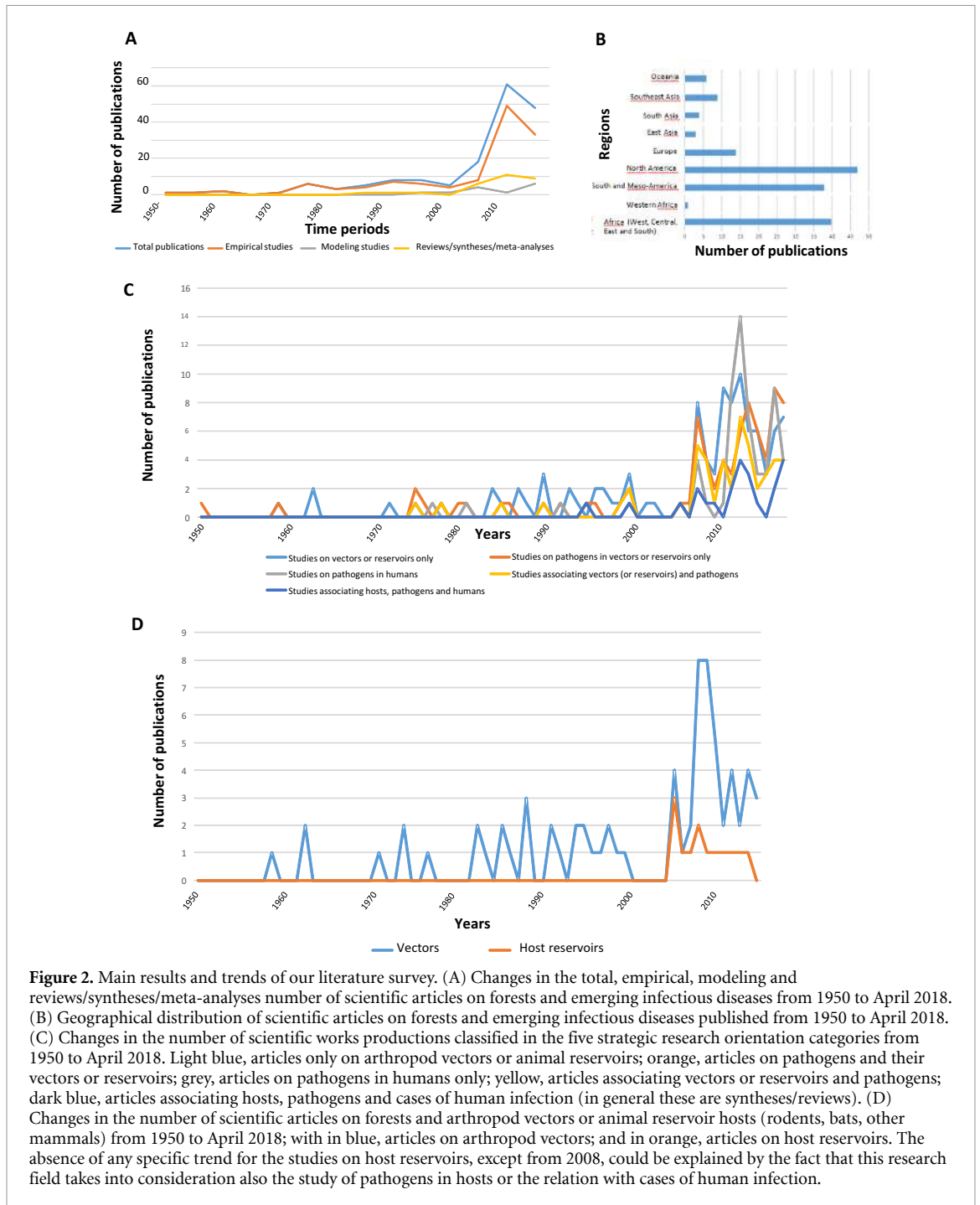
3. Forest-derived human infections macroecological patterns

Macroecology of infectious diseases identifies large-scale patterns of relationships between pathogens and hosts, including humans, and their vectors or reservoirs [14]. However, studies are still rare partly because of the difficulties of large-scale and long-term data collection [15], and because medicine and veterinary sciences often focused on small-scale local processes [but see 14, 16–19]. For instance, two studies highlighted the association of Ebola virus disease outbreaks in Central and West Africa with disturbance hotspots by deforestation as a plausible explanation for local species community changes—possibly including reservoirs of pathogens—thereby further

enhancing the risk of new infections in human communities close to the forest margins [20]. Definitely this pattern would not have been highlighted at smaller scale [21]. Forest loss and fragmentation caused by human activities is an important cause of biodiversity erosion worldwide, and this process is particularly pronounced in tropical regions, where much of Earth's biodiversity is located. One of the main effects of forest perturbation is the alteration of biological interactions, including the relationships between hosts and pathogens. On local scales, theory predicts a decrease in pathogen diversity due to the loss of hosts in such deforested contexts. However, forest loss and fragmentation could also increase pathogen infections from wildlife as a result of higher exposition due to human visits to these places for extraction of mineral resources, harvest of nature resources (game, ligneous and non-ligneous products), encroachment and implementation of new settlements [22]. Host-pathogen interactions are fundamentally important in forested ecosystems, but very few studies have identified and emphasized forest-derived pathogen large-scale biogeographical patterns. In this section, we identify the main macroecological patterns which have been identified in recent years concerning human, wildlife and pathogen interactions in scenarios of forest loss.

3.1. Sampling effort is important!

A general relationship may exist between the number of pathogen species and the spatial distribution of the sampling effort across biomes or regions as demonstrated in the present work for forest-derived infections in North America (figures 2(B) and 3(A)). One area could be considered as pathogen-rich just because of the high concentration of sampling events, e.g. primates and their parasites with particularly low sampling in Southeast Asia, Central and Western Africa, and South America, and better sampling in Eastern Africa and Brazil [23]. Therefore, large sample collection gaps (e.g. remote areas) and high concentration of sampling events (e.g. areas with facilitated 'highway-driven sampling') must be considered. The knowledge on pathogen biodiversity may be biased not only geographically, but also taxonomically because some host taxa or some infectious agents have been prioritized in emerging disease studies through time (e.g. bat species, tick-borne Lyme disease in Europe and North America, Leishmaniasis in South America), or were neglected in biomedical studies (e.g. fungal parasites). Some statistical tools, such as gap analysis, can be useful to reduce sampling bias between different regions and biomes [23]. The main message is that we know little about even the best studied primates and bats, and even less regarding the spatial and temporal distribution of pathogens within species. It is thus difficult to predict zoonotic pathogen emergence in humans without having more sampling for pathogens across

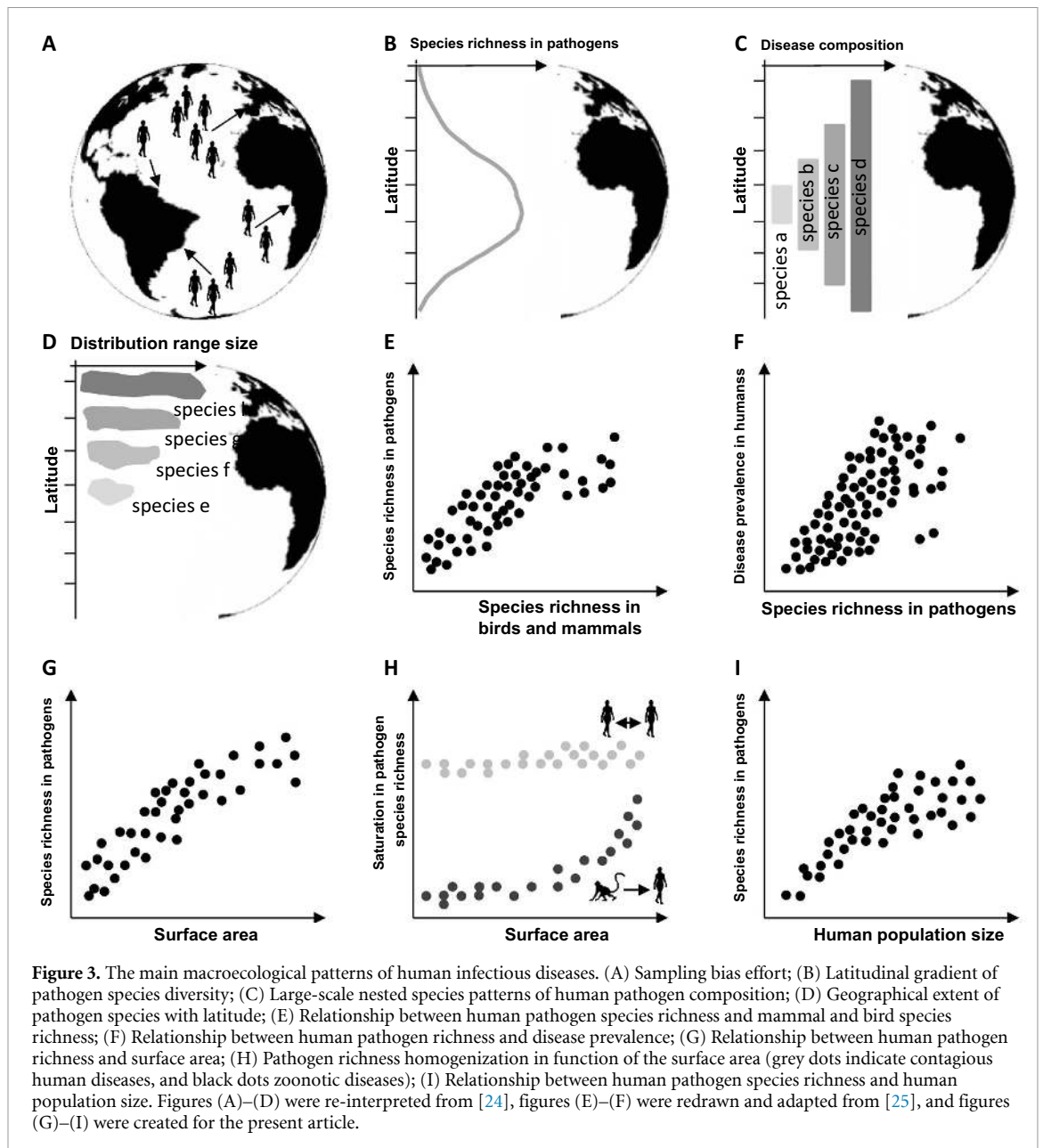


all animals, time, ecosystems and countries even if important advances have been achieved during the last decade.

3.2. Latitudinal gradient of pathogen species diversity

In general, human pathogen species richness peaks at equatorial latitudes and between the tropics, where large forest domains occur (figure 3(B)). Helminth parasite species across diverse host species also support a latitudinal diversity gradient [26]. Human pathogen diversity generally peaks in regions characterized by warm, wet and more seasonally

stable climatic conditions, and declines as climate becomes colder, with marked seasonal variations [17]. While the biogeography of human diseases corresponds well to this global biodiversity pattern, in terms of emergence a greater diversity of potential zoonotic diseases and their mammal species is concentrated in northern latitudes, especially rodent-borne infections in Europe [14]. The diversity of zoonotic mammal pathogens and their spatial distribution may erroneously be seen as synonymous with true human disease risk, rather than an estimate of underlying zoonotic potential mediated by many additional interactions. However, as far as we know,



forests, and notably in tropical domains, may well constitute the cradle for myriads of enzootic, zoonotic and sapronotic microbes through evolutionary processes that drive this phenomenon [27].

3.3. Large-scale nested species patterns of human pathogen composition

Nestedness of pathogen species assemblages occurs when the biotas at temperate latitudes with lower numbers of species tend to be subsets of biotas at richer sites located in the tropics (figure 3(C)). Specifically, many human parasite and microbial species are spatially restricted to the tropics. On the other hand, pathogen species present at higher latitudes are generally detected also in tropical regions [17]. Worldwide, the human pathogen nestedness could be

explained by extinction-dominated processes where pathogens, host reservoirs and vector species do not distribute in a consistent manner at higher latitudes, because of the absence of suitable climatic, environmental (rainforested) conditions, or absence of host reservoirs/vector species populations to sustain the disease life-cycle. On average, relationships between host and pathogen biodiversity are often unclear or lack consistent empirical support both across taxa and biomes. Thus, testing the hypothesis that tropical forests might constitute higher-risk regions for disease emergence and potential new epidemics in human than temperate forests, all other human and socio-economic parameters kept constant, remains a critical priority in infectious disease research, and international health prevention.

3.4. The extent of pathogen species geographic distribution increases at higher latitudes

One of the most well studied biogeographical patterns is the Rapoport's rule, according to which at higher latitudes taxa harbor larger geographic ranges. Human pathogens are spread unevenly across the globe, with foci in the tropics, and the Rapoport's rule could explain the geographical distribution of many of them [28] (figure 3(D)). Seasonal climate changes at higher latitudes favor species with greater tolerance for environmental variations, and consequently larger distribution ranges [28]. Different socio-economic conditions (large population size, transportation, medical care) may have also allowed the expansion of some pathogen groups in regions with variable climates over time, particularly person-to-person infectious diseases (figure 3(D)). Also, the greater diversity of biogeographic units and current habitat fragmentation at lower latitudes create spatial heterogeneities and habitat edges that could support an increased isolation of host reservoirs and vectors, favoring the development of many pathogens through coevolutionary processes [29].

3.5. Relationship between human pathogen richness and mammal and bird richness

After controlling for health spending, disease control and human population size, mammal and bird species richness explains most (72%) of the human pathogen richness variation across the globe [25] (figure 3(E)). Humans living in regions with high biodiversity of bird and mammal species (i.e. inter-tropical forests and closed ecosystems) are consequently exposed to a larger diversity of pathogens than people living in areas of low diversity. Although it is not known whether bird and mammal richness causes human pathogen diversity, or rather indicates a higher level of pathogens in the environment, this richness does not guarantee the existence of a pathogen species pool capable of jumping to humans [30]. Since most biodiversity hotspots are tropical forests, they also form a rich pool of microorganism diversity, a small fraction of them being potentially pathogens for human with an elevated risk of spillover through novel exposures.

3.6. Relationship between human pathogen richness and disease prevalence

Disease prevalence in humans is also strongly correlated with pathogen species richness in mammals and birds (figure 3(F)) with additional effects of climate and human population size [25]. Massive health programs to control different infectious diseases, particularly in tropical areas, have considerably reduced pathogen prevalence, but not pathogen richness *per se* [25]. For instance, malaria control programs in Africa have reduced malaria prevalence, but the pathogen is still present. As local outbreaks of a neglected or newly developed infectious disease can

rapidly progress to more severe stages, reducing the global pathogen prevalence will require the implementation of diagonal public health actions to target not only the more classically recognized infectious diseases (i.e. HIV/AIDS, malaria and tuberculosis) but also such co-occurring often neglected diseases (e.g. schistosomiasis, soil nematodes, Buruli ulcer), because they also contribute to the global disease burden of a country [25].

3.7. Relationship between human pathogen richness and surface area

According to the species-area relationship, the size of a site will limit the number of species and of vector or host reservoir that can be harbored (figure 3(G)). The habitat heterogeneity (natural mosaic or heterogeneity resulting from pressures, e.g. rainforest clearing, forest edges) and food resource diversity (for host reservoirs or vectors) increase with the size of the area, thus offering a larger number of available niches and favoring the coexistence of a larger number of potential reservoir or vector species. Alternatively, the positive species-area relationship could be the result of a pattern generated by random sampling process [31]. Indeed, the number of newly discovered pathogen species in forests could simply depend on the size of the prospected sites.

3.8. Pathogen richness homogenization

Given the frequency and extent of human movements, homogenization also applies to human pathogens. Human pathogens are distributed around the globe, and the species distribution magnitude varies greatly in function of their host requirement, transmission mode, and taxonomy. However, when plotting the species composition similarity of the different human pathogen categories against the surface area (from local to global scale), human-specific pathogen assemblages are extremely homogenous across regions and continents due to their fast dispersal (figure 3(H)). By contrast, pathogens requiring non-human hosts to complete their life cycle, such as tropical rainforest zoonotic agents, are more localized [32] (figure 3(H)), as the distribution of host species is determined by biogeography and climatic constraints. At the local level, the maintenance of human zoonotic pathogen diversity also depends on having host reservoir species-rich habitats such as for rainforests [32]. As a member of the Coronavirus family, the current outbreak of novel coronavirus disease 2019 (COVID-19) is a zoonotic disease, that jumped from some animals to humans, maybe traveling through other species on its way to infecting people, somewhere in the Wuhan province, and has spread rapidly within China and across many countries [33]. To some extent, COVID-19 has passed through a wildlife market in China to a global outbreak as a real-time demonstration of this global and

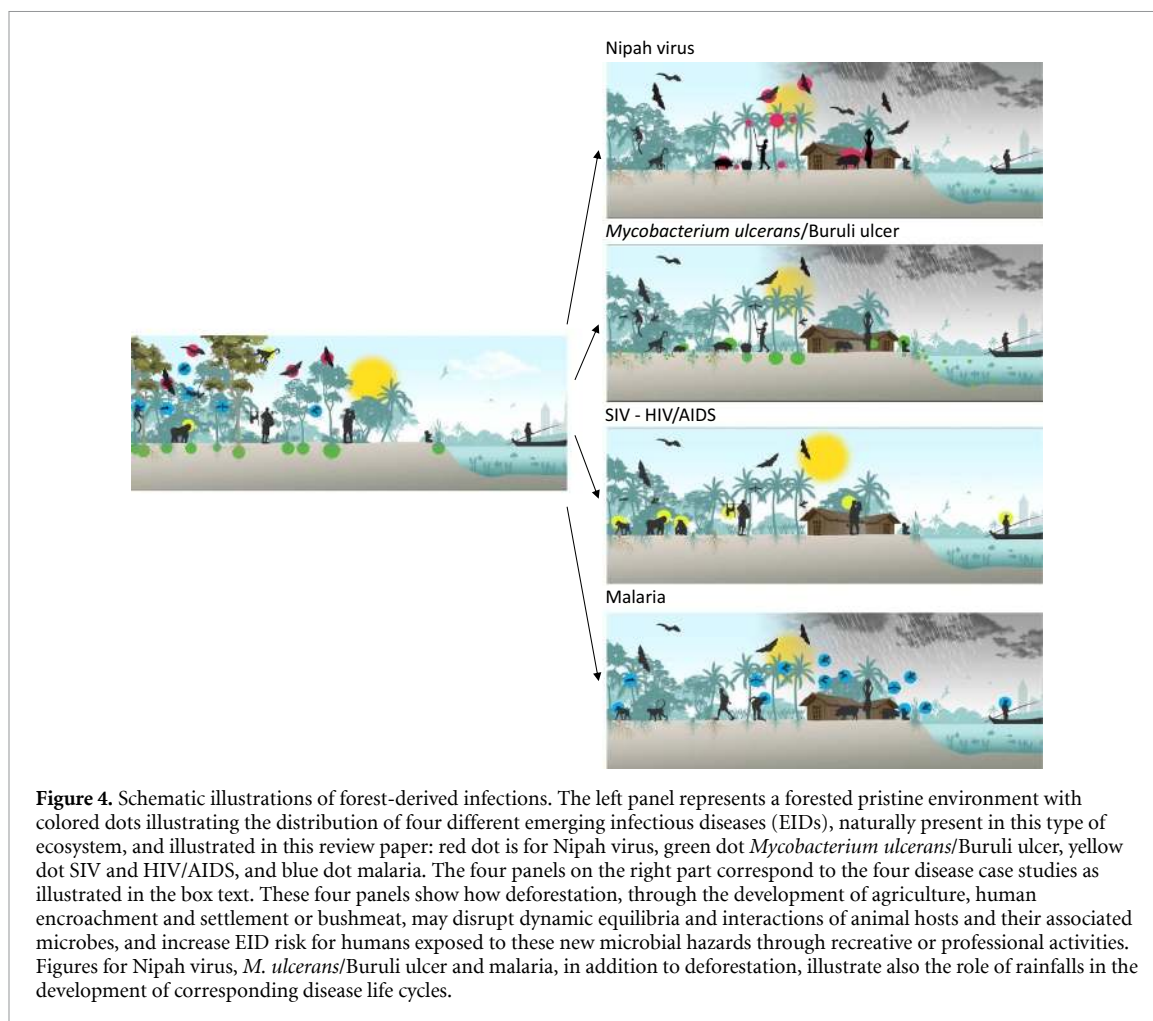


Figure 4. Schematic illustrations of forest-derived infections. The left panel represents a forested pristine environment with colored dots illustrating the distribution of four different emerging infectious diseases (EIDs), naturally present in this type of ecosystem, and illustrated in this review paper: red dot is for Nipah virus, green dot *Mycobacterium ulcerans*/Buruli ulcer, yellow dot SIV and HIV/AIDS, and blue dot malaria. The four panels on the right part correspond to the four disease case studies as illustrated in the box text. These four panels show how deforestation, through the development of agriculture, human encroachment and settlement or bushmeat, may disrupt dynamic equilibria and interactions of animal hosts and their associated microbes, and increase EID risk for humans exposed to these new microbial hazards through recreative or professional activities. Figures for Nipah virus, *M. ulcerans*/Buruli ulcer and malaria, in addition to deforestation, illustrate also the role of rainfalls in the development of corresponding disease life cycles.

rapid homogenization/uncontrolled spread of disease [34].

3.9. Relationship between human pathogen richness and human population size

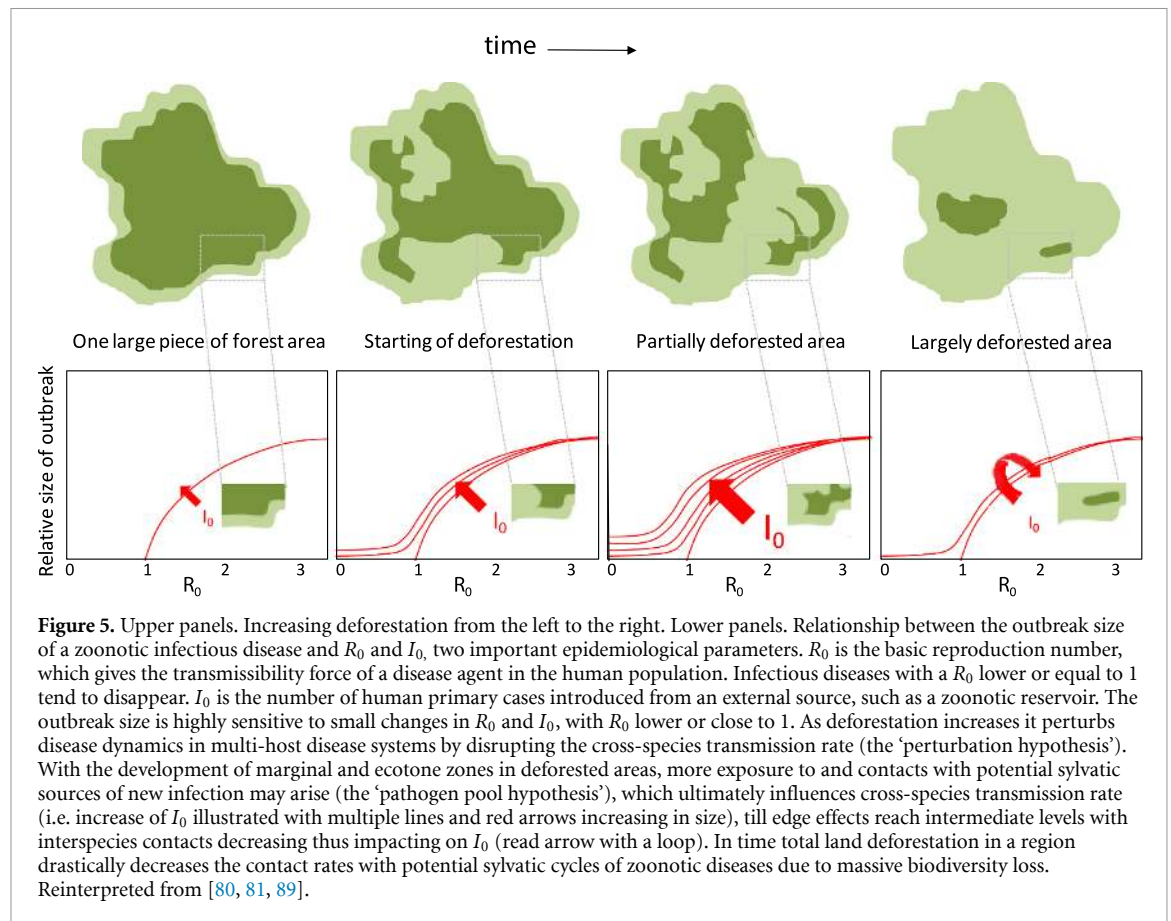
Generally, larger host populations can maintain diseases more easily due to the presence of a larger pool of susceptible individuals (i.e. without acquired immunity) and the higher number of new susceptible individuals entering the population by birth or immigration. In high-density populations, contacts between individuals should also be more frequent, thus favoring the spread of a contagious pathogen or a vector-borne infection [35]. Large islands support larger human populations with correspondingly higher pathogen species diversity, than smaller ones, supporting the island equilibrium theory [36]. Similarly, the diversity of human pathogens in a region increases with the human population size and the bird and mammal species richness [25]. In general, when all other potential confounding parameters are kept constant, pathogen diversity should be higher in larger human populations (figure 3(I)). In tropical rainforests, indigenous tribes and ethnic groups generally lived in small groups, thus reducing the opportunity for the circulation of a wide range of human

and zoonotic pathogens. However, as disturbance by deforestation will destroy the core habitats of host species and will modify uses of the forests by humans, possibly reservoirs of some zoonotic pathogens will thrive enhancing the risk of transmission in human communities close to the forest margins thereby enhancing the risk of new epidemics and pandemics.

As the space separating the local and global scales becomes thinner, every human pathogen could spread everywhere, reducing the possibility of finding macro-scale biogeographical signatures of human microbes. However, recent advances in human infectious disease macroecology strongly support several empirical findings in geographical ecology, and show that they still explain human pathogens distributions on a global level.

4. Microbial hazards, forest clearing and conversion and risk of novel infectious disease emergence

Forests have always been seen as places of threatening diseases and microbial hazards. For instance, the Death's servants *biloko* are dwarf-like creatures and haunt the densest and darkest part of the rainforest in the Democratic Republic of Congo, bringing



mysterious diseases [2]. Amazonian forests are populated by spirits, images of the fathers of living animals, and by other creatures, such as the ‘Devil spirit from the penis’ of the Trio Carib group (Guianas) that causes the *epah sesereimë*, likely a cervical cancer [3] and the forest evil spirits (*wiripë*) mediating diseases [4].

Native tribes also acquired traditional and extensive knowledge from living in the rainforest, relying on long-term empirical observations, oral tradition and wisdom of the ancients. In Amazonian, the Trio Amerindian tribe observed that hunting trips in swamp forests are associated with a higher risk of cutaneous Leishmaniasis, attributed to insect bites [3, 4]. The Wayãpi tribe are aware that seasonal heavy rains are favorable to mosquito reproduction, and consider mosquitoes as the bearers of illness, associating the rainy months with the upsurge of malaria (see Box) [37]. In Sub-Saharan Africa, bushmeat hunting and butchering are considered to be primary risk factors for disease transmission [38, 39]. In Cameroon, older people explain that Buruli disease (a skin ulceration caused by an environmentally persistent bacillus; see Box) may appear at the time of important bush fires for deforestation [40]. All these examples illustrate the long-term entanglement of human-forest interactions and traditional culture [41].

The current Anthropocene era is characterized by the overall disappearance of native tribes, their knowledges and wisdom, disappearance of natural landscapes, pollutions of ecosystems, global warming, and habitat changes. The ongoing dramatic global changes of climatic cycles have observed and predicted consequences on disease dynamics [42], linked to the overall geographic expansion of vectors [43–46]. Despite some encouraging trends, tropical forest loss and forest degradation, which remain locally the most spectacular effects [47–49], persist or are still increasing, particularly in lower income countries [50] or as consequence of profound political changes [51]. Beside deforestation, the more visible impact of anthropogenic disturbances on natural habitats is undoubtedly the loss of biodiversity [52]. Species disappearance has consequences on the structure of inter-specific biotic networks and functioning ecosystems [53, 54], and on human well-being, notably through the degradation of ecosystem goods and services [53, 55–57].

Locally, even the early stages of environmental disturbance affect the organization of species communities and the biotic interactions. Modifications in the distribution of natural resources, simplification of ecological niches, and microclimatic variations could favor the expansion of more generalist and adaptive species, and the reduction or disappearance of more

specialized, less resilient species. Although the mechanisms driving diversity at the metacommunity scale are not identified, the distribution of host species in edges between core forests and matrix landscapes may facilitate or impede inter-species contacts and the distribution of pathogens [58–60] (figure 4). It also modifies the trophic chains, and consequently affects all the disease transmission systems [20, 61].

Loss of biodiversity can exacerbate the risk of pathogen spillover [62]. In low diversity communities, vectors reach higher pathogen prevalence because they feed more frequently on primary reservoirs. On the opposite, vectors in high biodiversity communities feed on a wider range of hosts, some of which are poor pathogen reservoirs, resulting in lower pathogen prevalence at ecological community level [62–64]. The dilution effect proposes different mechanisms to link high species richness and reduced infection disease risk: lower frequency of encounters between competent hosts and pathogens, increased host recovery from infection, higher mortality of infected hosts, and decreased density of susceptible hosts [65]. Despite supportive evidence at small spatial scales, the dilution effect has been strongly debated [66–68], and on broad spatial scales higher biodiversity may on the opposite amplify disease risk [67].

Although the dilution effect is still an attractive model, it presents important limits and interpretations subject to semantic and *a priori* choices that may influence the conclusions: how ‘biodiversity’ was measured, how ‘increased infection’ and ‘increased transmission’ were assessed, how the ‘increased risk’ was evaluated [69]? This concept relies on the supposed evidence that anthropogenic disturbances reduce biodiversity, but this may not be true [70]. Moreover, in disturbed habitats, dominated by more efficient species, the decrease of low-competence species is definitively not a general pattern, and depends on the species life-history traits and their resilience [71]. In communities with the highest species richness, the likelihood of the occurrence of some highly efficient species could also increase, with a positive effect on parasite transmission, a phenomenon called the ‘identity effect’ [72]. The geographical scale is another important issue [67]. The dilution effect operates at the local scale [73], while at the global scale, higher avian and mammal diversity is associated with higher pathogen richness and prevalence in humans. In addition, communities can act differently on competing drivers of transmission (i.e. host density, contact rates, transmissibility), causing concurrent transmission increases and decreases in the same host-pathogen system [74]. A striking example of the limit of the dilution effect is malaria: deforestation is associated with increased malaria incidence in South America [75, 76], whereas the opposite trend is observed in Southeast Asia where the main vector is dependent on forest habitats.

Feedbacks between deforestation and emerging diseases may also exist, with deforestation significantly increasing malaria incidence while high malaria burden simultaneously reduces forest clearing [77]. Contradictory effects of deforestation on emerging infections found in the literature illustrate the intimate ties that exist between these environmental changes and human health, stimulating more case-by-case analyses that are required to precisely predict the impacts of deforestation on derived infections [78, 79].

Besides these theoretical considerations, the edge effects and ecosystem fragmentation are more immediate and likely understudied links between habitat disturbances and disease risk [60, 80–84]. Together with more frequent contacts between humans and hosts and/or vectors and bioclimatic condition modifications [85], increased contact areas also may lead to a rise in the number of index cases and promote the introduction of a pathogenic agent in the surrounding human populations. Particularly in the case of infections with low transmissibility between humans, more primary cases and increased contact frequency with a suspected reservoir or intermediate host will have important consequences on the resulting outbreak strength, as observed for Nipah virus [86, 87] and EBOV [20, 88] (figure 5).

The current levels of forest anthropization can cause changes in the ecology of host reservoirs, vectors or pathogens, promoting the proliferation of zoonotic and sapronotic diseases [59]. Synanthropic species are wild species that prosper in areas where humans are present, both in rural and urban conditions. Many rodents, opossums and bats inhabit or even benefit from ecotone zones that contain mixed forested, pasture and human-dwelling areas. These species can be important components of the transmission ecology of pathogenic microbes [90]. Additionally, synanthropic hosts can be associated with blood sucking arthropods (e.g. mosquitoes, ticks) close to anthropogenic areas and that can transmit pathogens to humans. For instance in Panama, *Borrelia* bacteria that cause relapsing fever are associated with a wide diversity of mammal hosts, including opossums, monkeys, armadillos, horses and calves, in human-occupied settings [90].

Furthermore, forest fragmentation and the associated edges could favor the introduction of human strains in wild hosts. In French Guiana, toxoplasmosis is caused by Amazonian *Toxoplasma gondii* strains associated with forest cycles circulating in wild felids and showing a much higher genetic diversity than strains from urban and anthropized habitats and hosted by domestic cats [91, 92]. Microsatellite DNA analysis shows genetic structure differences between urban and forest strains, introgression of urban strains into forest strains in forested edge areas, and strain hybridization [93]. These genetic re-assortments could have consequences for

human health, and unknown effects on wildlife [93]. Similarly, in peri-urban satellite forests close to Cayenne, rodents, marsupials and bats are infected by the Dengue virus serotype 2 that causes outbreaks in humans. This suggests a possible role of wild mammals in dengue virus maintenance between epidemics [94]. However, such splits of strains from urban to forest cycle is far less studied than the peridomestication of disease cycles favoring introgression of wild strains into more anthropized environments.

Another key question concerns the cascading relationships between habitat disturbance and pathogen genetic diversity, and between pathogen diversity and the likelihood of spillover and disease outbreak [60, 83, 84]. Furthermore, the links between microbial strain diversity and the likelihood of pathogen spillover are unclear. These relationships are under the influence of a set of drivers, including some unidentified ones, and they are never simply linear. For instance, in malaria, outbreaks are mediated through selection imposed by the host immunity [95] and drug resistance [96]. Therefore, it is currently unrealistic to claim a general trend between habitat change, microbial intra- and inter-species diversity, and likelihood of disease emergence.

5. Concluding remarks and recommendations

For centuries, forests have been the cradle of humankind by providing food, medications, places for ritual practices, mysticism, etc. We showed that forests are also the source of many threats to human health. We first highlighted some important diseases, geographical and taxonomic trends through time in the published studies on the links between forests and human emerging infections, particularly the focus on some regions (e.g. North America), on specific pathogens and the disease they cause (e.g. Lyme, Leishmaniasis) and on some particular host taxonomic groups (e.g. bats, rodents). Specifically, in many articles, the links between forest and emerging diseases were not at all or poorly discussed, or the disease life-cycle system was analyzed partly, particularly in the case of vector-borne infections where only the arthropod compartment was analyzed. We then delineated current main macroecological patterns for human emerging infections. As shown with examples based on our own personal field and research experience, the pathogen spillovers and outbreaks are related to: (i) land use change, notably through deforestation for timber production, agriculture development and land transformation for infrastructure needs; and (ii) increase of human populations living in or beneath core forests who have contributed to the modification of the natural, sylvatic equilibrium between microbial forms, their reservoirs/hosts and vectors, and the human

intruders. Through the example of Buruli ulcer, we stressed the important role of deforestation and land conversion in the alteration of animal communities and trophic networks at habitat interfaces, thus affecting interspecies—including human—contacts and landscape spillovers. Ecological dynamics at edge habitats is the most important mechanism influencing pathogen transmission as demonstrated for *Plasmodium* species that transmit from primate reservoirs into humans and back, via mosquito vectors in South American deforested landscapes (see Box). Moreover, the interface between land-use changes for agriculture development, husbandry practices and varieties of other human activities facilitates spillovers and disease outbreaks, as shown for Nipah virus in Cambodia (see Box). Bushmeat consumption and hunting contact rates are another important cause of pathogen emergence within human populations as highlighted with the example of HIV/SIV (see Box).

In the present review, we limited our analysis to forest human-based emerging infections only, and we did not consider wildlife-based diseases [97, 98] because drivers of spillovers and outbreaks are likely to be quite different between these two categories. We strongly recommend that a similar study can be done pinpointing on this aspect and discussing on comparison between wildlife and human diseases for a better understanding of wildlife-human-forest alteration interfaces.

Finally, we need to think about the next step to prevent, or at least limit the emergence and spread of threats to human populations from forests (see table 2). Many authors recommend the ‘OneHealth’ approach. However, we need to rely on a true, integrative and transdisciplinary OneHealth approach [99]. In this approach, health management requires the participation of traditional health actors, such as physicians and veterinarians, and of other actors from ecology, environmental sciences, social science education and citizen participatory science. Yet, a multidisciplinary team may not be sufficient to put in place a true OneHealth/EcoHealth approach and to tackle the complexity of emerging disease risks. Integrating the work of these different disciplines is a real challenge, and companion modeling tools might help constructing models and strategies for health management between disciplines and actors, involving local communities, local and national authorities, and non-governmental organizations [100]. Control and prevention measures can be useless if not understood and adopted by concerned populations, including low-cost solutions such as bamboo skirts used to protect palm sap from Nipah virus contamination by bats [101]. The involvement of local populations in the construction of socio-ecological system models can help truly implement these OneHealth and EcoHealth approaches and may better address to solve these new important global threats. Furthermore, the

Table 2. Main recommendations and future research orientations on forest-derived infections^a.

| Recommendation. | Possible avenues to address recommendation. |
|--|--|
| Anticipate and organize plans and actions. | <ul style="list-style-type: none"> • Develop models to explain and predict the role of deforestation and other habitat changes on the risk of emerging threats and the geography of pathogen spread. • Develop prevention and control strategies, tools and capacities for infectious hazards interlinked with forest loss. • Establish and maintain experts' networks to detect, monitor, and understand emerging infectious disease threats, notably in regions with increased urbanization, uncontrolled population growth or migration to urban and peri-urban areas, and poor-vector and reservoir-control intervention and surveillance capacities. • Develop integrated surveillance systems, including satellite imagery systems, for early detection of habitat changes. |
| Research and knowledge development. | <ul style="list-style-type: none"> • Characterize knowledge gaps on the risk factors for transmission of emerging infections at the forest–animal–human interfaces. • Study the pathogen <i>per se</i> through real-time genomic surveillance, check for molecular, proteome, metagenome and epigenetic changes that may predispose to spread or virulence and host spillover, and develop comparative genomics. • Understanding the dynamics of preceding animal outbreaks (enzoonosis), detecting and monitoring infectious agents in animal populations and the environment (soil, water...) can help informing any subsequent human outbreaks and spillovers. • Identify the presence of animal reservoirs and vectors and their competences for carrying specific pathogens (at least prevalence level), and analyze their dynamics in time and space, and at the community level. • Understand the catalysts of disease transmission and identify early the appearance of new syndromes or clusters of symptoms in communities. |
| Education and training. | <ul style="list-style-type: none"> • Implement more creative and inclusive surveillance approaches (e.g. vegetation density and composition, rainfall, plant and animal phenology, animal and human movements, vector presence and dynamics). • Improve forest-derived modeling studies and data analyses and training in interpretation of major emerging threats. • Training in interpretation of major emerging threats due to land-use changes, and notably deforestation and forest fragmentation (including for instance early signs of animal population changes (e.g. migrations, outbreaks, deaths or disappearance) need to be available to educate the workforce in all sectors. • Understand human behavior and traditional practices as key drivers of emergence and transmission of emerging threats, and promote practices at low/least risks. • Health education should be promoted, locally/ethnically adapted to the different idioms, and cultures. |

Table 2. (Continue).

| Recommendation. | Possible avenues to address recommendation. |
|---|--|
| Transdisciplinary and synergies to reinforce work forces. | <ul style="list-style-type: none"> • Anticipate future needs for EIDs prevention and mitigation by horizon and environmental scanning of major forested blocks and their evolution timeframe on the long term. • Focus on areas undergoing rapid socio-ecological changes such as deforestation and forest fragmentation, large human population size, high density, precarious ways of life, and important resource demand. • Analyze syndemic clustering between endemic, e.g. neglected tropical diseases, and EIDs, exploring how anthropological behaviors, social attitudes, political, economic and ecological factors, could create syndemics, especially in poor and undersurvelled populations. • Develop national and international short- and medium-term research agendas and increased cross-disciplinary collaboration and promotion of inter-sectoral collaboration (economists, social and environmental scientists, practitioners, veterinarians, forestry experts, livestock farmers and other animal keepers, decision analysts, and experts in politics and logistics) and knowledge synthesis. • Continue integrated, systems-based research, including animal and ecological health, going beyond the One Health approach. |

^aThe list of recommendations and orientations has been synthesized from the recent literature and collective knowledge from the authors. The following references have been particularly helpful [103–113].



Figure I. Local pig farm showing bat faeces on the canvas roof, Cambodia. Copyright, J Cappelletti.

Sub-box 1

Deforestation and host foraging behavior: Nipah virus

Deforestation can impact disease emergence through the modification of the foraging behavior of reservoir hosts. Following the loss of habitats, some wildlife species may change their feeding behavior and use human productions as food resources, increasing potential contacts at the human-domestic-wildlife interface, as exemplified by the emergence of the Nipah virus (NiV) (see figure 4).

Fruit bats of the *Pteropus* genus has been identified as the reservoir of the NiV, and its emergence occurred in Malaysia in 1999 [114, 115] and recurrent outbreaks in Bangladesh and India.

Several studies have suggested a link between forest land-use changes, agricultural practices and NiV emergence. In Malaysia, the NiV emergence zone was characterized by a significant intensification of agricultural production, particularly pigs and mangoes [116, 117] (see figure 1). The hypothesis that NiV was transmitted through fruits contaminated by *Pteropus* bats at the index farm was supported by its isolation from fruits partially eaten by pigs [115].

In Bangladesh, villages in the epidemic region have higher human population densities and are located in more fragmented forest areas and agricultural fields [118]. The greater diversity of food sources in these cultivated and mosaic landscapes might provide *Pteropus* bats with permanent resources, promoting their sustainable establishment and increasing the risk of contacts with humans.

In the Philippines, a NiV outbreak affected 17 people and 10 horses in 2014 in a deforested area [119]. While no human cases have been detected in Cambodia and Thailand to date, NiV has been isolated in *Pteropus* bats from these two countries [120, 121] where GPS studies confirmed a foraging preference towards cultivated areas [122, 123].

Sub-box 2

Deforestation and foodweb structure: Buruli ulcer

Deforestation can impact disease emergence through the modification of trophic chains and the promotion or decline of specific functional groups of species in ecological communities. Natural habitat modifications or land-use changes could shift trophic interactions that can represent a direct (e.g. predation) or indirect (e.g. extinction cascade) modification of trophic networks.

M. ulcerans is a slow-growing bacillus that causes a rare, neglected tropical disease in humans, called Buruli ulcer. It is present in many flood plains and wetland areas in tropical Africa, Central and South America and South-east Asia (see figure II). Recent findings suggest that its aquatic persistence and dynamics are the result of complex interplays between environmental, abiotic factors, and biotic interactions [124]. In Cameroon and French Guiana, in two medium-term research surveys of aquatic ecosystems, it was demonstrated that this bacillus shows seasonal fluctuations in the environment [61, 125]; it persists in water bodies with higher *pH* and within low-abundance species communities [126]. These two surveys also highlighted the important role of deforestation and land-use changes in the emergence of this skin infection in humans (see figure 4). The highest bacilli concentrations were found in host functional groups lower in the food chain, indicating a diet high in algae, detritus, diatoms, bacteria and similar food



Figure II. River side and swampy areas near the Nyong river, highly favorable for the persistence of *M. ulcerans* causing Buruli ulcer, central Cameroon. Copyright, J-F Guégan.

resources. The size of the local aquatic taxa trophic niche was reduced in sites located in or near deforested areas [61]. This resulted in the concomitant decrease in the number of potential predator species per prey taxon and of potential prey species per predator taxon. The non-linear increase in the potential abundance of *M. ulcerans* in conditions of anthropogenic stress may be attributed to an increase of the preferred hosts, represented predominantly by low-trophic level organisms that accumulate bacilli from the aquatic environment through their diet.

At sites perturbed by human activities, some basal aquatic host species may flourish because of a decrease or disappearance of their natural predators, and could incidentally recover and concentrate the naturally-persistent microbes. Therefore, deforestation, rather than agriculture development and human settlement, appears to play a determinant role in the freshwater food web collapse and on its consequence on *M. ulcerans* load in the more perturbed sites.

Sub-box 3

Forest logging and mining in West Central Africa and the emergence of HIV/AIDS

Viruses causing AIDS in humans originated from those infecting chimpanzees and gorillas in Central Africa and those infecting sooty mangabeys in West Africa [127]. Molecular clock analyses have set the date of cross-species transmissions from chimpanzees to humans around 1920 for pandemic HIV-1 and around 1940 for the epidemic groups of HIV-2 [128, 129].

Factors that favoured the emergence of HIV/AIDS epidemic/pandemic included: (i) an increase in exposure risk, (ii) an increase in the probability of human-to-human transmission of the virus and (iii), an increase of the probability of virus adaptation to the human population [130]. Increase of exposure risk is demonstrated by the increase of bushmeat hunting and butchering (see figure 4), and of forest logging and mining in West Central Africa, since the early years of the 20th century (see figure III). For example, a detailed analysis of the evolution of forest logging in the Congo Basin between 1959 and 2004 showed a dramatic increase in Cameroon, Gabon and the former Zaire, now DRC [131]. Commercial logging with as corollary the settlement of temporary villages, the presence of commercial sex workers, and wood transportation by truck drivers, facilitated the spread of a sexually transmitted infection such as HIV [132]. A recent and additional dramatic consequence of human mobility between rural and urban areas of West Central Africa is, beside the emergence of the virus from the rainforest into the human population in urban centres, the backwards spread of the HIV virus from cities into villages as exemplified by the observed high HIV prevalence and genetic diversity in logging areas and in villages located along truck tracks [133, 134].

Today, the World Health Organization (WHO) estimates that 37 million people worldwide are carrying the AIDS virus, and, since the beginning of the epidemics in the early 1980's, HIV/AIDS has killed approximately 35 million persons around the world. Factors that contributed to emergence of HIV/AIDS pandemics are still present in Africa. Forest fragmentation and deforestation all over tropical Africa altogether with agricultural field developments, human behavior and practices and population demography constitute important settings for animal-human contacts and elevated zoonotic disease transmission risks.



Figure III. Deforestation for timber production in tropical areas may contribute to increase human-wildlife contacts, and facilitate the spread of new emerging viruses, e.g. HIV/AIDS, towards large cities. Copyright, B de Thoisy.

Sub-box 4

Deforestation, loss of forest cover and Malaria in Central and Southern America

Malaria is a potentially fatal human infectious disease caused by several species of protozoan parasites (genus *Plasmodium*) and is among the most debilitating infectious diseases worldwide. The parasite host range is large, from birds to humans. *Plasmodium* species infecting humans originate from primates and are transmitted to humans through the bite of infected mosquitoes of the genus *Anopheles*.

In Central America and Amazonia, early observations revealed high prevalence of malaria infection in many species of monkeys, particularly in the *Cebidae* family [135]. Those works also evidenced strong *Plasmodium* seroprevalences among several Amerindian ethnic groups, living strictly in the rainforest, without contact with urban or peri-urban forms of malaria, suggesting a zoonotic component of malaria [136]. Studies on monkeys in French Guiana confirmed the existence of a malaria sylvatic cycle [137, 138]. It was first shown that *Plasmodium brasilianum*, the parasitic agent classically considered to be restricted to monkeys, and *P. malariae*, one of the four human malaria agents, were a single species [139]. More recent investigations on the links between forest habitat disturbances and malaria showed a clear effect of deforestation on increased incidence of the disease [76, 139, 140]. But rather than deforestation *sensu stricto*, many explanatory factors associated with deforestation are mentioned: opening of forest tracks, logging methods, increased fires frequencies at forest edges [115], resulting in habitat modification of mosquito species communities, with creation of new breeding sites and development of favourable conditions for the expansion of efficient vectors for parasite transmission [78, 141] (see figure 4).

These elements are changing today's vision of the *Plasmodium* malaria cycle [142], questioning the role of habitat and ecosystem changes on the transmission of this parasite (see figure IV). In areas where the presence of human would be temporary (e.g. logging areas, gold mining camps, extractive activities), the introduction of human parasite to peripheral monkey species populations could allow its establishment and maintenance at edges, even when anthropogenic activities have locally stopped. These parasites could be at the origin of the re-emergence to cause human cases through contacts.



Figure IV. Human encroachment and settlement in forest satellites, here in suburbs of Cayenne, French Guiana, put into contact wildlife and human communities, and also favor the development of mosquito populations transmitting malaria. Copyright, J-F Guégan.

implication of local population may help improve the sustainability of surveillance programs, including for instance detectable and early signs of animal populations changes (e.g. migrations, outbreaks, deaths or disappearance). Indeed, long-term monitoring and communication systems are costly to implement and maintain but are essential to the prevention of emerging threats to health. Current research inadequately addresses the complexity, context specificity and social dimensions of forest-derived infections. Definitely, it requires long-term surveillance, cross-scale evaluation of the interactions between these changing forest landscapes and surveillance of wildlife communities, vectors and pathogens in order to underpin effective management of disease risk at the forest-human interface [60, 102]. To advance research on this critical topic, we suggest that future studies pay careful attention to the precise local paths of the disease local and global outbreaks. With continuing land conversion and fragmentation of world forests, notably in the tropics, it will drive dynamic changes in disease transmission, requiring a better understanding of the factors that condition the microbial diversity within these habitats and of the coevolutionary host-parasite processes ranging from host individuals to entire continents and long-term lifespans (see table 2).

We argue that our ability to understand the forest-pathogens relationships in detail and manage these threats depends on long-term monitoring programs, and regional and global coordinated and practical disease management taking into account both land-use changes, social issues, and biological conservation.

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Data availability statement

The data that support the findings in the section on the bibliographic study of our article are available from the corresponding author upon reasonable request.

ORCID iDs

Jean-François Guégan  <https://orcid.org/0000-0002-7218-107X>

Ahidjo Ayouba  <https://orcid.org/0000-0002-5081-1632>
 Julien Cappelle  <https://orcid.org/0000-0001-7668-1971>
 Benoît de Thoisy  <https://orcid.org/0000-0002-8420-5112>

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