1   **Title:** Global Change and Emerging Infectious Diseases

2   **Authors:** Nicole Nova[1,*], Tejas S. Athni[1], Marissa L. Childs[2], Lisa Mandle[1,3,4], Erin A. Mordecai[1]

3   **Affiliations:**
4   [1]Department of Biology, Stanford University, Stanford, CA 94305, USA
5   [2]Emmett Interdisciplinary Program in Environment and Resources, Stanford University, Stanford,
6   CA 94305, USA
7   [3]Natural Capital Project, Stanford University, Stanford, CA 94305, USA
8   [4]Woods Institute for the Environment, Stanford University, Stanford, CA 94305, USA

9   ***Corresponding author:**
10   Nicole Nova, Department of Biology, Bass Building, 327 Campus Drive, Stanford, CA 94305
11   nicole.nova@stanford.edu

14   **Abstract**

15   Our world is undergoing rapid planetary changes driven by human activities, often mediated by
16   economic incentives and resource management, affecting all life on Earth. Concurrently, many
17   infectious diseases have recently emerged or spread into new populations. Mounting evidence
18   suggests that global change—including climate change, land-use change, urbanization, and global
19   movement of individuals, species, and goods—may be accelerating disease emergence by reshaping
20   ecological systems in concert with socioeconomic factors. Here, we review insights, approaches, and
21   mechanisms by which global change drives disease emergence from a disease ecology perspective.
22   We aim to spur more interdisciplinary collaboration with economists and identification of more
23   effective and sustainable interventions to prevent disease emergence. While almost all infectious
24   diseases change in response to global change, the mechanisms and directions of these effects are
25   system specific, requiring new, integrated approaches to disease control that recognize linkages
26   between environmental and economic sustainability, and human and planetary health.

1   Introduction

Emerging infectious diseases (hereafter EIDs) pose a threat to humans, domestic animals, and wildlife (Woolhouse & Gowtage-Sequeria 2005). In recent decades, the interconnectedness between global change—including climate change, land-use change, and increased global mobility—and infectious disease emergence and spread has become a more widely accepted phenomenon (Barnosky et al. 2012; Daily & Ehrlich 1996; Myers 2017; Plowright et al. 2021). Concurrently, we have witnessed an increased rate of emergence of novel infectious diseases (e.g., Hendra, Nipah, SARS, MERS, swine flu [influenza A virus H1N1], COVID-19), establishment of diseases in new geographic regions (e.g., West Nile, Zika, dengue, yellow fever, Lyme), and resurgence of previously controlled diseases (e.g., dengue, Ebola, malaria, hantavirus diseases). The ongoing COVID-19 pandemic that continues to disrupt daily life, in addition to the escalating climate crisis, raises the critical question: Why are diseases emerging and expanding so rapidly, and how can we prevent future planetary health crises?

Emerging pathogens, like any invasive species, go through four distinct stages of invasion: introduction, establishment, spread, and impact (Levine 2008). For pathogens specifically, the process by which a pathogen spills over into a novel host population using the invasive species framework is as follows (Plowright et al. 2021). Introduction is when a novel species is exposed to a pathogen from a different species (usually a pathogen reservoir species). Establishment occurs when the novel species becomes infected. Next, spread occurs when the novel species is able to transmit the pathogen among individuals of the same species. Finally, impact is when pathogen transmission in the novel species causes outbreaks, either locally (epidemics) or globally (pandemics). Although there are numerous EIDs currently circulating in domestic animals and wildlife that do not infect humans, we focus on diseases that can. However, we acknowledge that EIDs in animal and plant populations are a concern, with potential implications for human health as well (Myers 2017).

Despite being unsustainable in its current form, post-industrial economic development has undoubtedly facilitated technological and scientific breakthroughs. At the same time, these advances have allowed mainstream Western research to finally illuminate the unsustainable nature of today's resource management, and the intricate connections between global change and EIDs, which other cultures have long recognized (Glidden et al. 2021; Griffin 2009). Although there are disease prevention practices underway to prevent or mitigate EID outbreaks on local scales (Glidden et al. 2021; Jones et al. 2020), the future of our planet and human health would benefit from sustainable resource management and equitable economic development at a global scale (Myers 2017). Major knowledge gaps remain, especially concerning the outcomes of proposed solutions to prevent or mitigate EIDs (Myers 2017). We need to acquire a better understanding of the underlying disease ecology, understand how it intersects with socioeconomics and resource economics to guide science-based policy, and develop solutions that are simultaneously feasible, sustainable, nature-based and society-based (Myers 2017).

As EIDs may be attributed to global change (Daily & Ehrlich 1996; Myers 2017), and global change is often driven by economic incentives, it is imperative that economists, epidemiologists, and disease ecologists work together to identify key mechanisms linking global change to disease emergence, and develop economic frameworks that value planetary health outcomes. Here, we provide a review of the evidence of proposed mechanisms by which key aspects of global change—anthropogenic disturbances of ecosystems and other human activities—drive disease emergence, using well-researched disease case studies from the disease ecology literature, with an emphasis on diseases that have (re)emerged in the last 25 years. To foster more collaboration between fields of

72 resource economics and disease ecology, we present foundational knowledge and recent case studies
73 from disease ecology for the resource economics audience by highlighting the need to account for
74 nonlinearity and complexity when modeling disease systems, and to search for mechanisms by which
75 anthropogenic disturbances affect disease outcomes. In addition, we briefly summarize technological
76 and modeling advances used or developed by disease ecologists to bridge methodological approaches
77 between the fields. Finally, we discuss the economic impacts of EIDs beyond medical and public
78 health costs, and possible feedback of EIDs on global change.

79 **2   Insights from disease ecology**

80 Infectious diseases are embedded within ecological systems in which interactions between
81 individuals, environmental factors, and other species determine the emergence and transmission of
82 disease. Most EIDs in humans originate in wildlife and/or are transmitted by vectors such as biting
83 arthropods like ticks, mosquitoes, flies, and fleas (Woolhouse & Gowtage-Sequeria 2005). As a
84 result, these diseases are connected to environmental conditions (e.g., temperature, rainfall,
85 topography, biodiversity, and species interactions) and human social systems (e.g., the built
86 environment, mobility, behavior, and immunology). Disease ecology, the study of how infectious
87 disease dynamics respond to these environmental and human drivers, aims to understand and predict
88 disease emergence and its consequences for humans and other species.

89   It is important to account for complexity and nonlinearity in economic systems. The same
90 applies to infectious disease systems (Anderson & May 1979; Nova et al. 2021; Sugihara et al. 2012).
91 Many ecological and social drivers interact to affect infectious disease dynamics, often via nonlinear
92 responses, and all of it is stochastic, adding to uncertainty in actual outcomes. For example,
93 secondary case distributions are strongly skewed for COVID-19 and other diseases, yielding
94 epidemics that are highly stochastic but explosive (Lloyd-Smith et al. 2005). To facilitate
95 collaboration among economists, disease ecologists, and epidemiologists it is important to share
96 methods and concepts used in the respective fields. In this section, we highlight the complex nature
97 of infectious disease systems with examples, provide an overview of the mechanistic relationship
98 between disease and environmental change, and share recent developments in technology and
99 methods used in disease ecology.

100 **2.1   Nonlinearity and complexity in infectious disease systems**

101 Key features of ecological systems, including infectious diseases, are their nonlinearity and
102 complexity (Anderson & May 1979; Keeling & Rohani 2008). Infectious disease transmission
103 responds nonlinearly to susceptible host availability: transmission first grows exponentially as a
104 pathogen emerges in a population, then reaches an inflection point as susceptible hosts are depleted
105 by infection and immunity, then either burns out, reaches an endemic equilibrium, cycles, or exhibits
106 other complex behaviors (Anderson & May 1979; Keeling & Rohani 2008). Transmission dynamics
107 can be predicted based on the transmission rate, recovery or mortality rate, and the rate at which
108 susceptible hosts are replenished through births, waning immunity, or migration (Anderson & May
109 1979; Keeling & Rohani 2008).

110   Not only does infectious disease transmission vary nonlinearly with susceptible host
111 availability, but the parameters that govern transmission, infection, and other vital rates can also
112 respond nonlinearly to environmental conditions (Figure 1), such as temperature and species
113 interactions, and even in response to awareness of disease in others (Heesterbeek et al. 2015; Koelle
114 & Pascual 2004; Weitz et al. 2020). For example, a 1ºC increase in temperature can increase,
115 decrease, or have null effects on transmission of a vector-borne disease depending on whether the

116 current temperature is below, above, or at the thermal optimum for transmission (Figure 1a)
117 (Caldwell et al. 2021). Moreover, many EIDs circulate through multiple hosts, including wildlife,
118 livestock, and vectors, coupling nonlinear infectious disease dynamics in multiple populations that
119 are linked by migration, contact, and pathogen spillover (Lloyd-Smith et al. 2009; Plowright et al.
120 2017).

2.2   Mechanisms of disease emergence via environmental change

122 Human population growth, industrialization, and mobility have led to pervasive changes in the
123 Earth's systems, including climate, land use, species invasions and extinctions, and global movement
124 of goods, plants, humans and other animals (Barnosky et al. 2012). These changes affect both wild
125 and domestic species, and human contact with them, through multiple pathways that can lead to
126 disease emergence (Lloyd-Smith et al. 2009; Plowright et al. 2017). For example, climate change can
127 alter the distribution, abundance, and seasonality of parasites, reservoir hosts, and vectors (Altizer et
128 al. 2013; Lafferty & Mordecai 2016). Invasions of mosquito vectors like *Aedes aegypti* and *Ae.*
129 *albopictus* have led to arbovirus (e.g., dengue, Zika, yellow fever, and chikungunya) outbreaks in
130 novel regions, including dengue and chikungunya in Italy, France, the United States, and islands off
131 the coast of Europe and Africa (Adalja et al. 2012; Amraoui & Failloux 2016; Gérardin et al. 2008;
132 Tomasello & Schlagenhauf 2013). Land use change creates edge habitats between human-made and
133 wild landscapes, facilitating human contact with wildlife and vectors that can lead to disease
134 transmission (Faust et al. 2018; MacDonald & Mordecai 2019). Loss of native species and habitats
135 can affect the health and behavior of wild animals that are reservoirs for EIDs (Plowright et al. 2015).
136 Mobility rapidly transports pathogens and vectors between populations and facilitates emergence
137 (Wesolowski et al. 2015). Anthropogenic global environmental change is intricately connected to the
138 emergence of infectious diseases in humans, highlighting the urgent imperative of connecting
139 planetary health to human health (Myers 2017).

2.3   Recent technological and modeling advances in disease ecology

141 Recent technological advances and modeling approaches in disease ecology have furthered our
142 understanding of EIDs. Breakthroughs in computer science, statistics, and data science have enabled
143 modelers to store, analyze, and utilize larger data sets, and run more informative simulations of
144 disease emergence. New technology, such as high-throughput sequencing and cloud-based platforms
145 for remote sensing data (e.g., Google Earth Engine), has improved data collection and sharing,
146 allowing disease ecologists to access and synthesize several data types to better identify spillover
147 pathways and disease transmission at various scales. For example, using viral genomic data in
148 addition to epidemiological data from a recent yellow fever outbreak in Brazil, Faria et al. (2018)
149 were able to identify the source of human cases as independent spillover events from monkeys, rather
150 than from transmission among humans as initially believed.

151   Modeling approaches in disease ecology often need to account for nonlinearity and complexity,
152 which is useful for understanding mechanisms of disease emergence. In recent years, there has been
153 an expanding array of causal inference methodologies for nonlinear systems in disease ecology often
154 borrowed from other fields, including instrumental variable analysis from econometrics (MacDonald
155 & Mordecai 2019), structural equation modeling from genetics (Pearl 2010), and empirical dynamic
156 modeling (EDM) from dynamic systems theory (Nova et al. 2021; Sugihara et al. 2012). Examples of
157 other methods developed within the field of disease ecology or epidemiology include: stochastic
158 simulation and inference algorithms for epidemic modeling using partially-observed Markov
159 processes (King et al. 2016), inference of incidence from viral genetic data using phylodynamics
160 (Volz et al. 2009), and time series methods for inference of disease drivers and forecasting (Becker &

161 Grenfell 2017), including forecasting uncertainty using EDM (Nova et al. 2021). In the last two
162 decades, sophisticated mechanistic and data-driven models—specifically tailored for infectious
163 disease dynamics, including EIDs—have unraveled important relationships between aspects of global
164 change and disease emergence.

3   Anthropogenic disturbances linking global change to emerging infectious diseases

166 Many human activities are economically driven and occur at a global scale. Several of these activities
167 have significant environmental impacts and cascading effects on EIDs. Here, we give an overview of
168 how these global anthropogenic changes affect infectious disease systems, showcasing the evidence
169 of mechanisms linking anthropogenic disturbance of the environment to disease spillover,
170 transmission, and emergence.

3.1   Climate change

172 Anthropogenic climate change is driven by human activities elevating carbon emissions into the
173 atmosphere, and it may have profound effects on EIDs. Diseases with environmental components in
174 their transmission cycles, including vector-borne, water-borne, food-borne, soil-borne, and
175 environmental contaminant pathogens, are climate sensitive because of the thermal physiology of
176 ectotherms (i.e., animals that regulate body temperature via external heat sources) and the impact of
177 water and humidity on survival and in some cases reproduction (Altizer et al. 2013; Mordecai et al.
178 2019; Shocket et al. 2021). Because temperature, humidity, and rainfall affect multiple aspects of
179 vector and host life cycles and transmission cycles, and these responses are often hump-shaped or
180 exhibit threshold effects (Figure 1), anticipating climate-driven changes in infectious disease systems
181 is a challenge (Caldwell et al. 2021; Lafferty & Mordecai 2016; Mordecai et al. 2019; Shocket et al.
182 2021). Climate change is expected to drive increases in some diseases and regions and decreases or
183 shifts in others (Altizer et al. 2013; Lafferty & Mordecai 2016). Moreover, attributing changes in
184 infectious disease dynamics to climate change is challenging because of the complexity of these
185 systems in which multiple environmental changes are concurrent (Parmesan et al. 2013).

186   Here, we provide mechanistic evidence linking EIDs to climate change with an emphasis on
187 climate warming, as it is a well-supported effect of climate change and mean temperature has the
188 most clearly understood effect on EIDs. However, we do acknowledge that other facets of climate
189 change can also contribute to disease emergence. For example, Rift Valley fever is linked to El Niño-
190 Southern Oscillation (ENSO), and extreme ENSO events are predicted to increase in frequency and
191 strength with climate change (Shocket et al. 2021). Further, for many mosquito-borne diseases, the
192 effects of rainfall on disease transmission are complex in addition to being nonlinear, since the
193 rainfall–transmission relationship largely depends on the vector species and pathogen (Figure 1b)
194 (Shocket et al. 2021).

195   The clearest evidence of climate change-mediated infectious disease emergence is for vector-
196 borne and environmentally transmitted diseases emerging at higher latitudes and elevations with
197 climate warming (IPCC 2021 Working Group II Chapter 2). This is because the change from disease
198 absence to presence is the easiest to observe and connect to climate warming. For example, *Ixodes*
199 spp. ticks, tick-borne encephalitis, and Lyme disease have increased in incidence and expanded in
200 range in northern parts of Europe and North America (Caminade et al. 2019; Couper et al. 2021;
201 Gilbert 2021). In Nepal, dengue, chikungunya, malaria, and Japanese encephalitis and their vectors
202 have emerged and expanded upslope (Dhimal et al. 2021). Parasitic, vector-borne, and rodent-borne
203 diseases like cryptosporidiosis, filariasis, tularemia, and hantavirus-induced hemorrhagic fever have
204 emerged in the Arctic and sub-Arctic (Huber et al. 2020; Pauchard et al. 2016; Sachal et al. 2019). In

205 Europe, helminth (worm) parasites and host snails are increasing and expanding with climate change
206 (Caminade et al. 2019). These widespread changes in range limits of diseases and vectors show that
207 climate change is already affecting human and wildlife health.

208   Conversely, the impacts of climate change on diseases within their currently endemic ranges
209 are more difficult to detect in the face of multiple changing environmental conditions. Moreover,
210 climate change is expected to expand the distributions and incidence of some diseases in some
211 regions, while contracting them in others (Lafferty & Mordecai 2016; Mordecai et al. 2019) (Figure
212 1a). For 11 different pathogens transmitted by 15 mosquito vector species, the optimal temperature
213 for transmission varied from 23–29ºC and the temperatures that prevented transmission ranged from
214 9–23ºC at the low end and 32–38ºC at the high end (Mordecai et al. 2019). This implies that diseases
215 with cooler thermal optima and limits—including West Nile and other temperately distributed
216 mosquito-borne viruses, as well as malaria, with an optimum of 25ºC—are likely to increase in
217 temperature suitability at their cooler-temperature range limits, while declining in warmer areas
218 (Mordecai et al. 2019, 2020; Shocket et al. 2020). However, diseases with warmer thermal optima
219 like dengue and Zika, which peak at 29ºC, are expected to broadly expand in climate suitability and
220 could emerge at higher elevations and latitudes, in places where housing quality, socioeconomic
221 conditions, and lack of adequate vector control permit transmission (Mordecai et al. 2019, 2020;
222 Ryan et al. 2019, 2021). The widespread expansions of the invasive mosquitoes *Ae. aegypti* and *Ae.*
223 *albopictus*, which are competent for transmitting many viruses, poses a substantial threat of arbovirus
224 emergence following in the tracks of the four serotypes of dengue, chikungunya, and Zika, each of
225 which have (re)invaded across the Americas in the last three decades (Gubler 2010; Gubler et al.
226 2017; Lounibos 2002). Likewise, the invasion and rapid spread of West Nile virus across North
227 America exemplifies how an introduced pathogen can rapidly establish in novel vectors and wildlife
228 hosts and cause spillover to humans when climate suitability is high (Kilpatrick et al. 2010; Paull et
229 al. 2017).

230   In contrast to the anticipated changes in *temperature suitability* for disease transmission,
231 realized changes in *disease incidence* can be much more complex. Dengue incidence is rapidly
232 increasing globally, particularly in the Americas, Asia, and Africa (Stanaway et al. 2016), but the
233 degree to which climate change has driven these expansions versus unplanned urbanization,
234 population growth, and mobility remain difficult to quantify because they act synergistically (Gubler
235 2010; Stewart-Ibarra & Lowe 2013; Wesolowski et al. 2015). Conversely, malaria declined
236 dramatically worldwide during the 20th century (Gething et al. 2010), and particularly steeply in most
237 of sub-Saharan Africa in the last 20 years due to successful intervention campaigns (Bhatt et al.
238 2015; Smith et al. 2013) and potentially also due to declining climate suitability (Mordecai et al.
239 2020). The few exceptions in which malaria has increased have been documented in highland Kenya,
240 Ethiopia, Colombia, and Nepal, which are attributed to climate warming (Dhimal et al. 2021; Siraj et
241 al. 2014) in concert with land use change (Afrane et al. 2012), and in Brazil, Colombia, Peru,
242 Guyana, French Guiana, Venezuela, and Nicaragua, which are attributed to deforestation, gold
243 mining, and other land use changes (Douine et al. 2020; MacDonald & Mordecai 2019). In sum, even
244 when climate warming increases the temperature suitability for disease transmission, this does not
245 always translate into higher incidence of disease because of the concurrent effects of socioeconomic
246 development, land use change, behavior, and disease control programs.

247   Tick-borne diseases like Lyme disease and tick-borne encephalitis have also expanded with
248 climate warming, driven by warmer winters, northward expansions of the vector and reservoir host
249 species, and climate-driven changes in human and other animal behavior (Caminade et al. 2019;
250 Couper et al. 2021; Gilbert 2021). The complex tick life cycle, which requires a series of three blood

251   meals, often on different wild animal hosts that vary in competence for transmitting pathogens,
252   makes establishing direct links between climate change and tick-borne disease difficult (Couper et al.
253   2021; Ostfeld & Brunner 2015). Like other arthropods, ticks are sensitive to climate, particularly
254   temperature and humidity, which affect survival, behavior, and activity periods (Ostfeld & Brunner
255   2015). Temperature and precipitation affect tick species range limits (Hahn et al. 2016; Ostfeld &
256   Brunner 2015), and warming temperatures are implicated in the expansion of *Ixodes scapularis*
257   northward (Clow et al. 2017). Climate affects seed availability, reservoir host abundance, nymphal
258   tick abundance, and in turn Lyme disease risk in both Europe and North America (Bregnard et al.
259   2020; Ostfeld et al. 2006). Like other diseases with environmental transmission cycles, tick-borne
260   diseases are expanding in step with climate change, but the mechanisms can be complex and difficult
261   to quantify (but see Couper et al. 2021; Gilbert 2021).

262   **3.2   Land-use change and agricultural intensification**

263   Land-use change is a gradual process by which wild pristine habitat (or already human-modified
264   land) is altered by human activities, whereby the human-modified land serves a functional role, often
265   for economic purposes (Lambin & Meyfroidt 2011; Winkler et al. 2021). This process often involves
266   deforestation, road construction, dam building, irrigation, mining, urbanization, and clearing of land
267   for agriculture and livestock husbandry. Agricultural intensification may involve the expansion of
268   land-use change geographically, and/or increase the density of domestic animals locally. These
269   activities create edge habitats at the human–wildlife interface, increasing the potential for exchange
270   of pathogens between wildlife, domestic animals, and humans (Glidden et al. 2021; Nova 2021;
271   Plowright et al. 2021). This is exemplified by two viruses that emerged in the 1990s at the human–
272   wildlife interface facilitated by land-use change: Nipah and Hendra, which circulate within pteropid
273   fruit bats in Southeastern Asia and Australia, respectively (Daszak et al. 2006). Although their
274   ecology is different, the mechanisms by which these viruses emerged are similar, involving bats
275   visiting trees located near human settlements (Plowright et al. 2017; Sokolow et al. 2019).

276   In Bangladesh, indirect bat-to-human transmission has characterized the seasonal emergence
277   and spillover of Nipah virus since 2001. Local residents consume raw date palm sap from the silk
278   cotton and Indian mast trees, which is also contaminated by the opportunistic feeding of pteropid bats
279   (Hahn et al. 2014). These two trees flower during an otherwise barren season, potentially providing
280   an oasis of resources for bats (Hahn et al. 2014). Consequently, when the pteropid bats lick shaved
281   regions of the trees, leave urine or fecal matter in the sap collection pot, or die from falling into the
282   pots, they can promote bat-to-human, foodborne spillover (McKee et al. 2021). Bangladeshi villages
283   where spillover has occurred are characterized by forest fragmentation that promotes pteropid bats to
284   shift their roosting near consistent anthropogenic food sources (i.e., household gardens, agroforests)
285   (Faust et al. 2018; Hahn et al. 2014). The fragmentation dates back to the 17th century when
286   deforestation and rice cultivation were encouraged by Mughal rulers, and the British East India
287   Company furthered the fragmentation process through the Permanent Settlement system in the mid-
288   to-late 18th century (McKee et al. 2021). Thus, gradual modifications of the natural landscape rooted
289   in colonialism have confined pteropid bats' native habitat to smaller roost populations embedded
290   within a matrix of anthropogenic food resources (McKee et al. 2021).

291   In contrast, the spillover pathway may involve an intermediate host species, usually a
292   domestic animal, which bridges the spillover process between the reservoir bats and humans
293   (Plowright et al. 2017; Sokolow et al. 2019). Between the early 1970s and the late 1990s in
294   peninsular Malaysia, pig and mango production together experienced a rapid boom, nearly tripling in
295   output (Pulliam et al. 2012). This increased production was a result of agricultural intensification, in

296 which land was often simultaneously cultivated with both swine and fruit. In a pig farm in northern
297 Malaysia, hundreds of mango trees were planted adjacent to the swine enclosures, which attracted
298 pteropid fruit bats (Pulliam et al. 2012). A drip zone tends to form around these trees, where bats'
299 excretion of contaminated urine, fecal matter, and saliva (via partially-masticated fruit) tend to fall
300 (Plowright et al. 2015). This provided the means for the transmission of Nipah virus into the pigsties
301 (Daszak et al. 2006; Pulliam et al. 2012). Infected pigs served as amplifier hosts for the pathogen and
302 the subsequent transport of infected swine from the index farm fueled a large pig-to-human outbreak
303 in southern Malaysia and Singapore in 1998–1999 (Daszak et al. 2006).

304   Similarly, Hendra virus primarily spills over from bats to horses, and subsequent horse-to-
305 human transmission often leads to fatal cases. Land-use change, human population growth, and
306 shifting bat distributions are collectively expanding the area of overlap between bats and grazing
307 horses (Plowright et al. 2015). Agricultural and urban development and their byproducts—habitat
308 loss, fragmentation, and edge effects—serve to redistribute and restructure the availability and
309 quality of food resources in a given landscape (Faust et al. 2018). Urban and peri-urban areas provide
310 alternative—albeit lower quality—food sources for bats when ephemeral nectar in their native
311 flowering forests diminish due to seasonal conditions (Plowright et al. 2015). Particularly sensitive to
312 winter and spring food shortages, bats forgo migration in favor of residing near anthropogenic
313 sources of sustenance, such as fruiting trees planted in horse paddocks (Plowright et al. 2015).
314 Similarly to the Nipah example above, drip zones of bat excretions or discarded saliva-laden fruit
315 emerge around these trees (Plowright et al. 2015). Subsequently, the horses may contract Hendra by
316 coming into contact with these excretions either via grazing underneath or adjacent to bat-visited or
317 bat-roosting trees, or consuming contaminated bat-discarded fruit (Plowright et al. 2015). Thus, land-
318 use change, especially the emergence of urban and suburban areas, may drive the recent emergence
319 of Hendra (Figure 1c).

320   Another example of an emerging disease that spills over via a domestic intermediary host is
321 Influenza A. Its natural reservoir hosts include aquatic birds and waterfowl, such as ducks and geese
322 (Olsen et al. 2006). Intensive and extensive agricultural practices have been found to drive the
323 likelihood of spillover (Jones et al. 2013). Anthropogenic land use in wetland areas, where rice
324 paddies are coupled with free-gazing duck farming, for example, promotes the interspecific contact
325 between reservoir wild waterfowl and domestic water birds (Gilbert et al. 2007). As a result, the
326 newly-infected domestic water birds can pass on the pathogen to other domestic farm poultry through
327 the pathways of environmental contamination and direct contact (Sims et al. 2005). In other cases,
328 livestock intensification and anthropogenic compacting of farming systems has promoted high
329 densities of pigs that live in close proximity. As a result, influenza virus can spill over from birds to
330 pigs through interspecific contact, and the resulting swine influenza can be subsequently passed onto
331 humans (Ma et al. 2009). Pigs are capable of becoming infected with both avian and human influenza
332 strains. When two viral strains infect the same cell, genomic reassortment can occur, mixing genetic
333 elements of the two ancestral strains into a novel strain that can combine features of the human
334 influenza (i.e., the ability to readily infect and transmit among humans) with elements of the avian
335 strain (i.e., antigenic novelty that can lead to severe disease in immunologically naive people) (Ma et
336 al. 2009). Therefore, pigs can act as mixing vessels and amplifying hosts for onward human
337 transmission, especially when they are transported between farms—a similar mechanism to that of
338 Nipah virus in Malaysia, 1998–1999.

339   Another mechanism by which land-use change has facilitated disease emergence is by
340 providing suitable habitat for rodent disease reservoirs. For example, hantaviruses are a group of
341 viruses that primarily infect small rodents (e.g., mice, rats, voles), but can also occur in shrews and

342 moles. Though hantavirus infection was previously thought to be confined to Europe and Asia, since
343 its documented emergence in 1993–1994 in the southwestern United States (Hjelle & Torres-Pérez
344 2010), hantaviruses are now recognized worldwide in distinct geographic clusters: North and South
345 America (e.g., Sin Nombre virus, Choclo virus), Europe (e.g., Dobrava virus, Puumala virus) and
346 Asia (e.g., Seoul virus, Hantaan virus). Research on hantaviruses has revealed multiple pathways by
347 which land use change affects hantavirus spillover, often amplifying transmission. In China,
348 cultivation of wheat, corn, and other crops provides food for Hantaan virus reservoir rodent hosts and
349 increased rodent density (Yan et al. 2007). Likewise, orchard land and timber forest provide adequate
350 Hantaan virus reservoir habitats, while also facilitating higher contact between rodents and farmers or
351 forest workers via contaminated feces and urine (Yan et al. 2007). In certain North and South
352 American cases, cropland with food and cover can provide preferred habitats for hantavirus
353 reservoirs, and peri-domestic buildings (e.g., storage facilities, barns, homes) may concentrate rodent
354 urine and feces (Dearing & Dizney 2010). Similarly, peri-domestic microhabitats in the vicinity of
355 human habitation—patios, ornamental gardens, subsistence crops, livestock barns—can foster higher
356 seroprevalence of a species of Choclo virus-infected rodent (Armién et al. 2009). In Panama, forest
357 fragmentation and crop production have shifted small animal communities to become dominated by
358 rodents that are reservoirs of hantaviruses, leading to outbreaks in humans (Armien et al. 2004;
359 Suzán et al. 2008). Taken together, land use change does not uniformly affect hantavirus spillover.
360 Indeed, a variety of geographic and land use contexts modulate hantavirus emergence pathways in
361 different ways.

3.3 Urbanization

363 The rise of cities and peri-urban areas has led to the expansion of some infectious diseases through
364 multiple mechanisms (Gubler 2010; Weaver 2013). Urbanization increases the density of paved and
365 built areas, decreases species diversity and natural habitat availability, alters species composition,
366 warms the microclimate, increases human density and mobility, and changes social structures,
367 inequality, and behavior (Bharti et al. 2015; Cator et al. 2013; Cosner et al. 2009; Grimm et al. 2008).
368 The resulting changes in urban environments transform the landscape for infectious disease
369 transmission, favoring urban-adapted vectors and pathogens while decreasing the transmission
370 potential of sylvatic and rural species (LaDeau et al. 2015; Lambin et al. 2010; Weaver 2013).
371 However, the mechanisms by which urbanization affects vector-borne disease are complex and
372 multivariate, and the effects may differ between urban and peri-urban areas.

373 The mosquito-borne viruses transmitted by *Ae. aegypti*—dengue, chikungunya, Zika, and
374 others—are the canonical diseases of urbanization (Gubler 2010; Weaver 2013). *Ae. aegypti* is an
375 urban-dwelling mosquito that breeds in container habitats, rests indoors, preferentially bites humans
376 during the daytime, efficiently transmits many viral pathogens, thrives at warm temperatures, and has
377 become established in tropical, subtropical, and even temperate environments worldwide, expanding
378 from its native range in Africa (Gubler 2010; Weaver 2013). Multiple features of urban environments
379 promote *Ae. aegypti* arbovirus transmission. First, the built environment and socio-ecological
380 conditions promote vector habitat: informal or poor-quality housing without sealed or screened
381 windows and doors and reliable access to piped water and sanitation lead to the accumulation of
382 standing water either in storage containers, trash, or household items (Krystosik et al. 2020). Second,
383 these vectors have short flight ranges and rest indoors, resulting in increased biting rates on humans
384 (Stoddard et al. 2013). Third, population mobility is high within cities, among cities, and between
385 cities and rural areas, resulting in high rates of virus importation and movement of epidemics
386 between cities and the countryside (Chew et al. 2016; Cuong et al. 2013; Stoddard et al. 2013;
387 Wesolowski et al. 2015). Fourth, urban areas have warmer microclimates, including heat island

388 effects and warmer nights and winters, which can accelerate vector and pathogen development (Cator
389 et al. 2013; Murdock et al. 2017; Paaijmans et al. 2010), as well as underground structures that can
390 serve as climate refugia for vectors (Giordano et al. 2020; Lima et al. 2016). Fifth, vector control
391 programs, which were aggressive, coordinated, and often environmentally destructive and socially
392 oppressive but largely successful in the 1940s–60s lapsed in the 1970s, resulting in widespread
393 resurgence and expansion of *Ae. aegypti* populations (Gubler 2010). As a result of these and other
394 mechanisms acting in concert, dengue has increased exponentially since the 1970s (Gubler 2010;
395 Stanaway et al. 2016), followed by waves of invasion of chikungunya and Zika (also transmitted by
396 *Ae. aegypti*) through Latin America and the Caribbean, Oceania, and parts of Asia, Africa, North
397 America, and Europe (Dhimal et al. 2015; LaBeaud et al. 2015; Tomasello & Schlagenhauf 2013;
398 Weaver 2013, 2014; Weaver et al. 2016).

399   While *Aedes*-transmitted viruses are the canonical diseases of urbanization, other diseases and
400 vectors have more complex responses to urbanization. For example, malaria often declines in cities
401 compared to rural areas in sub-Saharan Africa, Southeast Asia, and South America as the vector's
402 outdoor breeding habitat and cooler optimal temperatures inhibit urban transmission, while greater
403 accessibility of medical care may help to break transmission chains, although peri-urban areas can
404 have substantial malaria transmission (Cator et al. 2013; Hay et al. 2005; Keiser et al. 2004). Tick-
405 borne diseases like Lyme disease, which require tick vectors to acquire pathogens from wild animal
406 hosts like rodents and small mammals, often respond positively to forest fragmentation and
407 suburbanization but decline in cities (Allan et al. 2003; Linske et al. 2018; Ostfeld et al. 2006). By
408 contrast, some *Culex* mosquitoes and zoonotic mosquito-borne pathogens like West Nile virus can be
409 present in cities, especially in urban parks, greenspaces, or abandoned buildings (Kilpatrick et al.
410 2010; LaDeau et al. 2015; Leisnham et al. 2014). Urbanization has had lagged effects on the
411 establishment of the triatomine vector and *Trypanosoma cruzi* parasite that cause Chagas disease in
412 Peru, where more established urban areas have higher abundances of infected vectors associated with
413 construction materials and domestic animals, while the vector was not yet established in newly
414 urbanizing areas (Levy et al. 2014). Natural disasters that disrupt urban infrastructure or lead to rapid,
415 unplanned urbanization can further increase vector-borne disease risks, such as the surge in Zika
416 transmission that occurred in Ecuador following the 2016 earthquake (Ali et al. 2017; Sorensen et al.
417 2017) and the increase in urban mosquito abundance following the 2010 earthquake in Haiti (Samson
418 et al. 2015). In sum, urbanization affects vector-borne diseases by modifying the physical
419 environment, biological diversity, microclimate, and human-vector interactions in ways that favor
420 some vectors and diseases while suppressing others (Figure 1c).

421 **3.4   Global movement of goods, humans, and other animals**

422 Global movement of goods, humans, and other animals via airline travel, international shipping, and
423 human migration is increasing in volume, speed, and extent. This movement can facilitate disease
424 emergence by creating conditions for spillover in the global animal trade, by rapidly turning recently
425 emerged pathogens into global pandemics, by reintroducing pathogens into areas where previously
426 eliminated, and by creating the conditions for disease transmission via the introduction of non-native
427 species, especially disease vectors (Bell et al. 2004; Findlater & Bogoch 2018; Lounibos 2002;
428 Tatem et al. 2006). While movement patterns not directly attributable to humans have also played a
429 role in disease emergence—for example, migratory patterns of birds helped to disperse West Nile
430 virus throughout the Americas after its emergence in New York (Swetnam et al. 2018)—here we
431 focus on anthropogenic-driven change in global movement.

432   Dengue and other *Aedes*-transmitted arboviruses are classic examples of both the transport of
433   goods introducing non-native species and importation of pathogens by travel of infected individuals
434   (Tatem et al. 2006). *Ae. aegypti* mosquitoes were originally limited to West Africa but were imported
435   to the Americas, Europe, and Oceania on sailing vessels associated with slave trade in the 16th or 17th
436   century, where they subsequently established and spread throughout the Americas (Lounibos 2002).
437   For the next 200 years, outbreaks of yellow fever would repeatedly occur, especially in port cities
438   from Havana to New Orleans to Philadelphia where the virus was regularly reintroduced.
439   Additionally, since the 1930s, *Ae. albopictus* mosquitoes have spread from their original range in
440   Asia to the Americas, Europe, and Africa, imported in used tires and other goods that can harbor
441   larvae and dormant eggs (Lounibos 2002; Tatem et al. 2006). The establishment of both *Ae. aegypti*
442   and *Ae. albopictus* set the stage for subsequent arbovirus epidemics, including dengue, chikungunya,
443   and Zika. The geographic distribution of dengue expanded in the 1980s and 1990s, most likely due to
444   introduction by travelers, from Southeast Asia to the Pacific Islands, Caribbean, and tropical parts of
445   the Americas and Africa, with importations in cooler regions of North America and Europe
446   occasionally leading to limited local transmission (Findlater & Bogoch 2018; Gubler 2010; Wilder-
447   Smith & Gubler 2008). Air travel has been implicated in circulation and dissemination of dengue
448   serotypes in Asia and Brazil (Nunes et al. 2014; Tian et al. 2017). Starting in 2013 and 2015
449   respectively, chikungunya and Zika followed similar patterns with rapid spread likely driven by air
450   travel causing outbreaks throughout the Pacific Islands, South and Central Americas, the Caribbean,
451   and parts of North America, Asia, Africa, and Europe (Gubler et al. 2017).

452   The global wildlife trade also drives the national and international transport of animals and
453   animal products. The legal and illegal wildlife trade combined is estimated to be up to a US$320
454   billion industry (Glidden et al. 2021). Wildlife hunting for trade and consumption has been linked to
455   spillover and spread of several emerging diseases, including SARS, Ebola, and monkeypox (Glidden
456   et al. 2021). There are two main mechanisms by which wildlife trade promotes zoonotic disease
457   emergence. First, trade brings together high densities of species (both disease reservoirs and non-
458   reservoirs), often kept under poor conditions during transport or in markets, facilitating both lower
459   immunity to infection and cross-species transmission (Glidden et al. 2021). These conditions favor
460   pathogen adaptation to new host species, including humans coming into contact with infected
461   animals at high densities. Second, the global wildlife trade is a major contributor to loss of
462   biodiversity, and primarily the depletion of large predators and herbivores due to high demand for
463   body parts (e.g., tiger bones and elephant ivory) (Baker et al. 2013). The removal of large-bodied
464   wildlife via global trade can have implications for disease emergence locally, as a decrease in large
465   animals has been correlated with an increase in small mammals brought to market, such as rodents
466   and bats, which are reservoirs for many zoonotic diseases (Glidden et al. 2021). Handling and
467   consumption of bat meat has been linked to human spillover of Ebola (Leroy et al. 2009) and may
468   have contributed to the 2013–2016 Ebola epidemic centered in West Africa.

469   For directly-transmitted diseases, global airline travel has also served to turn recently emerged
470   pathogens into international epidemics (Findlater & Bogoch 2018). Over the last 20 years,
471   coronaviruses have become an unfortunately common example of the speed with which global
472   human movement can spread novel pathogens around the world. In November 2002, severe acute
473   respiratory syndrome (SARS) caused by the coronavirus SARS-CoV-1 was first detected in China,
474   an emergence that has subsequently been linked to spillover from the likely reservoir in bats to the
475   intermediate host of civets and ultimately to humans—a chain of events that was likely facilitated by
476   the global wildlife trade (Bell et al. 2004; Glidden et al. 2021; Nova 2021). In the span of a year,
477   SARS was reported in nearly 30 countries, with over 8,000 reported cases and over 700 reported
478   deaths (World Health Organization 2004). A second novel coronavirus, MERS-CoV, emerged in

479 2012 in Saudi Arabia causing Middle East Respiratory Syndrome (MERS). MERS also emerged
480 from an animal reservoir (dromedary camels, with a potential distant origin in bats) before spreading
481 around the world via infected travelers, with 27 countries reporting cases of MERS since 2012 (Nova
482 2021; World Health Organization 2019). SARS, whose last human case was detected in May 2004,
483 MERS has caused repeated outbreaks since its detection in 2012, with the largest outbreak outside of
484 Saudi Arabia occurring in South Korea in 2015 when a single infected traveler returning to the
485 country ultimately resulted in 186 additional cases (World Health Organization 2019). Most recently,
486 COVID-19 was detected in 2019 in Wuhan, China caused by the virus SARS-CoV-2. At its onset,
487 the COVID-19 pandemic was fueled by international human movement, with early importations and
488 spread associated with volume of air travel between countries with ongoing transmission (Lau et al.
489 2020; Yang et al. 2020).

490   In addition to short-term air travel, human migration driven by economic collapse, political
491 instability, and climate change can become a source of disease importation and re-introduction. For
492 example, in Venezuela, economic and healthcare collapse in combination with land-use change
493 driven by deforestation and gold mining has led to a resurgence of malaria, with reported annual
494 malaria cases more than tripling from 2000 to 2015 (Grillet et al. 2019). The crisis in Venezuela has
495 also precipitated a mass migration with over 3.4 million Venezuelans fleeing to other countries,
496 which has led to increases in imported cases in parts of Colombia, Brazil, Ecuador, and Peru (Doocy
497 et al. 2019; Jaramillo-Ochoa et al. 2019; Rodríguez-Morales et al. 2019).

498 **4   Disentangling and responding to the complex relationships among economic forces, global**
499 **change and the burden of EIDs**

500 Economic development has nuanced and context-dependent impacts on global change: increases in
501 consumption of food (especially animal products) and material goods in general can be expected to
502 lead to increases in land needed for food production, and in resource extraction (e.g., gold mining)
503 (Jayachandran 2021). However, as living standards rise, people are able to prioritize the environment
504 without sacrificing basic needs or may be able to access opportunities with lower environmental
505 impacts (Jayachandran 2021). Thus, economic development may drive disease emergence via global
506 change, while simultaneously suppressing it via a change in perception of environmental health. In
507 addition, feedback may occur between EIDs and global change as a result of the EIDs impacts on the
508 economy. Here, we review the impacts of EIDs on the economy and management, and how EIDs
509 potentially feed back into altering global change.

510 **4.1   Economic impacts of EIDs and management**

511 The economic consequences of EIDs reach far beyond direct medical and public health costs, with
512 cascading effects across sectors (Smith et al. 2019). The tourism sector and travel industry are often
513 heavily impacted in places experiencing a disease outbreak or that are perceived as high risk, as
514 governments issue travel restrictions or advisories, or people curtail travel to avoid exposure. The
515 COVID-19 pandemic has affected the travel and tourism industry globally, the effects of which are
516 ongoing (Vanzetti David & Peters Ralf 2021). International tourism fell 74% between January and
517 December 2020, resulting in an estimated cost of US$2.4 trillion to tourism and associated sectors in
518 that year (Vanzetti David & Peters Ralf 2021). SARS, MERS, swine flu (H1N1) and Zika all have
519 had measurable effects on tourism nationally or regionally (Joo et al. 2019; Rassy & Smith 2013;
520 UNDP 2017; Wilder-Smith 2006).

521   In the case of zoonotic EIDs associated with livestock production, the agricultural sector can
522 be affected, particularly via culling of livestock—and the resulting direct and indirect costs. In the
523 case of the 1998 Nipah virus outbreak in Malaysia, in which pigs were identified as an intermediate
524 host, 1.1 million pigs were culled, which resulted in costs to the government that included
525 compensation to farmers and lost tax revenue (FAO & APHCA 2002), as well as costs to industries
526 associated with pig production such as feed suppliers (Hosono et al. 2006). An alternative
527 intervention was soon put into place: mango trees were mandated by policy to be planted at a
528 minimum distance from the pig enclosures (Pulliam et al. 2012). This simple, yet effective approach
529 incurred minimal cost, while preventing further Nipah virus outbreaks (Sokolow et al. 2019). It also
530 illustrates the benefits that can come from ecological interventions, actions that target the ecological
531 context in which spillover processes occur, as opposed to more conventional medical or veterinary
532 approaches such as vaccination and treatment (Sokolow et al. 2019). This provides an important
533 alternative approach to culling of badgers to prevent spillover of tuberculosis in cattle, of vampire
534 bats to prevent transmission of rabies to humans, and of mink infected with SARS-CoV-2 to prevent
535 transmission back to humans, all of which were controversial and had debatable impacts on disease
536 control (Enserink 2020; Sokolow et al. 2019).

537   Reductions in trade of animals or animal products, due either to government restrictions or
538 changes in consumer behavior, can also have substantial impacts. Following an introduction of Rift
539 Valley fever virus in Saudi Arabia and Yemen in 2000, Arabian countries banned imports of live
540 animals from a number of African countries (Peyre et al. 2015). The ban resulted in a 75% reduction
541 in animals exported from Somalia, which had previously made up 90% of the country's total income
542 (Peyre et al. 2015). Similarly, the association between H1N1 flu and pigs led to declines in Mexican
543 pork exports to the US and Japan, despite no official restrictions in trade (Rassy & Smith 2013).

544   The costs of EIDs may also include reduced health and diminished productivity over the long
545 term, even after the disease is brought under control (Bonds et al. 2010). For example, the long-term
546 economic costs of the 2015–2017 Zika epidemic in Latin America and the Caribbean associated with
547 microcephaly cases and Guillain-Barré syndrome cases are estimated at US$11 billion, on par with
548 the short-term costs (US$7–US$18 billion), which are dominated by lost tourism revenue and the
549 cost of diagnosing and treating Zika (UNDP 2017). Accounting for the full range of direct and
550 indirect economic costs associated with EIDs can help illuminate the benefits of investing in
551 simultaneously preventative and sustainable measures.

552 **4.2   The feedback of EIDs on global change**

553 In addition to the direct role of global change affecting EID risk, important feedback loops exist
554 between EIDs and aspects of global change, often mediated by the economic impacts of EIDs. For
555 example, annual global carbon emissions declined roughly by 7% in 2020 compared to 2019 as a
556 result of restrictions that were implemented to address the COVID-19 pandemic and reduced
557 economic activities (Friedlingstein et al. 2020). These reductions were not uniform around the world;
558 instead, countries like the US with more severe outbreaks experienced greater emissions reductions,
559 compared to countries that more successfully controlled COVID-19 (Tollefson 2021). In the
560 Brazilian Amazon, deforestation increases malaria through ecological mechanisms, but increasing
561 malaria in turn appears to reduce deforestation rates, likely through socioeconomic mechanisms
562 (MacDonald & Mordecai 2019). EIDs also have the potential to exacerbate global change. For
563 example, quarantine and travel restrictions associated with the 2013–2015 outbreak of Ebola in West
564 Africa prevented Environmental Protection Agency personnel in Sierra Leone from monitoring
565 protected areas to prevent illegal deforestation and mining, while economic instability as a result of

566 the epidemic was expected to increase pressure on natural resources (Government of Sierra Leone
567 2014).

568   In the face of such complexity, disentangling the relationships among global change, economic
569 drivers, and EIDs in a particular context is important for designing effective policies and
570 interventions to improve health and address global change. For example, the organization Health in
571 Harmony identified lack of access to healthcare as a driver of logging in and around a national park
572 in Borneo (Jennings et al. 2018). A program that increased local communities' access to healthcare
573 and lowered healthcare costs resulted in lower deforestation rates and increased visitation to health
574 clinics (Jones et al. 2020). In the United States, where more than one-third of forest lands are
575 privately held by individuals or families (USDA Forest Service 2008), a survey of landowners by the
576 Pinchot Institute identified healthcare costs as a major reason for logging or selling their lands, often
577 for development (Mater 2008). In response, the Forest Health-Human Health Initiative is piloting a
578 program that allows landowners to generate carbon credits for sustainable forest management in
579 return for payments for healthcare and investments in rural health services (Pinchot Institute 2012).
580 The relationship between deforestation and malaria in the Brazilian Amazon suggests that reducing
581 deforestation could both secure environmental benefits and reduce the burden of malaria, if
582 alternative sustainable livelihood opportunities can be supported (MacDonald & Mordecai 2019).

583 **5   Conclusions**

584 Global change is generated by human activities at a local and global scale. The activities that lead to
585 anthropogenic disturbances of the environment—primarily, climate change, land-use change,
586 urbanization, and global movement of humans, other organisms, and goods—affect societies and
587 ecosystems in ways that favor the emergence of novel infectious diseases in human populations,
588 expansions or shifts of diseases to new geographic regions, or re-emergence of diseases in various
589 places.

590   Here, we present some general mechanisms by which human-mediated disturbances of the
591 environment drive disease (re)emergence based on the evidence in the disease ecology literature.
592 First, climate change will most likely shift, and may already have shifted, the geographical
593 distribution of zoonotic and vector-borne diseases, although predicting those changes and the final
594 disease impacts on human populations remains a major challenge. Second, land-use change
595 frequently drives the emergence of novel diseases in humans, as it increases opportunities for
596 diseases to spill over from wildlife, especially from disease reservoirs like rodents and bats, to human
597 populations. Third, urbanization is expected to favor urban-adapted disease vectors and pathogens,
598 while suppressing diseases of sylvatic or rural origin, but the effects of urbanization differ drastically
599 between urban and peri-urban areas. Fourth, accumulating evidence suggests that global movement
600 of people, wild and domestic animals and plants, and goods has directly increased the emergence and
601 spread of novel and already established human diseases via travel, migration, and trade.

602   By exploring the ecological and socioeconomic mechanisms driving the relationship between
603 global change and EIDs, we are able to develop local and global ecological interventions that reduce
604 disease burden and prevent disease (re)emergence, complementing biomedical and public health
605 interventions. Because diseases respond to both ecological and socioeconomic conditions, solutions
606 require interdisciplinary systems approaches that engage with local communities. Further, the
607 economic impacts of EIDs go far beyond medical and public health costs, and thus there are multiple
608 ways in which disease emergence may feed back into the economy and global change via changes in
609 policies and resource management.

610　　　　In light of all this complexity, there is an urgent need for understanding interactions and
611　feedback between ecological and economic drivers of disease emergence. Such an understanding can
612　help to implement economic solutions that are not in isolation from disease ecology, and vice versa,
613　in order to develop more effective strategies for disease emergence prevention. Looking ahead,
614　interdisciplinary research that combines disease ecology and resource economics can help to identify
615　critical gaps in our understanding and prevention of EIDs. This agenda will promote a more
616　sustainable future where we can proactively mitigate planetary health crises.

617　**6　Acknowledgements**

628 **1.   Literature cited**

629 Adalja AA, Sell TK, Bouri N, Franco C. 2012. Lessons Learned during Dengue Outbreaks in the
630   United States, 2001–2011. *Emerg. Infect. Dis.* 18(4):608–14

631 Afrane YA, Githeko AK, Yan G. 2012. The Ecology of Anopheles Mosquitoes under Climate
632   Change: Case Studies from the Effects of Environmental Changes in East Africa Highlands.
633   *Ann. N. Y. Acad. Sci.* 1249:204–10

634 Ali S, Gugliemini O, Harber S, Harrison A, Houle L, et al. 2017. Environmental and Social Change
635   Drive the Explosive Emergence of Zika Virus in the Americas. *PLoS Negl. Trop. Dis.*
636   11(2):e0005135

637 Allan BF, Keesing F, Ostfeld RS. 2003. Effect of forest fragmentation on Lyme disease risk.
638   *Conserv. Biol.* 17(1):267–72

639 Altizer S, Ostfeld RS, Johnson PTJ, Kutz S, Harvell CD. 2013. Climate change and infectious
640   diseases: From evidence to a predictive framework. *Science*. 341(6145):514–519

641 Amraoui F, Failloux A-B. 2016. Chikungunya: an unexpected emergence in Europe. *Curr. Opin.*
642   *Virol.* 21:146–50

643 Anderson RM, May RM. 1979. Population biology of infectious diseases: Part I. *Nature*.
644   280(5721):361–67

645 Armién AG, Armién B, Koster F, Pascale JM, Avila M, et al. 2009. Hantavirus infection and habitat
646   associations among rodent populations in agroecosystems of Panama: implications for human
647   disease risk. *Am. J. Trop. Med. Hyg.* 81(1):59–66

648 Armien B, Pascale JM, Bayard V, Munoz C, Mosca I, et al. 2004. High seroprevalence of hantavirus
649   infection on the Azuero peninsula of Panama. *Am. J. Trop. Med. Hyg.* 70(6):682–87

650 Baker SE, Cain R, Kesteren F Van, Zommers ZA, D'Cruze N, MacDonald DW. 2013. Rough trade:
651   Animal welfare in the global wildlife trade. *Bioscience*. 63(12):928–38

652 Barnosky AD, Hadly EA, Bascompte J, Berlow EL, Brown JH, et al. 2012. Approaching a state shift
653   in Earth's biosphere. *Nature*. 486(7401):52–58

654 Becker AD, Grenfell BT. 2017. TSIR: An R package for time-series susceptible-infected-recovered
655   models of epidemics. *PLoS One*. 12(9):e0185528

656 Bell D, Roberton S, Hunter PR. 2004. Animal origins of SARS coronavirus: Possible links with the
657   international trade in small carnivores. *Philos. Trans. R. Soc. B Biol. Sci.* 359(1447):1107–14

658 Bharti N, Lu X, Bengtsson L, Wetter E, Tatem AJ. 2015. Remotely measuring populations during a
659   crisis by overlaying two data sources. *Int. Health*. 7(2):90–98

660 Bhatt S, Weiss DJ, Cameron E, Bisanzio D, Mappin B, et al. 2015. The effect of malaria control on
661   Plasmodium falciparum in Africa between 2000 and 2015. *Nature*. 526:207–11

662 Bonds MH, Keenan DC, Rohani P, Sachs JD. 2010. Poverty trap formed by the ecology of infectious

663   diseases. *Proc. R. Soc. B Biol. Sci.* 277(1685):1185–92

664 Bregnard C, Rais O, Voordouw MJ. 2020. Climate and tree seed production predict the abundance of
665   the European Lyme disease vector over a 15-year period. *Parasit. Vectors*. 13(1):408

666 Caldwell JM, LaBeaud AD, Lambin EF, Stewart-Ibarra AM, Ndenga BA, et al. 2021. Climate
667   predicts geographic and temporal variation in mosquito-borne disease dynamics on two
668   continents. *Nat. Commun.* 12(1):1233

669 Caminade C, McIntyre KM, Jones AE. 2019. Impact of recent and future climate change on vector-
670   borne diseases. *Ann. N. Y. Acad. Sci.* 1436(1):157–73

671 Cator LJ, Thomas S, Paaijmans KP, Ravishankaran S, Justin JA, et al. 2013. Characterizing
672   microclimate in urban malaria transmission settings: a case study from Chennai, India. *Malar. J.*
673   12(1):84

674 Chew CH, Woon YL, Amin F, Adnan TH, Abdul Wahab AH, et al. 2016. Rural-urban comparisons
675   of dengue seroprevalence in Malaysia. *BMC Public Health*. 16(1):824

676 Clow KM, Leighton PA, Ogden NH, Lindsay LR, Michel P, et al. 2017. Northward range expansion
677   of Ixodes scapularis evident over a short timescale in Ontario, Canada. *PLoS One*.
678   12(12):e0189393

679 Cosner C, Beier JC, Cantrell RS, Impoinvil D, Kapitanski L, et al. 2009. The effects of human
680   movement on the persistence of vector-borne diseases. *J. Theor. Biol.* 258(4):550–60

681 Couper LI, MacDonald AJ, Mordecai EA. 2021. Impact of prior and projected climate change on US
682   Lyme disease incidence. *Glob. Chang. Biol.* 27(4):738–54

683 Cuong HQ, Vu NT, Cazelles B, Boni MF, Thai KTD, et al. 2013. Spatiotemporal Dynamics of
684   Dengue Epidemics, Southern Vietnam. *Emerg. Infect. Dis.* 19(6):945–53

685 Daily GC, Ehrlich PR. 1996. Global change and human susceptibility to disease. *Annu. Rev. Energy*
686   *Environ.* 21(1):125–44

687 Daszak P, Plowright R, Epstein JH, Pulliam J, Abdul Rahman S, et al. 2006. The emergence of Nipah
688   and Hendra virus: pathogen dynamics across a wildlife-livestock-human continuum. *Dis. Ecol.*
689   *Community Struct. Pathog. Dyn.* 186–201

690 Dearing MD, Dizney L. 2010. Ecology of hantavirus in a changing world. *Ann. N. Y. Acad. Sci.*
691   1195:99–112

692 Dhimal M, Gautam I, Joshi HD, O'Hara RB, Ahrens B, Kuch U. 2015. Risk factors for the presence
693   of chikungunya and dengue vectors (*Aedes aegypti* and *Aedes albopictus*), their altitudinal
694   distribution and climatic determinants of their abundance in central Nepal. *PLoS Negl Trop Dis*.
695   9(3):e0003545

696 Dhimal M, Kramer IM, Phuyal P, Budhathoki SS, Hartke J, et al. 2021. Climate change and its
697   association with the expansion of vectors and vector-borne diseases in the Hindu Kush
698   Himalayan region: A systematic synthesis of the literature. *Adv. Clim. Chang. Res.* 12(3):421–

699   29

700   Doocy S, Page KR, de la Hoz F, Spiegel P, Beyrer C. 2019. Venezuelan Migration and the Border
701   Health Crisis in Colombia and Brazil. *J. Migr. Hum. Secur.* 7(3):79–91

702   Douine M, Lambert Y, Musset L, Hiwat H, Blume LR, et al. 2020. Malaria in Gold Miners in the
703   Guianas and the Amazon: Current Knowledge and Challenges. *Curr. Trop. Med. Reports.*
704   7(2):37–47

705   Enserink M. 2020. Coronavirus rips through Dutch mink farms, triggering culls. *Science.*
706   368(6496):1169.

707   FAO, APHCA. 2002. Manual on the Diagnosis of Nipah virus infection in animals. Bangkok,
708   Thailand. https://www.fao.org/3/ac449e/ac449e.pdf

709   Faria NR, Kraemer MUG, Hill SC, De Jesus JG, Aguiar RS, et al. 2018. Genomic and
710   epidemiological monitoring of yellow fever virus transmission potential. *Science.*
711   361(6405):894–99

712   Faust CL, McCallum HI, Bloomfield LSP, Gottdenker NL, Gillespie TR, et al. 2018. Pathogen
713   spillover during land conversion. *Ecol. Lett.* 21(4):471–83

714   Findlater A, Bogoch II. 2018. Human Mobility and the Global Spread of Infectious Diseases: A
715   Focus on Air Travel. *Trends Parasitol.* 34(9):772–83

716   Friedlingstein P, O'Sullivan M, Jones MW, Andrew RM, Hauck J, et al. 2020. Global Carbon Budget
717   2020. *Earth Syst. Sci. Data.* 12(4):3269–3340

718   Gérardin P, Guernier V, Perrau J, Fianu A, Le Roux K, et al. 2008. Estimating Chikungunya
719   prevalence in La Réunion Island outbreak by serosurveys: Two methods for two critical times of
720   the epidemic. *BMC Infect. Dis.* 8(1):99

721   Gething PW, Smith DL, Patil AP, Tatem AJ, Snow RW, Hay SI. 2010. Climate change and the
722   global malaria recession. *Nature.* 465(7296):342–45

723   Gilbert L. 2021. The Impacts of Climate Change on Ticks and Tick-Borne Disease Risk. *Annu. Rev.*
724   *Entomol.* 66(1):373–88

725   Gilbert M, Xiao X, Chaitaweesub P, Kalpravidh W, Premashthira S, et al. 2007. Avian influenza,
726   domestic ducks and rice agriculture in Thailand. *Agric. Ecosyst. Environ.* 119:409–15

727   Giordano B V, Gasparotto A, Liang P, Nelder MP, Russell C, Hunter FF. 2020. Discovery of an
728   *Aedes (Stegomyia) albopictus* population and first records of *Aedes (Stegomyia) aegypti* in
729   Canada. *Med. Vet. Entomol.* 34(1):10–16

730   Glidden CK, Nova N, Kain MP, Lagerstrom KM, Skinner EB, et al. 2021. Human-mediated impacts
731   on biodiversity and the consequences for zoonotic disease spillover. *Curr. Biol.* 31(19):R1342–
732   R1361

733   Government of Sierra Leone. 2014. The Economic and Social Impact of Ebola Virus Disease in
734   Sierra Leone.

735   https://reliefweb.int/sites/reliefweb.int/files/resources/Joint%20preliminary%20assessment%20s
736   ocio%20economic%20impact%20of%20EVD%20in%20Sierra%20Leone.pdf [Accessed
737   September 1, 2021]

738   Griffin R. 2009. *Indigenous knowledge for sustainable development: Case studies of three*
739   *indigenous tribes of Wisconsin*. University of Wisconsin-Stevens Point, College of Natural
740   Resources. https://epapers.uwsp.edu/thesis/2009/griffin.pdf [Accessed October 1, 2021]

741   Grillet ME, Hernández-Villena J V, Llewellyn MS, Paniz-Mondolfi AE, Tami A, et al. 2019.
742   Venezuela's humanitarian crisis, resurgence of vector-borne diseases, and implications for
743   spillover in the region. *Lancet Infect. Dis.* 19(5):e149–e161

744   Grimm NB, Foster D, Groffman P, Grove JM, Hopkinson CS, et al. 2008. The changing landscape:
745   ecosystem responses to urbanization and pollution across climatic and societal gradients. *Front.*
746   *Ecol. Environ.* 6(5):264–72

747   Gubler DJ. 2010. The Global Threat of Emergent/Re-emergent Vector-Borne Diseases. In *Vector*
748   *Biology, Ecology and Control*, pp. 39–62. Springer, Dordrecht

749   Gubler DJ, Vasilakis N, Musso D. 2017. History and Emergence of Zika Virus. *J. Infect. Dis.*
750   216(suppl_10):S860–S867

751   Hahn MB, Gurley ES, Epstein JH, Islam MS, Patz JA, et al. 2014. The role of landscape composition
752   and configuration on Pteropus giganteus roosting ecology and Nipah virus spillover risk in
753   Bangladesh. *Am. J. Trop. Med. Hyg.* 90(2):247–55

754   Hahn MB, Jarnevich CS, Monaghan AJ, Eisen RJ. 2016. Modeling the Geographic Distribution of
755   Ixodes scapularis and Ixodes pacificus (Acari: Ixodidae) in the Contiguous United States. *J.*
756   *Med. Entomol.* 53(5):1176–91

757   Hay SI, Guerra CA, Tatem AJ, Atkinson PM, Snow RW. 2005. Opinion — Tropical Infectious
758   Diseases: Urbanization, malaria transmission and disease burden in Africa. *Nat. Rev. Microbiol.*
759   3(1):81–90

760   Heesterbeek H, Anderson RM, Andreasen V, Bansal S, Angelis D De, et al. 2015. Modeling
761   infectious disease dynamics in the complex landscape of global health. *Science.* 347(6227):
762   aaa4339

763   Hjelle B, Torres-Pérez F. 2010. Hantaviruses in the Americas and their role as emerging pathogens.
764   *Viruses*. 2(12):2559–2586

765   Hosono H, Kono H, Ito S, Shirai J. 2006. Economic Impact of Nipah Virus Infection Outbreak in
766   Malaysia 1. *SciQuest*. (November 1998):1998–2000

767   Huber I, Potapova K, Ammosova E, Beyer W, Blagodatskiy S, et al. 2020. Symposium report:
768   emerging threats for human health – impact of socioeconomic and climate change on zoonotic
769   diseases in the Republic of Sakha (Yakutia), Russia. *Int. J. Circumpolar Health*. 79(1):1715698

770   Jaramillo-Ochoa R, Sippy R, Farrell DF, Cueva-Aponte C, Beltrán-Ayala E, et al. 2019. Effects of
771   Political Instability in Venezuela on Malaria Resurgence at Ecuador–Peru Border, 2018. *Emerg.*

772   *Infect. Dis.* 25(4): 834–836

773   Jayachandran S. 2021. How economic development influences the environment. NBER Working
774   Paper Series. http://www.nber.org/papers/w29191 [Accessed September 1, 2021]

775   Jennings J, Webb K, Minovi D. 2018. A community-based approach integrating conservation,
776   livelihoods, and health care in Indonesian Borneo. *Lancet Planet. Heal.* 2:S26

777   Jones BA, Grace D, Kock R, Alonso S, Rushton J, et al. 2013. Zoonosis emergence linked to
778   agricultural intensification and environmental change. *Proc. Natl. Acad. Sci. U. S. A.*
779   110(21):8399–8404

780   Jones IJ, MacDonald AJ, Hopkins SR, Lund AJ, Liu ZYC, et al. 2020. Improving rural health care
781   reduces illegal logging and conserves carbon in a tropical forest. *Proc. Natl. Acad. Sci. U. S. A.*
782   117(45):28515–24

783   Joo H, Maskery BA, Berro AD, Rotz LD, Lee YK, Brown CM. 2019. Economic Impact of the 2015
784   MERS Outbreak on the Republic of Korea's Tourism-Related Industries. *Heal. Secur.*
785   17(2):100–108

786   Keeling MJ, Rohani P. 2008. *Modeling Infectious Diseases in Humans and Animals*. Princeton
787   University Press

788   Keiser J, Utzinger J, Castro MC De, Smith TA, Tanner M, Singer BH. 2004. URBANIZATION IN
789   SUB-SAHARAN AFRICA AND IMPLICATION FOR MALARIA CONTROL. *Am. J. Trop.*
790   *Med. Hyg.* 71(2_suppl):118–27

791   Kilpatrick AM, Fonseca DM, Ebel GD, Reddy MR, Kramer LD. 2010. Spatial and Temporal
792   Variation in Vector Competence of Culex pipiens and Cx. restuans Mosquitoes for West Nile
793   Virus. *Am. J. Trop. Med. Hyg.* 83(3):607–13

794   King AA, Nguyen D, Ionides EL. 2016. Statistical inference for partially observed markov processes
795   via the R package pomp. *J. Stat. Softw.* 69:1–43

796   Koelle K, Pascual M. 2004. Disentangling Extrinsic from Intrinsic Factors in Disease Dynamics: A
797   Nonlinear Time Series Approach with an Application to Cholera. *Am. Nat.* 163(6):901–13

798   Krystosik A, Njoroge G, Odhiambo L, Forsyth JE, Mutuku F, LaBeaud AD. 2020. Solid Wastes
799   Provide Breeding Sites, Burrows, and Food for Biological Disease Vectors, and Urban Zoonotic
800   Reservoirs: A Call to Action for Solutions-Based Research. *Front. Public Heal.* 7:405

801   LaBeaud AD, Banda T, Brichard J, Muchiri EM, Mungai PL, et al. 2015. High Rates of O'Nyong
802   Nyong and Chikungunya Virus Transmission in Coastal Kenya. *PLoS Negl. Trop. Dis.*
803   9(2):e0003436

804   LaDeau SL, Allan BF, Leisnham PT, Levy MZ. 2015. The ecological foundations of transmission
805   potential and vector-borne disease in urban landscapes. *Funct. Ecol.* 29(7):889–901

806   Lafferty KD, Mordecai EA. 2016. The rise and fall of infectious disease in a warmer world.
807   *F1000Research*. 5:2040

808   Lambin EF, Meyfroidt P. 2011. Global land use change, economic globalization, and the looming
809   land scarcity. 108(9): 3465–3472

810   Lambin EF, Tran A, Vanwambeke SO, Linard C, Soti V. 2010. Pathogenic landscapes: Interactions
811   between land, people, disease vectors, and their animal hosts. *Int. J. Health Geogr.* 9(1):54

812   Lau H, Khosrawipour V, Kocbach P, Mikolajczyk A, Ichii H, et al. 2020. The association between
813   international and domestic air traffic and the coronavirus ({COVID}-19) outbreak. *J. Microbiol.*
814   *Immunol. Infect.* 53(3):467–72

815   Leisnham PT, LaDeau SL, Juliano SA. 2014. Spatial and Temporal Habitat Segregation of
816   Mosquitoes in Urban Florida. *PLoS One*. 9(3):e91655

817   Leroy EM, Epelboin A, Mondonge V, Pourrut X, Gonzalez J-P, et al. 2009. Human Ebola outbreak
818   resulting from direct exposure to fruit bats in Luebo, Democratic Republic of Congo, 2007.
819   *Vector Borne Zoonotic Dis.* 9(6):723–28

820   Levine JM. 2008. Biological invasions. *Current Biology*. 18(2): R57–R60

821   Levy MZ, Barbu CM, Castillo-Neyra R, Quispe-Machaca VR, Ancca-Juarez J, et al. 2014.
822   Urbanization, land tenure security and vector-borne Chagas disease. *Proc. R. Soc. B Biol. Sci.*
823   281(1789):20141003

824   Lima A, Lovin DD, Hickner P V, Severson DW. 2016. Evidence for an overwintering population of
825   *Aedes aegypti* in Capitol Hill neighborhood, Washington, DC. *Am. J. Trop. Med. Hyg.*
826   94(1):231–35

827   Linske MA, Williams SC, Stafford KC, Ortega IM. 2018. *Ixodes scapularis* (Acari: Ixodidae)
828   Reservoir Host Diversity and Abundance Impacts on Dilution of *Borrelia burgdorferi*
829   (Spirochaetales: Spirochaetaceae) in Residential and Woodland Habitats in Connecticut, United
830   States. *J. Med. Entomol.*

831   Lloyd-Smith JO, George D, Pepin KM, Pitzer VE, Pulliam JRC, et al. 2009. Epidemic Dynamics at
832   the Human-Animal Interface. *Science.* 326(5958):1362–67

833   Lloyd-Smith JO, Schreiber SJ, Kopp PE, Getz WM. 2005. Superspreading and the effect of
834   individual variation on disease emergence. *Nature*. 438(7066):355–59

835   Lounibos LP. 2002. Invasions by Insect Vectors of Human Disease. *Annu. Rev. Entomol.* 47(1):233–
836   66

837   Ma W, Kahn RE, Richt JA. 2009. The pig as a mixing vessel for influenza viruses: Human and
838   veterinary implications. *J. Mol. Genet. Med.* 03(01):158

839   MacDonald AJ, Mordecai EA. 2019. Amazon deforestation drives malaria transmission, and malaria
840   burden reduces forest clearing. *Proc. Natl. Acad. Sci.* 116(44):22212–18

841   Mater CM. 2008. *Wisconsin and Pennsylvania Forestland Owner Offspring Study Results for 2007-*
842   *2008*. Pinchot Institute. https://www.dropbox.com/s/pwfi5qxtih67wkc/Mater 2021_NASF
843   Offspring DC 2008.WI%26PA.pdf?dl=0 [Accessed September 1, 2021]

McKee CD, Islam A, Luby SP, Salje H, Hudson PJ, et al. 2021. The Ecology of Nipah Virus in Bangladesh: A Nexus of Land-Use Change and Opportunistic Feeding Behavior in Bats. *Viruses*. 13(2):169

Mordecai EA, Caldwell JM, Grossman MK, Lippi CA, Johnson LR, et al. 2019. Thermal biology of mosquito-borne disease. *Ecol. Lett.* 22(10):1690–1708

Mordecai EA, Ryan SJ, Caldwell JM, Shah MM, LaBeaud AD. 2020. Climate change could shift disease burden from malaria to arboviruses in Africa. *Lancet Planet. Heal.* 4(9):e416–e423

Murdock CC, Evans M V, McClanahan TD, Miazgowicz KL, Tesla B. 2017. Fine-scale variation in microclimate across an urban landscape shapes variation in mosquito population dynamics and the potential of *Aedes albopictus* to transmit arboviral disease. *PLoS Negl. Trop. Dis.* 11(5):e0005640

Myers SS. 2017. Planetary health: protecting human health on a rapidly changing planet. *Lancet.* 390(10114):2860–68

Nova N. 2021. Cross-species transmission of coronaviruses in humans and domestic mammals, what are the ecological mechanisms driving transmission, spillover, and disease emergence? *Front. Public Heal.* 9:717941

Nova N, Deyle ER, Shocket MS, MacDonald AJ, Childs ML, et al. 2021. Susceptible host availability modulates climate effects on dengue dynamics. *Ecol. Lett.* 24(3):415–425

Nunes MRT, Palacios G, Faria NR, Sousa EC, Pantoja JA, et al. 2014. Air Travel Is Associated with Intracontinental Spread of Dengue Virus Serotypes 1–3 in Brazil. *PLoS Negl. Trop. Dis.* 8(4):e2769

Olsen B, Munster VJ, Wallensten A, Waldenström J, Osterhaus ADME, Fouchier RAM. 2006. Global patterns of influenza a virus in wild birds. *Science.* 312(5772):384–88

Ostfeld RS, Brunner JL. 2015. Climate change and Ixodes tick-borne diseases of humans. *Philos. Trans. R. Soc. B Biol. Sci.* 370(1665):20140051

Ostfeld RS, Canham CD, Oggenfuss K, Winchcombe RJ, Keesing F. 2006. Climate, Deer, Rodents, and Acorns as Determinants of Variation in Lyme-Disease Risk. *PLOS Biol.* 4(6):e145

Paaijmans KP, Imbahale SS, Thomas MB, Takken W. 2010. Relevant microclimate for determining the development rate of malaria mosquitoes and possible implications of climate change. *Malar. J.* 9(1):196

Parmesan C, Burrows MT, Duarte CM, Poloczanska ES, Richardson AJ, et al. 2013. Beyond climate change attribution in conservation and ecological research. *Ecol. Lett.* 16(s1):58–71

Pauchard A, Milbau A, Albihn A, Alexander J, Burgess T, et al. 2016. Non-native and native organisms moving into high elevation and high latitude ecosystems in an era of climate change: new challenges for ecology and conservation. *Biol. Invasions*. 2(18):345–53

Paull SH, Horton DE, Ashfaq M, Rastogi D, Kramer LD, et al. 2017. Drought and immunity

880   determine the intensity of west nile virus epidemics and climate change impacts. *Proc. R. Soc. B*
881   *Biol. Sci.* 284(1848):20162078

882   Pearl J. 2010. An introduction to causal inference. *International Journal of Biostatistics*. 6(2):1–59

883   Peyre M, Chevalier V, Abdo-Salem S, Velthuis A, Antoine-Moussiaux N, et al. 2015. A Systematic
884   Scoping Study of the Socio-Economic Impact of Rift Valley Fever: Research Gaps and Needs.
885   *Zoonoses Public Health*. 62(5):309–25

886   Pinchot Institute. 2012. The Forest Health-Human Health (FHHH) Initiative.
887   https://americancarbonregistry.org/resources/forest-health-human-health [Accessed September
888   1, 2021]

889   Plowright RK, Eby P, Hudson PJ, Smith IL, Westcott D, et al. 2015. Ecological dynamics of
890   emerging bat virus spillover. *Proc. R. Soc. B Biol. Sci.* 282(1798):20142124

891   Plowright RK, Parrish CR, McCallum H, Hudson PJ, Ko AI, et al. 2017. Pathways to zoonotic
892   spillover. *Nat. Rev. Microbiol.* 15(8):502–10

893   Plowright RK, Reaser JK, Locke H, Woodley SJ, Patz JA, et al. 2021. Land use-induced spillover: a
894   call to action to safeguard environmental, animal, and human health. *Lancet Planet. Heal.*
895   5(4):e237–45

896   Pulliam JRCC, Epstein JH, Dushoff J, Rahman SA, Bunning M, et al. 2012. Agricultural
897   intensification, priming for persistence and the emergence of Nipah virus: A lethal bat-borne
898   zoonosis. *J. R. Soc. Interface*. 9(66):89–101

899   Rassy D, Smith RD. 2013. The economic impact of H1N1 on Mexico's tourist and pork sectors.
900   *Health Econ.* 22(7):824–34

901   Rodríguez-Morales AJ, Suárez JA, Risquez A, Villamil-Gómez WE, Paniz-Mondolfi A. 2019.
902   Consequences of Venezuela's massive migration crisis on imported malaria in Colombia, 2016–
903   2018. *Travel Med. Infect. Dis.* 28:98–99

904   Ryan SJ, Carlson CJ, Mordecai EA, Johnson LR. 2019. Global expansion and redistribution of
905   *Aedes*-borne virus transmission risk with climate change. *PLoS Negl. Trop. Dis.* 13(3):e0007213

906   Ryan SJ, Carlson CJ, Tesla B, Bonds MH, Ngonghala CN, et al. 2021. Warming temperatures could
907   expose more than 1.3 billion new people to Zika virus risk by 2050. *Glob. Chang. Biol.*
908   27(1):84–93

909   Sachal S, Deilgat M, Speechley M. 2019. Case 11 : Crypto Climate Creep: The Movement of
910   Tropical Infectious Disease to the Arctic. *West. Public Heal. Casebooks*. 2019(2019):

911   Samson DM, Archer RS, Alimi TO, Arheart KL, Impoinvil DE, et al. 2015. New baseline
912   environmental assessment of mosquito ecology in northern Haiti during increased urbanization.
913   *J. Vector Ecol.* 40(1):46–58

914   Shocket MS, Anderson CB, Caldwell JM, Childs ML, Couper LI, et al. 2021. Environmental drivers
915   of vector-borne diseases. In *Population Biology of Vector-Borne Diseases*, pp. 85–118. Oxford

916   University Press

917   Shocket MS, Verwillow AB, Numazu MG, Slamani H, Cohen JM, et al. 2020. Transmission of West
918   Nile virus and other temperate mosquito-borne viruses occurs at lower environmental
919   temperatures than tropical mosquito-borne diseases. *Elife*. 9:e58511

920   Sims LD, Domenech J, Benigno C, Kahn S, Kamata A, et al. 2005. Origin and evolution of highly
921   pathogenic H5N1 avian influenza in Asia. *Vet. Rec.* 157(6):159–64

922   Siraj AS, Santos-Vega M, Bouma MJ, Yadeta D, Ruiz Carrascal D, Pascual M. 2014. Altitudinal
923   changes in malaria incidence in highlands of Ethiopia and Colombia. *Science*. 343(6175):1154–
924   58

925   Smith DL, Cohen JM, Chiyaka C, Johnston G, Gething PW, et al. 2013. A sticky situation: the
926   unexpected stability of malaria elimination. *Philos. Trans. R. Soc. B Biol. Sci.*
927   368(1623):20120145

928   Smith KM, Machalaba CC, Seifman R, Feferholtz Y, Karesh WB. 2019. Infectious disease and
929   economics: The case for considering multi-sectoral impacts. *One Heal.* 7(June 2018):100080

930   Sokolow SH, Nova N, Pepin KM, Peel AJ, Manlove K, et al. 2019. Ecological interventions to
931   prevent and manage zoonotic pathogen spillover. *Philos. Trans. R. Soc. B Biol. Sci.*
932   374(1782):20180342

933   Sorensen CJ, Borbor-Cordova MJ, Calvello-Hynes E, Diaz A, Lemery J, Stewart-Ibarra AM. 2017.
934   Climate Variability, Vulnerability, and Natural Disasters: A Case Study of Zika Virus in
935   Manabi, Ecuador Following the 2016 Earthquake. *GeoHealth*. 1(8):298–304

936   Stanaway JD, Shepard DS, Undurraga EA, Halasa YA, Coffeng LE, et al. 2016. The global burden of
937   dengue: an analysis from the Global Burden of Disease Study 2013. *Lancet Infect. Dis.*
938   16(6):712–23

939   Stewart-Ibarra AM, Lowe R. 2013. Climate and non-climate drivers of dengue epidemics in southern
940   coastal Ecuador. *Am. J. Trop. Med. Hyg.* 88(5):971–81

941   Stoddard ST, Forshey BM, Morrison AC, Paz-Soldan VA, Vazquez-Prokopec GM, et al. 2013.
942   House-to-house human movement drives dengue virus transmission. *Proc. Natl. Acad. Sci.*
943   110(3):994–99

944   Sugihara G, May R, Ye H, Hsieh C, Deyle E, et al. 2012. Detecting Causality in Complex
945   Ecosystems. *Science*. 338(6106):496–500

946   Suzán G, Marcé E, Giermakowski JT, Armién B, Pascale J, et al. 2008. The effect of habitat
947   fragmentation and species diversity loss on hantavirus prevalence in Panama. *Ann. N. Y. Acad.*
948   *Sci.* 1149:80–83

949   Swetnam D, Widen SG, Wood TG, Reyna M, Wilkerson L, et al. 2018. Terrestrial Bird Migration
950   and West Nile Virus Circulation, United States. *Emerg. Infect. Dis.* 24(12):2184–94

951   Tatem AJ, Rogers DJ, Hay SI. 2006. Global Transport Networks and Infectious Disease Spread. In

952   *Advances in Parasitology*. 62:293–343.

953   Tian H, Sun Z, Faria NR, Yang J, Cazelles B, et al. 2017. Increasing airline travel may facilitate co-
954   circulation of multiple dengue virus serotypes in Asia. *PLoS Negl. Trop. Dis.* 11(8):e0005694

955   Tollefson J. 2021. COVID curbed carbon emissions in 2020 - but not by much. *Nature*.
956   589(7842):343

957   Tomasello D, Schlagenhauf P. 2013. Chikungunya and dengue autochthonous cases in Europe,
958   2007–2012. *Travel Med. Infect. Dis.* 11(5):274–84

959   UNDP. 2017. A Socio-economic Impact Assessment of the Zika Virus in Latin America and the
960   Caribbean. http://www.undp.org/content/undp/en/home/librarypage/hiv-aids/a-socio-economic-
961   impact-assessment-of-the-zika-virus-in-latin-am.html [Accessed September 1, 2021]

962   USDA Forest Service. 2008. Who Owns America's Forests? Forest Ownership Patterns and Family
963   Forest Highlights from the National Woodland Owner Survey.
964   https://www.nrs.fs.fed.us/pubs/inf/NRS-INF-06-08.pdf [Accessed September 1, 2021]

965   Vanzetti David, Peters Ralf. 2021. Covid-19 and Tourism an Update.
966   https://unctad.org/system/files/official-document/ditcinf2021d3_en_0.pdf [Accessed October 1,
967   2021]

968   Volz EM, Kosakovsky Pond SL, Ward MJ, Leigh Brown AJ, Frost SDW. 2009. Phylodynamics of
969   infectious disease epidemics. *Genetics*. 183(4):1421–30

970   Weaver SC. 2013. Urbanization and geographic expansion of zoonotic arboviral diseases:
971   mechanisms and potential strategies for prevention. *Trends Microbiol.* 21(8):360–63

972   Weaver SC. 2014. Arrival of Chikungunya Virus in the New World: Prospects for Spread and Impact
973   on Public Health. *PLoS Negl Trop Dis*. 8(6):e2921

974   Weaver SC, Costa F, Garcia-Blanco MA, Ko AI, Ribeiro GS, et al. 2016. Zika virus: History,
975   emergence, biology, and prospects for control. *Antiviral Res.* 130:69–80

976   Weitz JS, Park SW, Eksin C, Dushoff J. 2020. Awareness-driven behavior changes can shift the
977   shape of epidemics away from peaks and toward plateaus, shoulders, and oscillations. *Proc.*
978   *Natl. Acad. Sci.* 117(51):32764–71

979   Wesolowski A, Qureshi T, Boni MF, Sundsøy PR, Johansson MA, et al. 2015. Impact of human
980   mobility on the emergence of dengue epidemics in Pakistan. *Proc. Natl. Acad. Sci.*
981   112(38):11887–92

982   Wilder-Smith A. 2006. The severe acute respiratory syndrome: Impact on travel and tourism. *Travel*
983   *Med. Infect. Dis.* 4(2):53–60

984   Wilder-Smith A, Gubler DJ. 2008. Geographic Expansion of Dengue: The Impact of International
985   Travel. *Med. Clin. North Am.* 92(6):1377–90

986   Winkler K, Fuchs R, Rounsevell M, Herold M. 2021. Global land use changes are four times greater
987   than previously estimated. *Nat. Commun.* 12(1):1–10

988   Woolhouse MEJ, Gowtage-Sequeria S. 2005. Host range and emerging and reemerging pathogens.
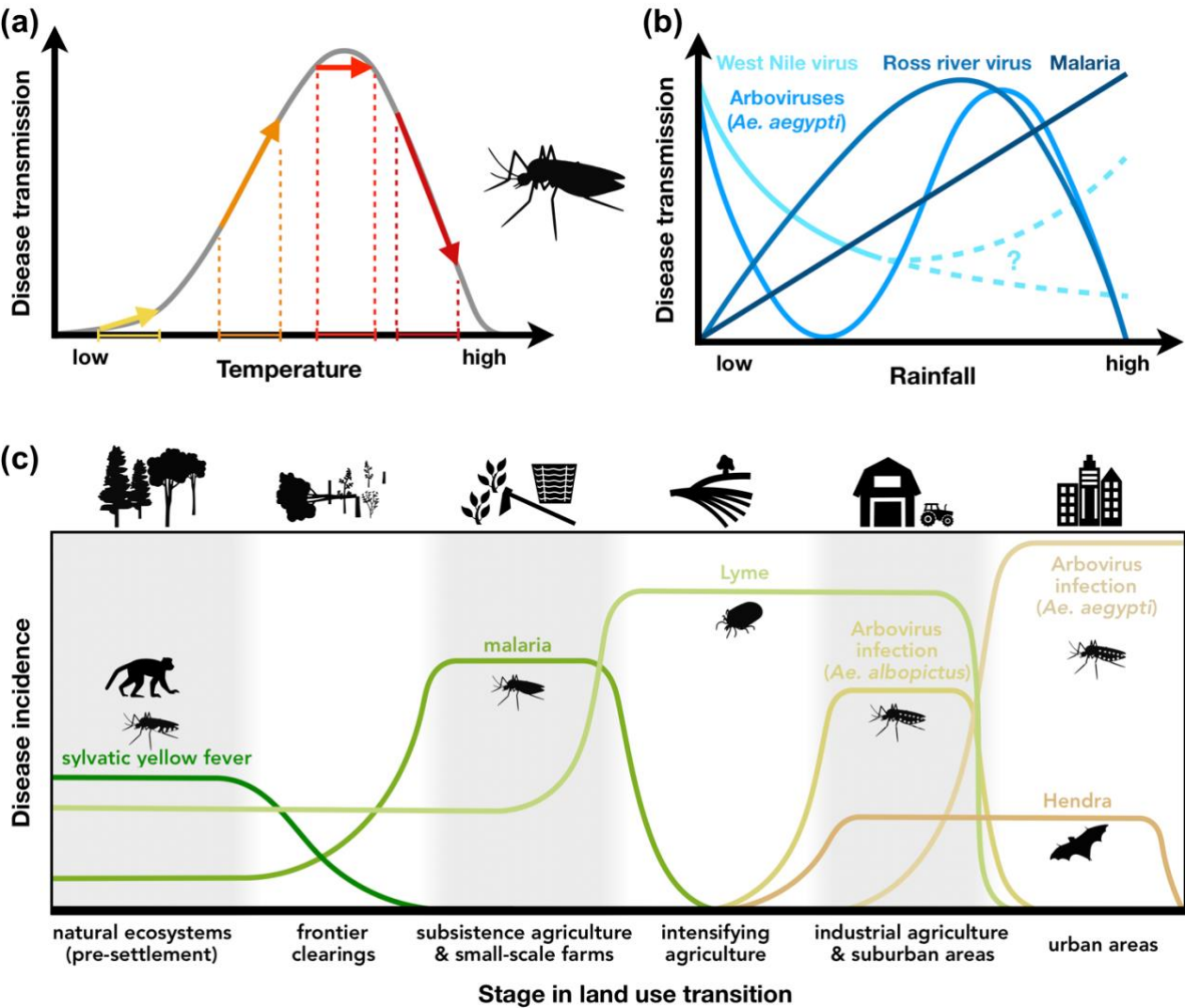989   *Emerg. Infect. Dis.* 11(12):1842–47

990   World Health Organization. 2004. Summary of probable SARS cases with onset of illness from 1
991   November 2002 to 31 July 2003. https://www.who.int/publications/m/item/summary-of-
992   probable-sars-cases-with-onset-of-illness-from-1-november-2002-to-31-july-2003 [Accessed
993   October 1, 2021]

994   World Health Organization. 2019. WHO MERS global summary and assessment of risk, July 2019.
995   https://www.who.int/publications-detail-redirect/10665-326126 [Accessed October 1, 2021]

996   Yan L, Fang L-Q, Huang H-G, Zhang L-Q, Feng D, et al. 2007. Landscape elements and Hantaan
997   virus-related hemorrhagic fever with renal syndrome, People's Republic of China. *Emerg.*
998   *Infect. Dis.* 13(9):1301–6

999   Yang J, Li J, Lai S, Ruktanonchai CW, Xing W, et al. 2020. Uncovering two phases of early
1000   intercontinental COVID-19 transmission dynamics. *J. Travel Med.* 27(8):taaa200

1001

Figure

1004 **Figure 1. Impacts of global change on emerging infectious diseases can be nonlinear, complex,**
1005 **and exhibit threshold effects.** *(a)* Global changes that affect temperature, such as climate change,
1006 land-use change, and urbanization, may have nonlinear effects on disease transmission. For many
1007 mosquito-borne diseases, accumulating evidence suggests a hump-shaped relationship between
1008 temperature and disease transmission. Thus, a given temperature increase can have different effects
1009 on disease transmission in different contexts depending on the baseline, causing a small increase
1010 (yellow arrow), a large increase (orange arrow), no change (red arrow), or a decrease (dark red
1011 arrow) in disease transmission. *(b)* The effects of rainfall on mosquito-borne disease transmission are
1012 more complex, since the rainfall–transmission relationship (linear or nonlinear) depends on vector
1013 ecology and factors related to human behavior and the built environment, or largely remains
1014 unresolved (dashed lines). *(c)* Global changes that alter the landscape and habitat, such as land-use
1015 change and urbanization, may promote or suppress transmission of emerging diseases depending on
1016 the disease and its ecology. Here are some examples of how different vector-borne or zoonotic
1017 emerging diseases are expected to vary across a land-use gradient. *Ae.* stands for *Aedes*, and
1018 arboviruses are *Aedes*-borne viruses (e.g., dengue, Zika, and yellow fever). Figure adapted from
1019 (Shocket et al. 2021).