Towards a 'people and nature' paradigm for biodiversity and infectious disease

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Abstract

Zoonotic and vector-borne infectious diseases are among the most direct human health consequences of biodiversity change. The COVID-19 pandemic increased global health policymakers' attention on the links between ecological degradation and disease, and sparked discussions around nature-based interventions to mitigate zoonotic emergence and epidemics. Yet though disease ecology provides an increasingly granular knowledge of wildlife disease in changing ecosystems, we still have a poor understanding of the net consequences for human disease. Here, we argue that a renewed focus on wildlife-borne diseases as complex socio-ecological systems – a *"people and nature"* paradigm – is needed to identify local interventions and transformative system-wide changes that could reduce human disease burden. We discuss longstanding scientific narratives of human involvement in zoonotic disease systems, which have largely framed people as ecological disruptors, and discuss three emerging research fields that provide wider system perspectives: how anthropogenic ecosystems construct new niches for infectious disease; feedbacks between disease, biodiversity and social vulnerability; and the role of human-to-animal pathogen transmission ("spillback") in zoonotic disease systems. We conclude by discussing new opportunities to better understand the predictability of human disease outcomes from biodiversity change, and to integrate ecological drivers of disease into health intervention design and evaluation.

Introduction

Biodiversity and ecosystem processes underpin human health and wellbeing, from food and water security to helping maintain a stable climate. Understanding these varied contributions to health, and their potential erosion by anthropogenic stressors such as climate change and land use change, has therefore emerged as one of the most pressing themes in biodiversity science (1–3). Directly measuring and attributing the contributions of biodiversity to health, and so communicating their value to policymakers, is a significant challenge due to their complexity: biodiversity-health links are mostly indirect (e.g. pollination contributing to food security), occur at scales ranging from the microbial to the planetary, and are mediated by numerous social and environmental forces that vary over space and time (4). Zoonotic and vector-borne infectious diseases (i.e. infections transmitted to humans from animals and/or by arthropod vectors) are unusual in being both an urgent concern for global public health, and a relatively direct, visible and measurable link between human health and local ecosystem processes (5). The past decade has seen a swift succession of infectious disease crises, from massive regional epidemics of dengue, chikungunya, Ebola and Lassa fever, to the worldwide spread of COVID-19, Zika and mpox. These have turned public awareness and global health policy attention towards how the biodiversity crisis and climate change may be impacting infectious disease trends, and sparked significant discussions around how best to include nature-based interventions in emerging disease and pandemic risk governance (6–8). This is, consequently, an important historical juncture in which insights from ecology and biodiversity sciences could significantly contribute to improving global public health.

The emerging consensus in disease ecology is that anthropogenic ecosystem degradation and resulting ecological community changes - arising, for example, through habitat fragmentation, land use and climate change - on average tend to increase local pathogen transmission and disease in wildlife (5,9–11). Long-term research in certain well-studied systems, such as Lyme disease in the US and Hendra virus disease in Australia, has demonstrated that these changes can have significant downstream impacts on infection risk to humans (12–14). Yet the intervening role of social and socioeconomic processes in determining realised human disease outcomes remains poorly understood, particularly outside the high-income settings where biodiversity-disease relationships have been most intensely studied. Zoonotic and vector-borne disease systems are inherently socio-ecological in nature: modifications of landscapes for agriculture, industry and cities construct new niches and stressors for hosts, vectors and their pathogens, so shaping infection hazards (9,15); social factors such as gender, wealth, livelihoods and nutrition influence human-wildlife contact and exposure to pathogens, susceptibility to disease, and access to healthcare (16) (Figure 1). What these processes look like varies widely across different regions and socioeconomic settings worldwide, and may depend more on political-economic and historical than proximate ecological circumstances (17–19). Identifying policy strategies to improve the health of both people and ecosystems therefore requires understanding not only the predictability of ecological drivers of disease hazards, but also how human activities, social and economic processes shape, mitigate or amplify them. In this article, we review several major emerging themes in socio-ecological research into biodiversity and zoonotic and vector-borne disease risks. We outline the history of scientific perspectives on ecosystems and zoonotic infection, discuss three priority areas for research, and close by discussing opportunities to better integrate ecological drivers of disease into health intervention planning at various scales.

Towards a renewed 'people and nature' lens on biodiversity and disease

In an influential commentary published a decade ago, Georgina Mace offered a historical view of how scientific framings of biodiversity conservation have evolved over the past half-century, and their consequences for research, practice and policy (20). She outlined how dominant paradigms within conservation science have historically been motivated either by protecting nature's intrinsic value from threats posed by human activities (which she termed *"nature despite people"*) or, more recently, maintaining the benefits and utility that people receive from ecosystems (i.e. ecosystem services; *"nature for people"*). As many scholars have discussed, these long standing scientific framings of the relationships between people, wildlife and ecosystems remain wedded to conservation's western philosophical roots, which position 'nature' as distinct and separate from human societies (and thus as an external resource open to exploitation) (21,22). However, she also highlighted the more recent adoption of transdisciplinary conservation science framings that view humans as an inherent part of ecosystems, and which draw influence variously from socio-ecological systems theory, political ecology and Indigenous perspectives on nature (20). Although still nascent in biodiversity sciences, such *"people and nature"* perspectives – which emphasise the importance of socio-ecological interactions and feedbacks for maintaining resilient and healthy ecosystems – seem particularly well-suited to the challenges of understanding the complex ecologies of health and disease (23,24).

Socio-ecological systems approaches in biodiversity science share both intellectual roots and personnel with foundational early work on emerging infectious diseases, which described how pathogens emerge from the convergence of social, political-economic, eco-evolutionary and environmental forces that span scales from molecular to global (25–28). Over a quarter-century later, this whole systems perspective persists in critical scholarship on disease emergence (18,29–31) and in the holism of organising frameworks such as One Health and Planetary Health, which both frame human health as intrinsically dependent on the health of wildlife and ecosystems (32). However, in practice, scientific knowledge production around emerging disease risks has stayed largely fragmented, discipline-specific, and centred in Global North institutions. Epidemiological research has focused on proximate social risk factors and spread of disease in human populations, while rarely considering wider landscape and ecosystem contexts that shape infection and susceptibility. Conversely, disease ecology research has hugely advanced our understanding of multi-host infection dynamics in changing ecosystems, but while largely taking a simplified view of human involvement centred mainly on ecological disruption (31). Dominant scientific and popular narratives tend to frame zoonotic spillover in *"nature despite people"* conservation terms: human activities and behaviours produce risks by degrading biodiversity, encroaching into wild habitats or creating interfaces (e.g. expanding forest edges, live animal markets), across which pathogens make the "jump" across the nature-society divide to threaten global health security. Humans are characterised as target hosts that receive (mostly) unidirectional flows of pathogens from risky wildlife host species (33,34), with zoonotic spillovers typically framed as rare and high-consequence events that might be preventable, including through conservation interventions. This kind of ecological outbreak narrative has substantially shaped wider perceptions of zoonotic risks and influenced practice in research and policy, from local epidemiological investigation practices (35) to global advocacy around ecological levers for pandemic prevention (6).

Global health security-based framings of biodiversity and disease leave many important questions untouched, even as evidence points towards more complex realities. Only a small subset of known wildlife pathogens follow the pattern of sporadic spillovers leading to sustained global human-to-human epidemics or pandemics (such as SARS-CoVs, influenza A viruses and Ebola) and, although ecological processes may contribute to index case spillover events, following emergence their overall burden and distribution depends on societal factors. In contrast, serological evidence for many zoonoses indicates that human infections are not rare and isolated events, but instead occur frequently in populations at risk, often during childhood, and do not always cause significant disease. This includes World Health Organization priority pathogens such as Lassa, Crimean-Congo haemorrhagic fever and Rift Valley fever viruses (36–38), indicating that many infections typically considered "emerging threats" should instead be treated as neglected endemic diseases (39). Indeed, a recent IUCN situation report concluded that most of the global human burden of zoonotic disease is endemic and attributable to recurring spillovers within anthropogenic habitats, transmitted by vectors, livestock and synanthropic wildlife (40). Growing evidence also suggests that human-to-animal pathogen transmission ("spillback") might contribute substantially to pathogen maintenance and evolution in anthropogenic landscapes - a contrast to the general framing of ecosystems as sources of infection (41).

Together this evidence indicates that cross-species transmission of microorganisms - among wildlife and livestock, to humans and from humans - is a relatively ubiquitous ecological process (42,43). Understanding how biodiversity (and its loss) contributes to the burden of human infectious disease therefore requires a renewed attention to the social and ecological interactions that determine not only human infection risk (i.e. the spillover process) (44), but also the consequent impacts of disease on individuals and populations. This requires asking broader questions, such as: how do human-constructed habitats shape both short-term routes and dynamics of pathogen transmission, and the longer-term evolution of hosts, vectors and microorganisms? Under what circumstances, and for whom, are ecologically-driven changes in infection risk most consequential for health? What sets of interventions - ecosystem-based, health systems-based or otherwise - could be most effective at simultaneously curbing biodiversity loss and reducing the burden of disease? To explore these questions in more depth, the following sections discuss emerging research priorities in three areas: how human landscapes construct distinct niches for infectious disease; feedbacks between ecosystems and social vulnerabilities to infection; and the potential role of spillback in generating and sustaining disease risks.

Ecological communities and disease dynamics within anthropogenic ecosystems

Since most pathogen exposures occur around homes or during the course of people's livelihoods (e.g. via agricultural activities), zoonotic risks arise within environments that are often profoundly shaped by human activities (45). Within these settings, invertebrate vectors such as mosquitoes and ticks play an important role in spreading infection either from animals to humans (e.g. borrelioses, rickettsial fevers, yellow fever, West Nile) or from human to human (e.g. dengue, chikungunya). High densities of livestock can support increased vector populations, act as bridging hosts for wildlife-borne infections, and act as key reservoirs for pathogen evolution and emergence (e.g. highly-pathogenic avian influenzas in industrial poultry setups). Direct transmission from wildlife appears rarer with certain exceptions, such as viruses transmitted by synanthropic rodents (e.g. arenaviruses and hantaviruses). The local taxonomic and

171 functional diversity of host and vector communities thus determines the specific diversity, prevalence,
172 transmission potential and evolution of pathogen hazards (9) (Figure 2).
173
174 Human landscape modifications construct new configurations of infectious disease, both by creating new
175 resource aggregations and ecological opportunities for resilient or invasive hosts and vectors to exploit,
176 and by releasing remaining species from top-down ecological regulation by the predators and competitors
177 that are extirpated (46,47). On average these community transitions tend to decrease biodiversity and
178 favour increasing transmission and prevalence of multi-host pathogens in the remaining, more resilient
179 host species (the dilution effect) (10,48). However, the overall picture for human disease burden is likely
180 complex, as the responses of different pathogens - many of which are maintained in complex multi-species
181 sylvatic cycles - inherently depend on the varied responses of their host and vector assemblages. For
182 example, increasing land use intensity tends to favour rodent and bat zoonotic hosts and certain
183 competent *Culex* and *Aedes* mosquito vectors of human disease, while other groups such as primates and
184 certain *Anopheles* mosquitoes decline (33,49,50). Rather than directional trends in total disease
185 incidence, the end result may often be compositional shifts in pathogen diversity, and thus a transition in
186 disease syndromes and burden in human populations (Figure 2). For example, in Brazil, increasing
187 anthropogenic landscape transformation correlates with a transition away from parasitic diseases
188 transmitted by rural vectors (malaria, leishmaniasis) and towards urban mosquito-transmitted arboviral
189 diseases (dengue, Zika, chikungunya) (51). Importantly, those host species that persist in close proximity
190 to humans tend to be more stressed, which can impact immunocompetence, pathogen prevalence and
191 shedding, and so exacerbate human risks (reviewed in (52)). A key emerging theme for disease ecology is
192 therefore to better understand how infection dynamics are shaped by the distinct ecologies and climates
193 of anthropogenic landscapes, and how this might differ across regional syndromes, modes and intensities
194 of land use (43).
195
196 Understanding and predicting how host and pathogen communities vary across anthropogenic landscapes
197 requires a deeper knowledge of why certain species can tolerate or thrive in close proximity to people
198 (*synanthropy*). At the species-level, species trait syndromes that are associated with resilience to
199 anthropogenic pressures (e.g. fast life histories, wide geographic ranges) also tend to correlate to
200 observed zoonotic reservoir status (53,54) - meaning that (known) zoonotic hosts disproportionately
201 persist in human-disturbed landscapes (33,55). However, increasing evidence indicates that synanthropy
202 is not entirely species-intrinsic, but can be region-specific, historically contingent, and plastic in response
203 to the opportunities and stresses of specific human-driven environments. Such differences can
204 significantly impact spatial interaction networks and foci of infection risk. For example, the space-use of
205 macaques in fragmented forest-agricultural landscapes in Borneo influences their prevalence of zoonotic
206 malaria infection (*Plasmodium knowlesi*), and micro-hotspots of human exposure emerge at forest edges
207 where interactions between macaques, mosquito vectors and people are highest (56); conversely,
208 macaque *P. knowlesi* prevalence is often lower in peri-urban habitat, likely owing to the absence of suitable
209 vectors (57). The social inequalities that structure urban communities, environments and infrastructure -
210 for example, historical district redlining in US cities (58) - can strongly impact local species diversity and
211 composition, with poorly-understood implications for pathogen exposure. At a broader scale, the
212 multimammate rat *Mastomys natalensis* shows significant variation in synanthropy between its East and
213 West African subpopulations, living in closest proximity to human homes in rural West Africa - where it acts

as principal Lassa fever reservoir - but rarely occurring in urban areas due to competitive exclusion by invasive rodent species (59). Over longer timescales, adaptations to urban living can lead to evolutionary transitions to synanthropy (60), such as the divergence of human-specialist *Aedes aegypti* mosquitoes from their tree hole-breeding ancestor several hundred years ago (61), or recent morphological changes in urbanizing UK urban fox populations (62).

Compared to minimally-disturbed ecosystems, anthropogenic habitats often show markedly different - and in many cases more extreme - environmental variability over time, such as seasonal fluctuations in water availability (e.g. transient versus year-round), vegetation and food resources (e.g. cropping cycles) and local climates (e.g. heat island effects). Many zoonotic and vector-borne diseases are climate sensitive, with temperature, rainfall and humidity impacting host and vector population dynamics, physiology and body condition, behaviour and host-pathogen interactions, as well as pathogen persistence in the external environment (63). Theory and a burgeoning empirical literature show that the synergistic effects of land use and climate can therefore significantly modify the timing, intensity and spatial patterns of spillover and epidemics. Such impacts may often be mediated by wildlife host populations and behaviour, such as the combined impacts of El Niño droughts and fragmented habitat in driving Hendra virus spillover from fruit bats (13), or oscillations in rodent host populations and viral infection in agro-ecosystems where seasonal cropping determines food availability (64). However, climate-land use-disease synergies have so far been more commonly studied for vector-borne infections (65), for example evidence that impacts of temperature on malaria transmission in Venezuela are more pronounced in gold mining areas (66), and that dengue incidence increases more sharply after extreme rainfall in rural than urban areas of Brazil (potentially because better urban drainage infrastructure reduces vector breeding sites) (67). Accounting for these kinds of interactions will be crucial to improve outbreak forecasting and preparedness in a changing climate (68). Importantly, as these examples indicate, human-driven landscapes and their amplification or mitigation effects on climate-sensitive diseases are not homogeneous, but are instead shaped by historical and spatial socioeconomic disparities (69). In densely built-up and poorer neighbourhoods, urban heat island effects and low-quality water and sanitation infrastructure can increase both thermal suitability and breeding site availability for mosquito reproduction and vector competence, and thereby concentrate arbovirus burden in marginalised communities (68,70). As we discuss below, such interactions at the nexus of ecology, economy, climate and infection have potential to further reinforce and embed existing social vulnerabilities.

How does the socio-ecological context shape exposure and susceptibility to infection?

To date, research into relationships between ecosystem change and human infectious disease has principally focused on changing assemblages of hosts, vectors and pathogens (i.e. shifts in hazards, as described above; Figure 1) (71). Focusing too narrowly on this dimension, however, risks missing the bigger picture of how ecological degradation contributes to how people are differentially exposed and made vulnerable to infection (25,27). Social inequalities across dimensions such as gender, race, wealth and livelihood, and ensuing biosocial determinants such as nutrition, coinfection, stress and immunocompetence, shape who is most susceptible to disease, who gets sick following infection, and whether disease is then diagnosed, treated and reported (27). The interplay of ecological and biosocial processes in driving infectious disease outcomes is complex (Figure 1) and still poorly understood.

257 Degradation of ecosystem processes can influence not only circulating pathogen diversity but also human
258 immunocompetence and susceptibility to disease (for example by impacting nutrition, water security and
259 coinfection); poverty and social marginalisation increase exposure to many of these pathogens while
260 simultaneously reducing access to healthcare, reinforcing disparities in the health and economic burden of
261 disease (for example between between richer and poorer communities; Figure 1). More ecologically-
262 degraded areas are also less resilient to climate extremes and disasters such as flooding and landslides
263 (72), which affects infrastructure, livelihoods and infection risks, and potentially further increases pressure
264 on local biodiversity. Theoretical models indicate that feedbacks like these can have serious societal
265 implications, for example suggesting the existence of self-sustaining rural 'poverty traps', driven by vicious
266 cycles of disease and economic burden (73,74). The statistical challenge of disentangling such complex
267 relationships from observational data means that empirical examples remain sparse, with some notable
268 exceptions: for example, there is evidence for negative malaria-deforestation feedbacks in the Brazilian
269 Amazon (75).

271 In some circumstances, the combined effects of ecosystem degradation, climate change and
272 socioeconomic inequalities can simultaneously increase both people's exposure to (multiple) wildlife-
273 borne pathogens and, once infected, their susceptibility to disease. Such interactions between multiple
274 pathologies are studied in public health as *syndemics* (synergistic epidemics), i.e. situations where the co-
275 occurrence of more than one health or social condition produces worse outcomes than either in isolation,
276 with a canonical human example being substance misuse, violence and HIV/AIDS (76–78). Biodiversity-
277 disease research has largely focused on hazards within single-pathogen single-disease systems, so
278 shared drivers and probable syndemic interactions with wider social, ecological and health conditions -
279 including inequalities, comorbidities and coinfection - are still poorly understood (79–81). Studying these
280 interactions has clear importance for public health, for example by helping to understand why many
281 important zoonotic and vector-borne infections show such wide variation in clinical severity. Social and
282 ecological circumstances shape individuals' lifetime pathogen exposure histories and thereby
283 susceptibility to subsequent infections, which could either increase (e.g. heterotypic dengue virus
284 infection (82)) or decrease the risk of severe disease (e.g. early-life microbial exposures potentially priming
285 the immune system against future infections (83,84)). Closely overlapping niches across different
286 pathogens - arising via shared host and vector communities - can also cluster coinfection risks in space,
287 time and in response to ecological drivers, increasing the potential for complex multi-pathogen
288 interactions (43). For example, *Aedes*-borne arboviruses (e.g. dengue, Zika, chikungunya) and water-borne
289 infections (e.g. leptospirosis) tend to cluster in poorer and peripheral neighbourhoods of tropical cities
290 (85). Coinfection can worsen or complicate individual disease outcomes (80), but co-circulation at
291 population level can also worsen outcomes by interfering with prompt diagnosis, especially for rarer and
292 non-specific infections. In West Africa, initial misdiagnosis of Lassa fever as malaria can delay appropriate
293 treatment by several days, increasing the risk of mortality (86). Historical and ongoing ecosystem
294 degradation and social marginalisation, for example in racialised and Indigenous communities, also impact
295 rates of noncommunicable disease in ways that exacerbate susceptibility to many infections (87); one
296 particularly visible recent example was the clustering of COVID-19 morbidity and mortality in many
297 marginalised communities (88). One so-far-neglected role for ecologists in supporting public health
298 decisions would be better understanding and mapping such compound hazards, their intersection with
299 other health and economic conditions; and, crucially, the upstream forces (including neocolonial

relationships with Global North states and financial interests; Figure 1) that generate and sustain them (17,18).

These socio-ecological complexities present both opportunities and challenges for designing and evaluating interventions to reduce disease risks. Social and behavioural change interventions can be designed to target human activities that increase risk of direct and indirect forms of animal contact (e.g., wildlife hunting or use of animal products as fertiliser, food storage practices), aim to increase knowledge, risk perception, or self-efficacy, and take care not to stigmatise cultural and need-based practices (89). Ecological interventions could be designed to reduce sylvatic circulation and human hazards for groups of priority pathogens at regional levels, for example via conservation, restoration or agroecosystem management activities (23,90). However, diseases differ widely in their hosts, pathogen life cycles, and socio-ecological contexts, indicating that effective one-size-fits-all ecological interventions may be rare (43). Certain global solutions might even risk unintended consequences for other dimensions of health if applied too broadly. For example, tighter regulation of deforestation and wildlife trade have been proposed as global solutions to mitigate emerging zoonosis risks (6). However, the militarised turn in conservation (91) and examples of land dispossessions under schemes such as REDD+ (92) indicate that the way that such programmes are implemented could have significant health consequences for local and Indigenous communities, with potential to be unjust and counterproductive; these may include loss of land, nutrition and income sources, stress, and exposure to other infections. More positively, multiple social and ecological 'weak points' could be targeted to variously interrupt transmission, reduce susceptibility to disease and improve healthcare access, and the most effective strategies for net health benefits may consider several at once. For example, hunting of wild meat is an important transmission pathway for multiple zoonoses, but also important for food, nutrition, and economic security (93–95); any disease risk interventions targeting this pathway would need to take these interactions (and possible unintended consequences of regulation) into account (Figure 1). The early and sustained involvement of affected communities is therefore critical to ensuring the design of socio-ecological interventions that are equitable, recognise and value local knowledges and perspectives, and thereby provide net benefits to health and wellbeing (see '*Future horizons*').

The role of human-to-animal pathogen transmission in sustaining zoonotic risks

Scientific narratives around disease emergence have traditionally centred on humans as target hosts of spillover infections from wildlife reservoirs. This anthropocentric view has been shaped, in large part, by ongoing (and understandable) research biases towards hosts and pathogens of known medical relevance to humans (96). However, continual improvements in genomic/metagenomic tools and network science methods now offer an increasingly broad, holistic view of a continuous flow of microorganisms among humans, non-humans and their environments (42,97). One important implication of this shift in perspective is that people and livestock are, in fact, increasingly large and well-connected nodes in multi-species pathogen sharing networks, and so may play substantial roles in transmitting infection to other species (i.e. zooanthroponosis or "spillback") (98). Pathogen transmission from people to wildlife poses a well-known threat to certain wildlife populations, such as respiratory virus spillback to some primates (41). In contrast, much remains unknown about how human-to-wildlife or livestock-to-wildlife transmission -

342 particularly to synanthropic species with whom we share landscapes - might be important in creating and
343 sustaining zoonotic risks.
344
345 The consequences of spillback can include the establishment of reservoirs within new hosts and
346 geographic locations: for example, multiple introductions of SARS-CoV-2 have established endemic
347 circulation in white-tailed deer in North America, which could ultimately serve as a source of novel variants
348 to re-emerge in humans (99). Human-to-wildlife transmission might also contribute to maintaining long-
349 term (enzootic) pathogen prevalence in highly dynamic synanthropic host populations, such as rodent
350 hosts of arenaviruses (100), but its importance remains poorly understood. Livestock populations can also
351 play important epidemiological roles, including by sustaining epizootics, supporting arthropod vector
352 populations, and acting as conduits for infection between disparate geographical areas; for example,
353 cattle may play a significant (but still poorly-defined) role in the ecology of Crimean-Congo haemorrhagic
354 fever (101). Genomic tools offer a promising route to identify these probable sources and sinks for infection
355 in multi-host systems (102); one recent such study at the global scale suggests that spillback may be very
356 widespread, although this pattern is difficult to conclusively distinguish from human-centric surveillance
357 biases at this scale (98). Moving forward, more granular datasets that are not solely focused on wildlife
358 hosts or vectors - for example, samples and individual-level metadata collected from human, wildlife and
359 livestock populations in the same landscapes (97) - will afford opportunities to test hypotheses around the
360 relative frequency, drivers, and epidemiological significance of spillback in human-driven landscapes.
361
362 **Future horizons**
363
364 Over the last two decades, research into biodiversity and infectious disease has largely been motivated by
365 two parallel missions - to reduce biodiversity loss and ecosystem degradation, and to improve human
366 health - and the tensions and trade-offs between these two desired outcomes have not always been as
367 clearly articulated as their possible synergies. Recent progress towards a reconciled scientific consensus
368 on biodiversity-disease relationships has helped to defuse long-standing debates in disease ecology and
369 provided important nuance (5,48,103). Yet we remain far from an actionable science that could, for
370 example, provide well-grounded predictions of how specific land use or conservation policies would affect
371 the net burden of human disease (and its wider social and economic ramifications). As we have discussed,
372 this issue arises from both the inherent challenge of measuring the many contributions of biodiversity to
373 human health, and the need to better understand the social settings that drive pathogen transmission and
374 disease (Figure 1). Nonetheless, patterns of infectious disease are underpinned by general ecological and
375 evolutionary processes that should provide sources of predictability that can inform policy, even across
376 diverse social contexts. In the later stages of her career Georgina Mace focused on bridging
377 communication gaps between research and policy; in that spirit, this final section offers a reflection on
378 emerging opportunities to implement biodiversity-disease knowledge to improve health.
379
380 Looking forward, a crucial step in any disease system is to identify the most effective intervention
381 strategies, which could aim to reduce hazards, exposure, vulnerability, or potentially a combination of all
382 three (Figure 1). More targeted activities could address proximal drivers of specific diseases, focusing
383 variously on lowering pathogen prevalence within host populations (e.g. land use policies, ecological
384 restoration, host or vector control, wildlife vaccination); interrupting human exposure pathways (e.g.

385 reducing hunting or trade of high-risk species, improving water and sanitation infrastructure); reducing
386 human susceptibility to disease (e.g. vaccination, improving food security, better prevention and
387 treatment of comorbidities); or ensuring prompt diagnosis and treatment following infection (e.g.
388 improving health systems infrastructure and accessibility). Improvements in statistical methods for causal
389 inference from observational data are increasingly entering ecology from the health sciences (104).
390 Applied to infection datasets from people and wildlife, these offer a promising means to infer likely drivers
391 of disease risk (e.g., climate, land use, poverty, etc.), which can then inform the iterative design and
392 evaluation of combined ecological, social and/or health system interventions. Large-scale, long-term
393 human disease surveillance datasets are crucial to robustly attribute climatic drivers (105,106), however
394 these datasets suffer from pervasive geographical surveillance biases that make detecting more localised,
395 transient, lagged or synergistic driver effects - such as local land use changes - far more challenging (43).
396 Unpacking how ecosystem-based interventions could reduce disease will often instead require starting
397 locally. Simultaneous surveys of people, wildlife, livestock and environments enable the identification of
398 key pathways and drivers of cross-species infection; combined with emerging genomic and network
399 science methods these have potential to significantly improve our understanding of pathogen diversity
400 and sharing across space, time and species.

401

402 These natural sciences-based approaches can provide a starting point by identifying critical risk pathways
403 to target, but achieving truly effective and just interventions will require breaking with the dominant
404 paradigm of exploitative, top-down and parachute research. Early and ongoing involvement of affected
405 communities in co-design and implementation is key to ensuring that proposed interventions are rooted in
406 local knowledge, perspectives and existing resilience or risk-mitigation strategies. Participatory studies
407 and intervention scenario mapping with at-risk communities, as well as integrating local representatives
408 into research teams and accountability structures, can help in achieving this (107). Ensuring sustainable
409 net benefits to local health and wellbeing will likely involve accounting for numerous ecosystem services
410 beyond just disease risk, as well as addressing social disparities such as healthcare access. Recent and
411 ongoing work on zoonotic malaria and Lassa fever has shown the value of such detailed socio-ecological
412 research for informing disease prediction and prevention (56,108). Shifting research norms towards
413 participatory and co-design approaches will require tackling significant institutional and structural barriers.
414 These include improving academic institutional and funding support for interdisciplinary research and the
415 development of equitable North-South research partnerships, recognising that these relationships are
416 slower to establish; require the development of trust, shared values and working frameworks across
417 significant geographical and economic distances; and so may often be slower to produce measurable
418 outputs. They also include the need for institutional commitments and funding to close persistent capacity
419 gaps between Global North and Global South research communities that sustain research and knowledge
420 inequities, including (but not limited to) building analytical, data science and modelling capacities, and
421 disincentivising parachute research.

422

423 In parallel with local-scale interventions, there is also an obvious need to provide better recommendations
424 around biodiversity impacts on disease to regional and global health and environmental policymakers. This
425 need has been clearly demonstrated in, for example, the formation of the United Nations' cross-sectoral
426 One Health Quadripartite collaboration and One Health High-Level Expert Panel (8), and recent discussions
427 around the priority of ecosystem-based "primary prevention" activities in global emerging disease

428   governance (6,7). At these broader scales, the limitations and biases inherent to pathogen surveillance
429   data for many emerging infections become more visible and problematic (43). However, it also becomes
430   possible to leverage data from wider biodiversity and environmental monitoring initiatives, to identify the
431   ecological changes that underpin disease risks; from host community responses to environmental
432   pressures (33,109), to geographies of viral diversity and their eco-evolutionary histories (110), to regional
433   syndromes of anthropogenic landscape and socioeconomic pressures and their political economic drivers
434   (111,112). Expanding this potential will require facilitating discussions between biodiversity monitoring,
435   disease ecology and disease surveillance communities, to align on data and metadata standards, ensure
436   relevant data are collected and reported wherever possible, and ensure proper source attribution (for
437   example, Verena's PHAROS database standard; https://pharos.viralemergence.org/). An important future
438   analytical challenge will then be to identify at what spatial scales such macro-level monitoring data could
439   be translated to useful information for disease prediction and intervention activities. For example, host,
440   vector and pathogen communities may show relatively consistent and predictable responses to land use
441   and climate change within individual ecoregions or regional biomes (113,114) (Figure 2). In many cases, it
442   may be possible to align these with similarly region-specific typologies of land change, agroecology,
443   priority social and health issues, and policy stakeholder networks, where such data exist and are accessible
444   (111). Integrating the disease costs of ecosystem change into health economic analyses - including the
445   distinct economic burdens of endemic and epidemic infections - may also be most tractable and policy-
446   relevant at these regional or national scales (115). There are clear synergies, then, between enhancing
447   biodiversity monitoring and strengthening health systems-based disease detection, treatment and
448   response: both have potential to significantly improve our baseline knowledge and situational awareness,
449   which will be especially valuable in the context of rapid global change.
450
451   Finally, "people and nature" perspectives on wildlife-borne disease offer a reminder to pose questions that,
452   as Richard Levins wrote, are big enough to encompass the problems we seek to address (25,116). As we
453   have discussed, much of the research into biodiversity and disease dynamics over the last two decades
454   has made important advances via the study of specific wildlife disease systems, theory development, and
455   proximal rather than upstream drivers. Yet the COVID-19 pandemic highlighted the current limits of this
456   ecological knowledge to either inform specific health and environmental decisions, or contextualise
457   disease emergence as a product of the wider, interlinked systemic crises of capital, inequality, biodiversity
458   and climate change (27,30) (Figure 1). Looking forward, socio-ecological approaches to biodiversity and
459   infectious disease research have great potential to contribute to this much wider understanding, and so
460   help to identify and advocate for transformative system-wide interventions - including expanding global
461   access to high-quality healthcare, climate change mitigation, economic redistribution, and challenging
462   powerful financial interests - that will support sustainable, just and healthy futures for ecosystems and
463   people.
464

Author contributions

466   Concept – RG, DWR, KEJ. Writing (initial draft) – RG. Writing (review and editing) – RG, DWR, SF, KEJ.
467

515

516 **Figure 1: Local social-ecological feedbacks generate and reinforce infectious disease risks.** Human

517 zoonotic and vector-borne disease risks (blue triangle) arise from the convergence of circulating pathogen

518 hazards within local ecosystems (shaped by host and pathogen diversity and prevalence), drivers that

519 create opportunities for exposure (e.g. agricultural activities, urbanisation and infrastructure, land

520 conversion, wildlife hunting, hydrometeorological extremes), and individual- and population-level factors

521 that influence vulnerability to disease and epidemics (e.g. individual physical condition and immunity;

522 health systems access and functionality; inequality and social cohesion). Although often discussed and

523 studied as separate phenomena (71), these processes are interdependent and subject to numerous

524 feedback effects (orange arrows and text) that can generate or reinforce disease risks. For example, a high

525 burden of disease (hazard) can impact community livelihoods and poverty (vulnerability), leading to

526 increased reliance on local environment, land use or wildlife (exposure) which in turn can further impact

527 local ecological communities (hazard). All these local processes are shaped by global forces including

528 unequal and extractive power relations, climate change and biodiversity degradation (yellow boundary),

529 which can simultaneously impact pathogen hazards, exposure processes, and vulnerabilities.

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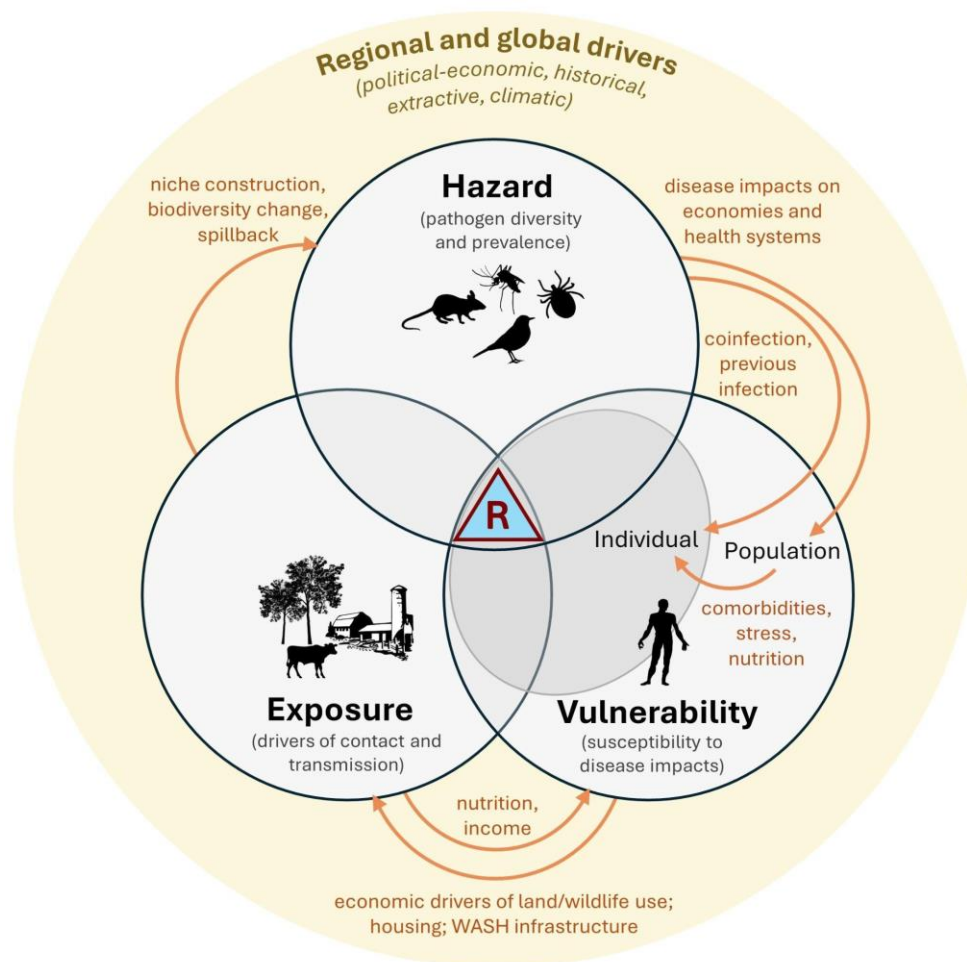
Figure 2: Compositional shifts in host and disease diversity under intensifying land use. Increasing
intensities of anthropogenic land use intensity, from minimally disturbed habitat, to rural ecosystems,
towards high-intensity agriculture and urbanisation, are generally associated with declines in overall local
biodiversity (top graph). This overall trend masks compositional shifts in host and arthropod vector
diversity that can significantly alter the local diversity and burden of zoonotic and vector-borne disease
(ZVBD; middle and bottom graphs), including losses of more human-sensitive taxa (and their specialist
pathogens; red line), the rise of highly synanthropic species (yellow line) and variable or hump-shaped
responses of other species (blue line). These community shifts will reshape local disease transmission
ecologies and create differences in disease diversity and burden in different types of landscape (examples
in bottom graph captions). A deeper understanding of these changes in host, vector and pathogen
diversity in response to human pressures at the level of biomes or ecoregions - for example, through
leveraging global biodiversity data - may help to improve prediction of the infectious disease
consequences of biodiversity change for many under-studied pathogens (see *"Future horizons"*).

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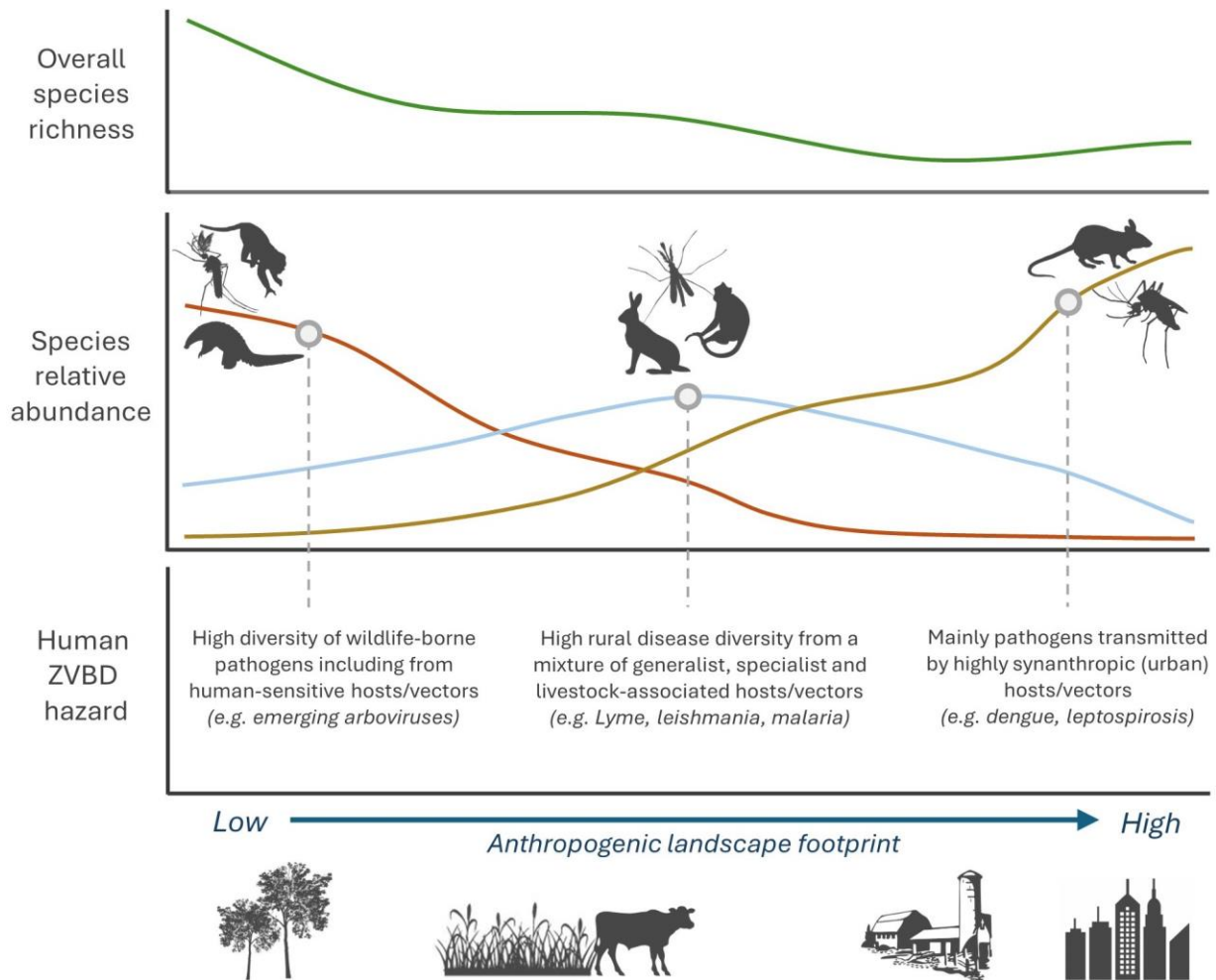
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