Global Change Biology

WILEY

Global Change Biology

RESEARCH ARTICLE OPEN ACCESS

Zoonotic Host Richness in the Global Wildland–Urban Interface

Rohan D. Simkin[1] | Barbara A. Han[2] | Volker C. Radeloff[3] | Shannon LaDeau[2] | Franz Schug[3] | Karen C. Seto[1]

[1]Yale School of the Environment, Yale University, New Haven, Connecticut, USA | [2]Cary Institute of Ecosystem Studies, Millbrook, New York, USA | [3]SILVIS Lab, Department of Forest and Wildlife Ecology, University of Wisconsin-Madison, Madison, Wisconsin, USA

Correspondence: Rohan D. Simkin (rohan.simkin@yale.edu)

Received: 26 July 2024 | **Revised:** 4 November 2024 | **Accepted:** 5 November 2024

Funding: This work was funded by NASA LCLUC MuSLI grant 80NSSC21K0310; NASA IDS grant 80NSSC24K0303; NASA grant 22-SMDSS22-0042; and US Department of Agriculture McIntire Stennis award WIS03072.

Keywords: disease ecology | land-use change | spillover | urbanization | wildland–urban interface | zoonotic disease

ABSTRACT

Where human settlements abut or intermix with wildlands, people may encounter animals that host zoonotic pathogens which can spillover to cause human disease. Known as the wildland–urban interface (WUI), this zone occupies around 5% of the Earth's surface and is home to 3.5 billion people. The rapid spread of SARS-CoV-2 has demonstrated the importance of understanding risk factors for disease among an increasingly urbanized population. However, the contribution of the WUI to zoonotic disease risk is poorly understood. Here, we show that low-level host richness occurs throughout most of the global WUI, and 20% of the human WUI population live in zones of particularly high zoonotic potential, where more than 20 host species could occur. Zones of high zoonotic potential are concentrated in low–middle-income countries (LMICs) across equatorial Africa, Brazil, Central America, and Southeast Asia where vulnerability is further elevated by widespread poverty, inadequate housing, and lack of easily accessible healthcare. Three of four people living in WUIs with high host richness (520 million people) are in LMICs. Of this population, 35% (183 million) live in and around cities in West, East, and South Africa. This means that WUI-based populations of LMICs may face the double threat of high zoonotic potential and vulnerability to disease. Our results identify global priorities for monitoring exposure to zoonotic diseases in the rapidly expanding WUI.

1 | Introduction

In the wildland–urban interface (WUI), buildings meet or intermingle with wildland vegetation. The WUI is defined as a zone that straddles the interface between urban and wildland, encapsulating areas of both (Radeloff et al. 2005; USDA 2001). The WUI is prevalent globally, covering roughly 5% of the land surface (Schug et al. 2023). It is where many human–environmental conflicts are concentrated (Bar-Massada, Radeloff, and Stewart 2014; Radeloff et al. 2018), and where people and buildings are most at risk from wildfires (Bento-Gonçalves and Vieira 2020).

The WUI is also a setting that creates opportunities for human contact with wildlife, including hosts of zoonotic diseases, which could lead to spillover transmission into human populations (Despommier, Ellis, and Wilcox 2006; Plowright et al. 2021). Human–wildlife contact in the WUI may occur when people enter wildland areas that provide habitat for zoonotic hosts while hunting, collecting firewood, harvesting timber, or clearing land for development (Faria et al. 2018). Contact may also occur where synanthropic species (those with a propensity to live near human dwellings) found in nearby natural habitats become established and proliferate in urban and agricultural areas (Ecke et al. 2022).

Contact between humans and zoonotic hosts can result in spillover via multiple pathways, including through direct contact with infected hosts, transmission from infected host to humans by insect vectors, ingestion of contaminated animal products, or exposure to environmentally persistent pathogens that are shed by infected hosts (Box 1) (Plowright et al. 2017). The risk of spillover may be exacerbated by livestock farming in the WUI (Connolly, Keil, and Ali 2020; Hassell et al. 2017). Domestic animals such as cattle, sheep, goats, and pigs are common intermediate hosts for wildlife zoonoses that are passed on to farm workers (Hassell et al. 2017; Shah et al. 2019).

The potential for zoonotic disease outbreaks is enhanced where significant concentrations of poverty are found in urban and periurban places. Cities in low- and middle-income countries (LMICs) are particularly vulnerable. Over 1 billion people globally live in slums and informal settlements—including more than 50% of the urban population of sub-Saharan Africa (UN-Habitat 2022).

BOX 1 | Spillover pathways at the wildland–urban interface.

Environmental:
Pathogens are shed by hosts into the environment, such as from urine or feces. Human spillover infection may occur through ingestion of the pathogen via contaminated water, food containing contaminated soil, or directly through the skin. In the WUI, environmental transmission may be particularly prevalent in informal settlements where sanitation is lacking, resulting in frequent contact with contaminated soil and water sources. Diseases in this category include soil-transmitted helminths (e.g., *Ascariasis*) and leptospirosis, which inflict a large burden across the globe, particularly in poor communities and informal settlements in the tropics and subtropics (Bethony et al. 2006; McCarthy and Moore 2000).

Vector borne:
Pathogens are transmitted via the bite of an infected blood-feeding insect that has previously fed on a competent non-human host. Vector-borne disease can be maintained in sylvatic cycles, and spillover into urban populations where hosts are present in the WUI, such as for yellow fever or Japanese encephalitis virus. Urban-adapted arthropods, such as *Aedes aegypti*, are responsible for rising rates of both sylvatic and urban viral outbreaks globally, including dengue fever (Chala and Hamde 2021).

Direct contact:
Spillover via direct contact between host and human may occur in the WUI during slaughter of wild animals (e.g., Ebola virus), or via a bite or scratch from an infected animal (rabies virus) (Hikufe et al. 2019; Lawson et al. 2019).

Animal product ingestion:
Ingestion of meat or other animal products can lead to spillover transmission in the WUI for pathogens such as mpox virus, where hunting takes place in the WUI and bushmeat is sold in urban markets (Islam et al. 2023). Spillover from wild animals in the WUI may also occur via ingestion of meat or dairy from domestic animals that contracted a disease through vectors (Crimean Congo hemorrhagic fever (Spengler, Bergeron, and Rollin 2016)) or direct contact with wildlife hosts (e.g., salmonellosis (Espinosa, Tago, and Treich 2020)).

Concentrations of disadvantage heighten the vulnerability of urban populations to infectious diseases where poor housing, lack of sanitation and overcrowding provide suitable conditions for infectious diseases to spread (Alirol et al. 2011; Levy et al. 2014).

The global WUI is home to approximately 3.5 billion people (Schug et al. 2023), which means that a large proportion of the global population (44%) could potentially be exposed to pathogens. However, the risk associated with exposure to zoonotic diseases in the WUI remains poorly understood. Prior research on urban zoonoses focuses on spillover *within* cities where urban-adapted wildlife species are the zoonotic reservoir or amplifying host (Albery et al. 2022; Bradley and Altizer 2007). However, urban populations do not exist in isolation from their surroundings, and the presence of hosts in surrounding wildlands may be an important risk factor for spillover, in addition to the conditions of the urban centers themselves. Given the ramifications that zoonotic diseases can have for humanity, as highlighted by the COVID-19 pandemic, there is need to understand the risk factors involved in the epidemiology of urban zoonotic diseases in the WUI.

Mounting concern about how to understand and mitigate spillover transmission at these key interfaces has led to important conceptual advances. For example, Hassell et al. (2017) demonstrate that complex ecological interactions among humans, wildlife, and livestock in urban and periurban areas are important drivers of disease spillover. The risk of zoonotic spillover associated with urban land encroaching upon wildlands is discussed in a broader review of urbanization as a driver of disease emergence by Neiderud (2015) and as a contributor to disease emergence driven by land-use change in Asia by Goldstein et al. (2022). However, quantitative investigations about spillover risk at WUIs have remained limited.

Our goal here was to quantify zoonotic host species richness in the WUI as a means of understanding zoonotic disease potential. The host species richness is indicative of pathogen richness and the diversity of interaction pathways with humans. This makes host species richness a key biophysical risk factor which, along with other biophysical and socioeconomic risk factors, determine the risk of spillover occurring. By quantifying zoonotic host richness, we aim to contribute to the broader understanding of zoonotic disease risk in the WUI.

To do this we conducted a global analysis of mammal host richness in the WUI that accounts for individual species habitat preferences. We focused on mammalian hosts because the majority of zoonoses that are emerging in humans persist in mammals (Han, Kramer, and Drake 2016). We identified places where large concentrations of human populations in the WUI overlap with high zoonotic potential. We compare zoonotic potential in high-income countries (HICs) and LMICs in order to highlight those that face the combined threat of high zoonotic potential and vulnerability to disease (World Bank 2023).

2 | Results

2.1 | Zoonotic Potential in the Global WUI

We quantified global zoonotic potential by modeling the distribution of 686 terrestrial mammal species associated with 144

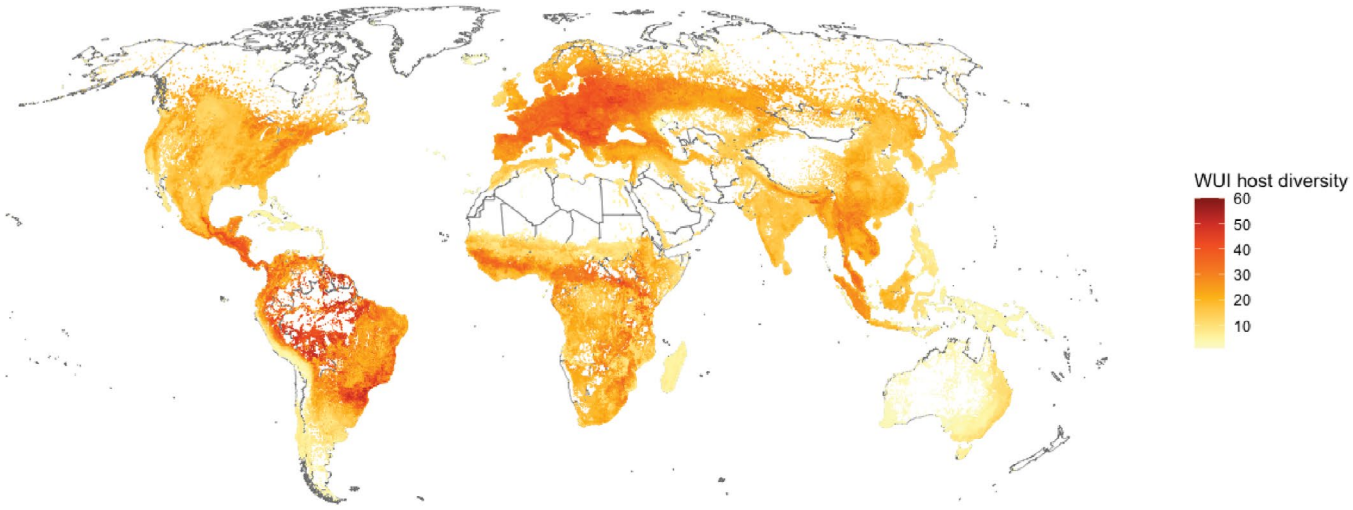

FIGURE 1 | Zoonotic mammalian hosts whose geographic ranges and habitat preferences overlap the wildland–urban interface (WUI) is widespread globally. Color scale represents the total number of host species with habitat in the WUI. Host richness is calculated as the mean host richness in a 10-km-resolution global grid. Host richness in the WUI follows global patterns of species richness, with hotspots occurring in urbanizing regions of Brazil, equatorial Africa, and Southeast Asia. A large hotspot of host richness also occurs in west-central Europe, where WUIs are widespread. Map lines delineate study areas and do not necessarily depict accepted national boundaries.

zoonotic diseases in the global WUI. We produced a habitat map for each host species individually by masking unsuitable habitat types within the species range (see methods for details and data sources). We further refined these maps to focus only on habitat within the WUI (Schug et al. 2023).

We found that the near-continental-scale distribution of some habitat generalist host species, such as natal multimammate mouse (*Mastomys natalensis*), tanezumi rat (*Rattus tanezumi*), and red fox (*Vulpes vulpes*), means that there is suitable habitat for at least one host species across most of the WUI. Host richness mirrors global trends in mammal biodiversity: The highest host richness globally occurs in biodiverse regions of South America and across the equatorial tropics. This includes the WUI throughout Central America, equatorial and southern Africa, and South East Asia. Additional pockets of higher host richness exist in southern India and southwest China. However, the WUI in India and China is generally less host rich than in other parts of Asia (Figure 1).

Beyond the equatorial tropics, host richness is high across a large area of west-central Europe (Figure 1). This region has widespread WUI where large established cities occur in landscapes with forest and grassland (Schug et al. 2023). Although the European WUI is typically less biodiverse than the tropics, the fact that it is so widespread allows for diverse assemblages of host species.

2.2 | People and Hosts in the WUI

Mammal orders that most commonly occur with human populations in the WUI are bats (Chiroptera), rodents (Rodentia), apes, monkeys and lemurs (Primates), ungulates (Artiodactyla), carnivores (Carnivora), and hedgehogs and moles (Eulipotyphla) (Figure 2). At the individual disease level, diverse and widespread mammalian hosts for diseases such as rabies, leptospirosis, toxoplasmosis, plague, and leishmaniasis (visceral and cutaneous forms) co-occur with the largest human populations (Figure 2).

We found that 20% of the human population in the WUI live in places with high zoonotic potential—with habitat for more than 20 host species. Three of four people living in WUIs with high host richness (73% or 520 million people) are in LMICs (Figure 3). This includes 183 million people in Africa (12% of its population) (Figure 3), mostly in areas surrounding cities in Eastern Africa (particularly Kenya, Uganda, Ethiopia, Tanzania, and Zimbabwe), Western Africa (particularly Nigeria, Ghana, Burkina Faso, and Cote d'Ivoire), and South Africa (Figure 4). In South America, over 47 million people (11% of its population) live in WUIs with high zoonotic potential (Figure 4). Much of this population is concentrated in regions surrounding Rio de Janeiro and Sao Paulo in Brazil. Another hotspot can be observed in Central America and the Caribbean where 31 million people live in zones of high zoonotic potential (17% of its population). In South East Asia, the population in WUIs with high zoonotic potential is 175 million people (25% of its population).

India and China have moderate zoonotic potential but are home to very large human populations. We found that 10% of the combined population of India and China (275 million people) are exposed to WUIs with high zoonotic potential. However, an additional 465 million people (16% of the combined population) live in WUIs with moderate host richness that is habitat for 10–20 host species (Figure 3).

Zones of high zoonotic potential also occur in highly urbanized HICs, especially in west-central Europe, where more than half of the population live in WUIs with more than 20 zoonotic host species (393 million people, 53%). In North America, large populations along the east coast live in WUIs with low-to-moderate host richness. High exposure risk occurs in smaller clusters along the West Coast (Los Angeles, San Francisco, Seattle) and the southeastern United States (Florida).

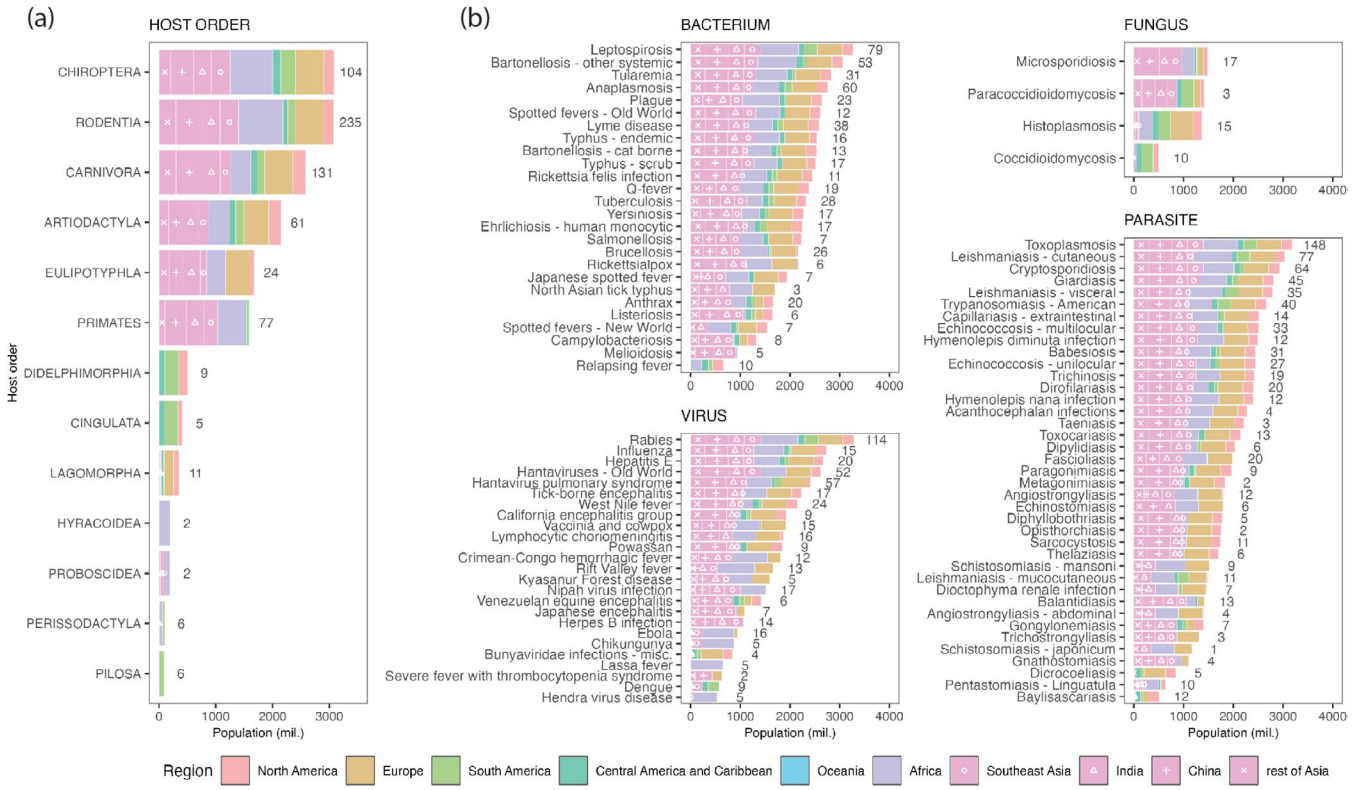

FIGURE 2 | Human populations globally that are at risk of zoonotic disease transmission due to overlaps between habitat for mammalian hosts of zoonotic diseases and the wildland–urban interface (WUI). (a) The global human population in the WUI containing habitat for at least one host of each mammal order. (b) The global population in the WUI containing habitat for at least one host of each disease. Diseases are ranked from top to bottom by the number of people exposed and only diseases with > 500 million people exposed are shown (full data in Supporting Information). Bar colors indicate the population within each global region. Asia is divided into four regions to provide greater detail (Southeast Asia, India, China, and all remaining Asian countries). Numbers at the end of bars indicate the number of host species present in each order for A, and number of host species for each disease for B.

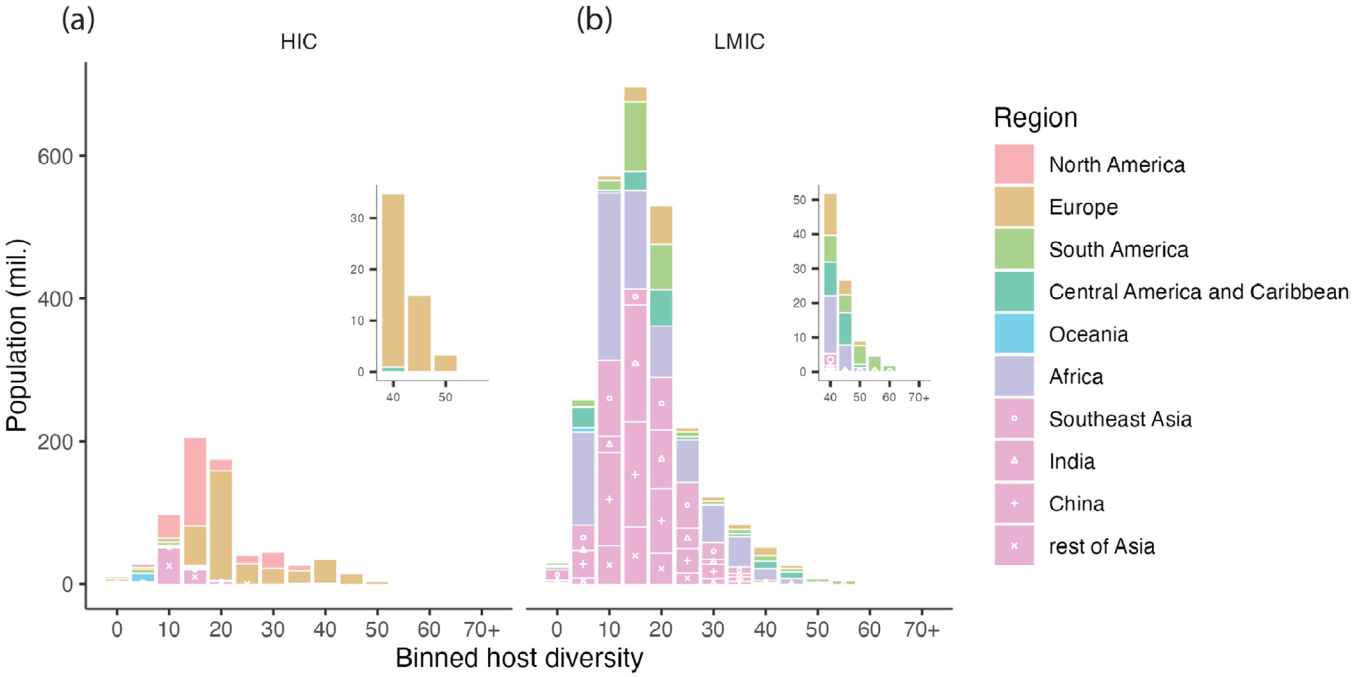

FIGURE 3 | The distribution of human populations varies by zoonotic host richness in the global WUI. The first column represents populations living in the WUI where there are no host species present. Column segments represent the populations of different global regions. A shows populations in high-income countries (HICs) and B shows populations in low–middle-income countries (LMICs), as defined by the World Bank (World Bank 2023). Inset panels show in greater detail the populations living in WUIs with at least 35 host species.

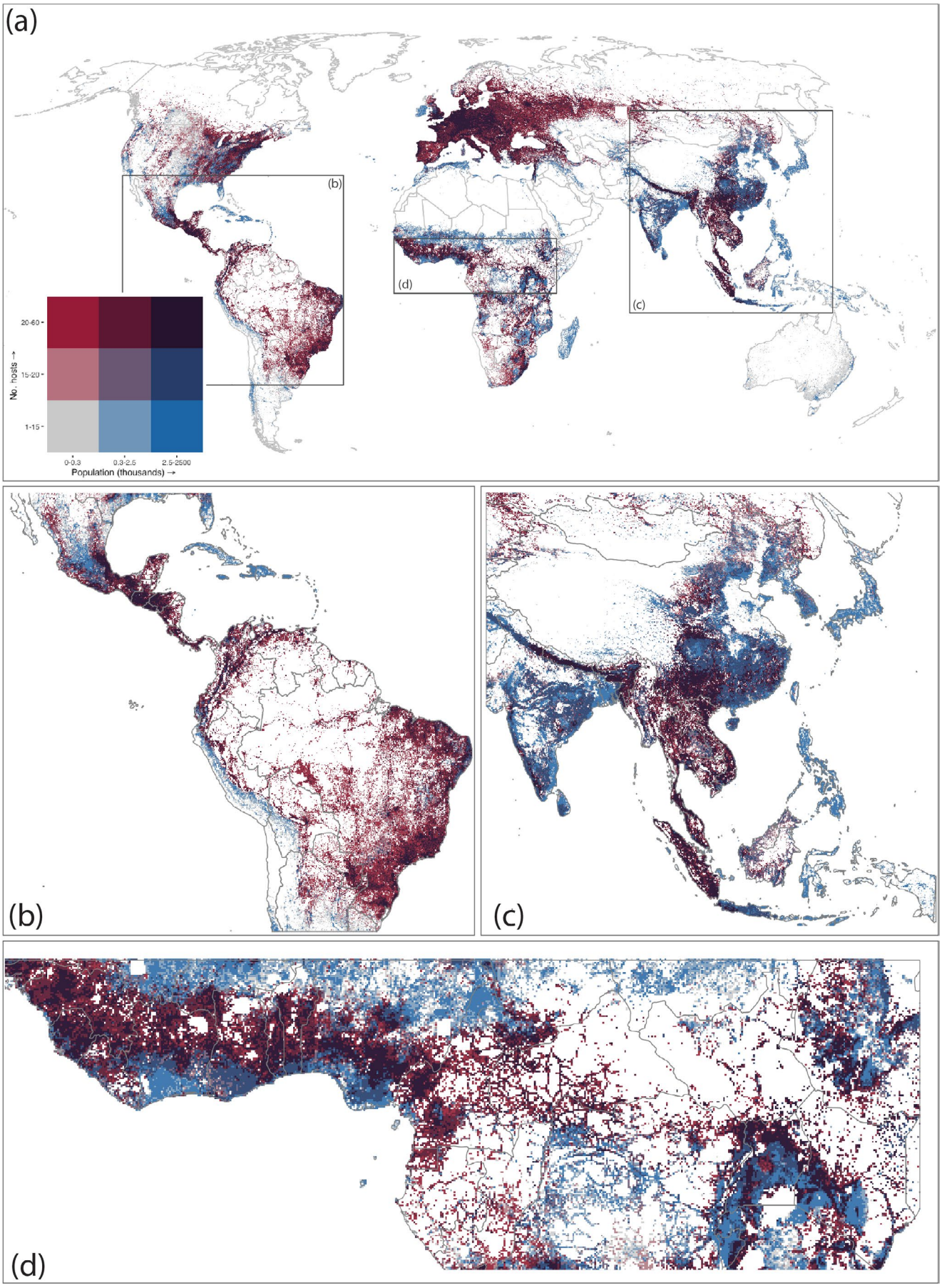

FIGURE 4 | Legend on next page.

FIGURE 4 | The distribution of zoonotic host richness (reds) and human population density (blues) in the wildland–urban interface varies globally. Panels (b and d) show in detail regions in the Global South with large populations living in the WUI with high zoonotic potential: Brazil and Central America, Southeast Asia, and equatorial Africa, respectively. Map lines delineate study areas and do not necessarily depict accepted national boundaries.

3 | Discussion

Our analysis demonstrates that hosts of zoonotic disease are widespread throughout the WUI. We find that 20% of the human population in the WUI are in zones of high zoonotic potential. Hotspots where large human populations intersect WUIs with high zoonotic potential are in Brazil, equatorial regions of East Africa and West Africa, and parts of Southeast Asia (Figure 4). In zones of high zoonotic potential, the presence of diverse host assemblages represents an important precondition for spillover and provides a measure of the biophysical risk associated with the WUI. Active clearing of wildlands, hunting, collection of firewood, and recreation in wildland areas can lead to direct exposure to pathogens of wildlife (Burthe et al. 2021). The presence of periurban agriculture can exacerbate risk of exposure where livestock act as intermediate or amplifying hosts for zoonotic diseases (Hassell et al. 2017). These zones of high zoonotic potential provide a way to better visualize the global risk of spillover in the WUI going forward.

Our analysis revealed that zoonotic potential in much of China and India is generally lower than in other parts of Asia. Cities in China and India are more often located in agricultural landscapes compared to other cities in Asia and these agricultural landscapes have relatively little WUI (Schug et al. 2023). Our analysis shows that these areas support fewer mammalian host species in the WUI as a result. India and China are, however, home to a combined population of around 2.8 billion people, or around one-third of the global population (United Nations Department of Economic and Social Affairs Population Division 2022). While the zoonotic potential is lower than in countries such as Brazil, the presence of such large human populations increases the risk of exposure to hosts at the population level and the risk of density-dependent pathogen spread following spillover infection (Figure 4).

A surprising result of our analysis was the identification of a zone of high zoonotic potential in northeastern North America and west-central Europe, which are not species-rich areas in general. High host richness here may be explained by the large WUI area in these regions and some geographic sampling bias, as discussed further below.

3.1 | Implications for Future Urbanization

The potential implications of the emergence of new zoonotic diseases are substantial as shown by the global spread of SARS-COV-2 in 2019, and the ongoing burden of neglected endemic zoonoses such as leishmaniasis and Chagas disease, which impose substantial health burdens, especially for some of the poorest populations (Halliday et al. 2015). More than half of the global population already lives in cities, and urban populations are set to increase by 2.5 billion people by 2050 (United Nations Department of Economic and Social Affairs Population Division 2018). As much as 90% of this growth is predicted to occur in Asia and Africa (particularly in China, India, and Nigeria) (United Nations Department of Economic and Social Affairs Population Division 2018), that is, in the places that we identify as already having large populations living in WUIs with high zoonotic potential. This concentration of the global population into urban areas, and subsequent urban land expansion could facilitate increasing frequency of contact between people and the hosts that we identified. This means that management of exposure to zoonoses in the WUI could become an increasingly important component of infectious disease and pandemic prevention globally.

3.2 | The Role of Socioeconomic Factors

Our quantification of the global WUI reveals hotspots of potential human contact with zoonotic mammalian hosts. These hotspots are based on biophysical factors, including the proximity of houses to wildlands, and the richness of host species, which may influence the nature and frequency of transmissible contacts leading to zoonotic spillover. However, given contact, the vulnerability of human populations to disease is also spatially variable. Vulnerability encompasses a complex set of economic, cultural, and political factors that govern the degree of exposure, susceptibility to infection, and progression to disease once exposure occurs. Socioeconomic conditions, political instability, conflict, and environmental health all differentially influence disease vulnerability.

Poverty is a particularly important amplifier of vulnerability to disease (Winck et al. 2022), which is why our finding that most people living in WUIs with high zoonotic potential are found in LMICs is cause for concern. Urban growth in LMICs is often informal, and poverty, poor-quality housing, lack of access to healthcare, and insecure land tenure can all contribute to elevated vulnerability to zoonotic disease (Chumo et al. 2023). In many cases, marginalized and disadvantaged communities have little choice in interacting with high-risk environments during their daily activities and often lack the means to make such activities safer (Evans et al. 2022). This includes, for example, the presence of domestic animals in living spaces (Barnes, Mumma, and Cumming 2018), or exposure to mosquitos from the use of outdoor spaces for washing and cooking where homes lack indoor plumbing (Evans et al. 2022). Impoverished communities in zones of high zoonotic potential therefore face the double threat of high zoonotic potential and vulnerability to disease.

Poverty is not, however, the only driver of zoonotic disease risk and some socioeconomic characteristics of HICs also increase vulnerability to disease. Our study demonstrates that hosts of zoonotic disease are present in widespread WUIs in WEST-central Europe and the East Coast of North America.

The low-density, sprawling style of urban development typical of North American cities, for example, brings people into contact with forests that contain hosts and vectors for Lyme disease (Bisanzio et al. 2020). Recreational use of wildlands within WUIs may also be more common in wealthier countries (Balmford et al. 2015). The roles that socioeconomic factors play in mediating exposure to hosts in the WUI, and for zoonotic diseases more broadly, are poorly understood and will be an important area of future research.

3.3 | Study Limitations

Our findings are based on the distribution of *known* hosts of *known* zoonotic pathogens in the global WUI. As such, they reflect both the current state of knowledge about mammal hosts and their associated zoonotic pathogens, including substantial gaps and biases in existing datasets. Pathogen sampling is biased toward well-studied, cosmopolitan host species, hosts that live in or near urban areas (Albery et al. 2021; Albery et al. 2022), zoonoses affecting human or domesticated animal health (Albery et al. 2021), and HICs, where more resources are available to fund research into zoonotic diseases (Han, Kramer, and Drake 2016). Ecological geospatial datasets such as the collections of expert range maps used in this study are also biased toward wealthier regions. This means there are potentially compounding biases where data on both host–pathogen associations and species distributions are lacking in developing regions. The results of our analysis reflect these biases. We note, for example, the large region of high host richness in west-central Europe. WUIs occupy around 15% of the land area in Europe, the highest of any global region (Schug et al. 2023). Widespread habitat for hosts of zoonotic diseases with the expansive WUI in Europe most likely contributes to the high host richness there. However, it is also likely that geographic biases in host and pathogen sampling may have contributed to the apparent concentration of hosts in this region. The reverse geographic bias may explain the sparsity of hosts in the WUI in places like Sri Lanka, Madagascar, Indonesian Borneo, and the Caribbean where high overall vertebrate richness suggests a higher pathogen richness than shown in our results (Guernier, Hochberg, and Guégan 2004; Jones et al. 2008).

Our method relies on mapping habitat suitable range—suitable habitat for a species within its known range. HSR maps do not necessarily indicate presence but give an indication of the potential for a species to occur. The presence/absence and abundance of a species within the WUI will also depend on other ecological parameters that are difficult to measure at a global scale, such as the level of habitat degradation, predator/prey interactions, hunting pressures, and other factors.

It is important to note that while our analyses characterize broad patterns of zoonotic risk at the WUI, the presence of suitable habitat does not presuppose transmissible contact between hosts and humans. For example, nuances in habitat usage may render transmissible contact unlikely (e.g., between rare arboreal species and humans). Conversely, our analyses treat species that thrive to high abundances in human-dominated settings similarly to species that may be present but much less likely to contaminate human dwellings, or whose abundances will decrease, not increase, in urban settings. Thus, translating these broad patterns into management actions will rely on integrating our understanding of host behavioral ecology and pathogen transmission modes in the WUI. The results of our study need to be considered in the context of these biases and limitations.

4 | Conclusions and Further Work

Human encroachment into wildlands and habitat fragmentation are two of the most important mechanisms fostering zoonotic disease outbreaks. Intrinsic to this spillover process is transmissible contact between host and human. Here, we identify global trends in zoonotic potential at spatial resolutions that are relevant for local human populations and, therefore, also local control and mitigation efforts. A main finding of our analysis is that populations in hotspots of the greatest zoonotic potential are predominated in LMICs where vulnerability to disease may be highest.

We highlight how hosts in the WUI can influence urban disease outcomes, linking existing knowledge about land-use patterns with zoonotic disease emergence in cities. For instance, understanding human–animal interactions at the WUI can provide a link between research on diseases with a known association with forest fragmentation (e.g., Brazilian spotted fever or Ebola virus disease (Rulli et al. 2017; Scinachi et al. 2017)) and diseases of urban poverty (Hotez 2017; Neiderud 2015), in places where forest fragmentation occurs due to expansion of urban slums. Human interactions and behaviors that govern the likelihood of pathogen transmission in the WUI will vary substantially by region, culture, socioeconomics, and the ecology of animal host communities. The pathways by which connectivity between cities and their surrounding landscapes could influence urban health are largely unexplored and represent an important area of future research. This will involve developing a deeper understanding of how urban and periurban residents interact with nature in ways that increase risk of infection. Examples could include assessing how factors, such as proximity to wildlands, forms of employment, or recreation that involve contact with nature, and patterns of rural–urban travel can influence localized disease outcomes[10].

Collectively, our findings emphasize the need to better integrate knowledge of landscape patterns into urban infectious disease research and policy. Understanding mechanisms of exposure and spread of zoonotic diseases in the rapidly expanding WUI will be a key component of global efforts to reduce zoonotic burdens and prevent future zoonotic outbreaks.

5 | Methods

5.1 | Host–Pathogen Associations

We analyzed previously collected data on the status of 731 mammal species and their association with 150 zoonotic diseases using primary literature cited within the Global Infectious Disease and Epidemiology Network (GIDEON)

(GIDEON 2023). We accessed and collated these data on August 12, 2019, after which each unique mammal host–zoonotic disease record was corrected (e.g., for false positives and incorrect associations) by checking primary literature. We updated disease names with the current names from GIDEON. This left a total of 144 zoonotic diseases. In several cases, it was unclear whether a mammal species is a true reservoir responsible for maintaining sylvatic transmission, a dead-end host not contributing to onward transmission, or a sentinel host whose seropositivity confirms pathogen exposure but not active infection or potential spillover transmission. While these distinctions are the subject of ongoing refinement by the scientific community, the host–zoonosis association data used in this paper treat all the above scenarios as positive associations, which offers a useful, but conservative estimate of where zoonoses occur globally. For more background on the development and application of host–pathogen data, see Han, Kramer, and Drake (2016).

Diseases spread entirely via human–human transmission were excluded. We included zoonotic diseases that have established endemic human–human transmission cycles if there is evidence that zoonotic spillover can still occur. This included diseases such as sylvatic malaria, sylvatic dengue, Zika, and chikungunya viruses. We acknowledge that spillover events directly from animal hosts are likely to be rare for these diseases but include them because they are part of the global picture of exposure to zoonotic hosts.

We harmonized mammal host names in the host–pathogen dataset with the Mammal Diversity Database (MDD) taxonomy v1.9.1 (MMD 2022). We excluded species that could not be matched by either the MDD scientific name directly or via the equivalent species name from the Mammal Species of the World (MSW) taxonomy (Wilson and Reeder 2005) as per the "MSW3" column in the MDD database. This gave us a total of 686 mammal host species.

5.2 | Global WUI

We here used a previously published global-scale raster dataset of the WUI with a 10 m spatial resolution (WUI) (Schug et al. 2023). This dataset distinguished between intermix WUI where buildings directly mix with wildland vegetation and interface WUI where buildings are in proximity to large patches of wildland vegetation. It additionally distinguished between WUI dominated by grassland versus dominated by other wildland vegetation and distinguished between four non-WUI background classes. The global WUI dataset is based on spatial analysis, extracting information from global land cover (European Space Agency World Cover (Zanaga et al. 2020)) and building density raster data (Global Human Settlement GHS-BUILT-S—R2022A (Pesaresi and P. 2022)). The global WUI data followed the conceptual WUI definition of the US Federal Register (Forest Service 2001), which is the most widely used WUI definition in the United States and many other countries (Radeloff et al. 2005; Schug et al. 2023). The global WUI workflow identified intermix WUI in each raster pixel where building density in a 500 m radius was $>0.5\%$ and wildland vegetation in the same radius was $\geq 50\%$. The workflow identified interface WUI in each raster pixel where building density in a 500 m radius was $>0.5\%$, wildland vegetation in the same radius was $<50\%$, and where a patch of wildland vegetation $>5\text{km}^2$ was less than 2.4 km away. Areas with a building density $>15\%$ were excluded from intermix WUI based on careful inspection of the data. For our analysis, we reclassified the global WUI dataset into WUI versus non-WUI raster pixels. We aggregated the data from its original 10 m resolution to a 300 m resolution using a nearest-neighbor approach to match the resolution and grid of the host species habitat maps (see below).

5.3 | Habitat Mapping

We used a recently published set of mammal expert range maps (Marsh et al. 2022) to define species ranges. We filtered the full set of species to those that matched our host–pathogen data and rasterized each range map at 300 m resolution. This created a binary raster range map for each species which we reprojected into the Equi7Grid projection (Bauer-Marschallinger, Sabel, and Wagner 2014) and analyzed in 100-km tiles.

We gathered species habitat and elevation preferences from Map of Life (Jetz, McPherson, and Guralnick 2012) (see www.mol.org). Global landcover was available in the European Space Agency's CCI landcover dataset v2.1.1 (European Space Agency 2017) and elevation in the global ALOS World 3D 30-m mesh digital elevation model v3.2 (DEM) (Tadono et al. 2016). We reprojected both datasets to the Equi7Grid and divided them into 100-km tiles. The CCI landcover layer has a native 300 m resolution, and we aggregated the DEM from 30 to 300 m using nearest-neighbor interpolation.

Range maps define the full range of a species and typically contain areas of nonsuitable habitat in which the species is not expected to occur. We used Map of Life habitat and elevation preferences to refine the range maps using a modified method based on the approach of Powers and Jetz (2019). The range maps are refined in three steps. First, using the CCI landcover layer to define land-use categories, we masked each species' rasterized range map to include only those land-use categories identified as suitable habitats by the MOL habitat preference dataset. Second, we further masked this map to exclude areas within the range map but outside the elevation limits identified in the MOL habitat preferences data. Finally, we used the global WUI dataset to mask the habitat map for each species to include only habitat in the WUI. This returned a tiled, binary habitat map for each species, constrained to suitable habitat and elevation within the WUI.

This method sometimes maps host species' habitat outside of the range where endemic transmission of a disease is known to occur. This means, for example, we identify hosts of yellow fever in Asia, where an outbreak caused by endemic transmission has not been recorded. In many cases, there is still poor understanding of pathogen cooccurrence where host species are recorded, but endemic transmission is not. We include the full host range

TABLE 1 | Global region classifications based on columns in the Natural Earth Admin 0–Countries 1:50 vector data, and the Equi7Grid dataset. Classification was done at the scale of each 100-km tile in the Equi7Grid projection. The AND/OR column indicates where conditions for both (AND) or either (OR) datasets must be met for classification into a given region.

Region	Natural earth data attributes	AND/OR	Equi7Grid attributes
North America	SUBREGION! = Central America and Caribbean	AND	ZONE == Northamerica
South America	SUBREGION! = Central America and Caribbean	AND	ZONE == Southamerica
Central America and Caribbean	SUBREGION == Central America OR (NAME_LONG == Cuba OR NAME_LONG == Jamaica OR NAME_LONG == Haiti OR NAME_LONG == Dominican Republic)	—	—
Europe	NAME_LONG == Spain	OR	ZONE == Europe
Oceania	—	—	ZONE == Oceania
Africa	—	—	ZONE == Africa
India	NAME_LONG == India	—	—
China	NAME_LONG == China	—	—
Southeast Asia	SUBREGION == South-Eastern Asia	—	—
Rest of Asia	NAME_LONG! = China AND NAME_LONG! = India AND SUBREGION! = South-Eastern Asia	AND	ZONE == Asia

to indicate the potential range of risk associated with spillover transmission of each disease in the WUI.

We calculate host richness by summing host species with suitable habitats identified by our refined range maps at the pixel level.

5.4 | Population

We used the ESA's Global Human Settlement Layer (GHSL) data (ver. R2022A) to estimate global population at 100 m resolution in 2020 (Schiavina et al. 2023). We also reprojected these data into the Equi7Grid at their native resolution and split them into 100-km tiles.

We estimated the size of the population exposed to different levels of zoonotic host richness by first reclassifying global maps of host richness into bins with intervals of five. We disaggregated the host richness maps to 100 m resolution to match the population raster and then summed the population at each binned host richness level.

We used country-level income as a proxy for understanding vulnerability to disease by dividing countries into HICs and LMICs, respectively, based on the World Bank classification for 2022 (World Bank 2023). This coarse categorization does not capture substantial economic, cultural, and political heterogeneity, which is difficult to quantify (Haug, Braveboy-Wagner, and Maihold 2021). It also glosses over the fact that many LMICs are exceptionally well prepared to respond to endemic spillover events that occur with regularity (e.g., Ebola virus spillovers in Uganda (Potter et al. 2022)). Here, we include it as a first proxy to highlight populations that may be disproportionately vulnerable and are deserving of more detailed study.

5.5 | Global Regions

We summarized our results across seven key global regions (Table 1, Figure 5). Asia is divided into four subregions: India, China, Southeast Asia, and all remaining Asian countries to avoid generalizing over a very large percentage of the global population. Each 100-km tile was assigned to one region. Where tiles spanned more than one region (i.e., along region borders), the tile was assigned to the region with the largest overlap. Regions were defined by a combination of Natural Earth Admin 0–Countries 1:50 global vector maps and the Equi7Grid metadata as shown in Table 1.

5.6 | Software

We conducted all analyses using R (version 4.3.2) (R Core Team 2023), specifically using the *sf* package (version 1.0–13) (Pebesma 2018) to handle vector data, *stars* (version 0.6–4) for raster data (Pebesma and Bivand 2023), and *tidyverse* (version 2.0.0) to analyze data and create figures (Wickham et al. 2019). Automation of the GIDEON API queries was performed using the *httr2* package (version 0.2.3) (Wickham 2023). Analyses were processed on Yale's high-performance compute clusters on a tile-by-tile basis, which allowed us to distribute tasks across multiple cores.

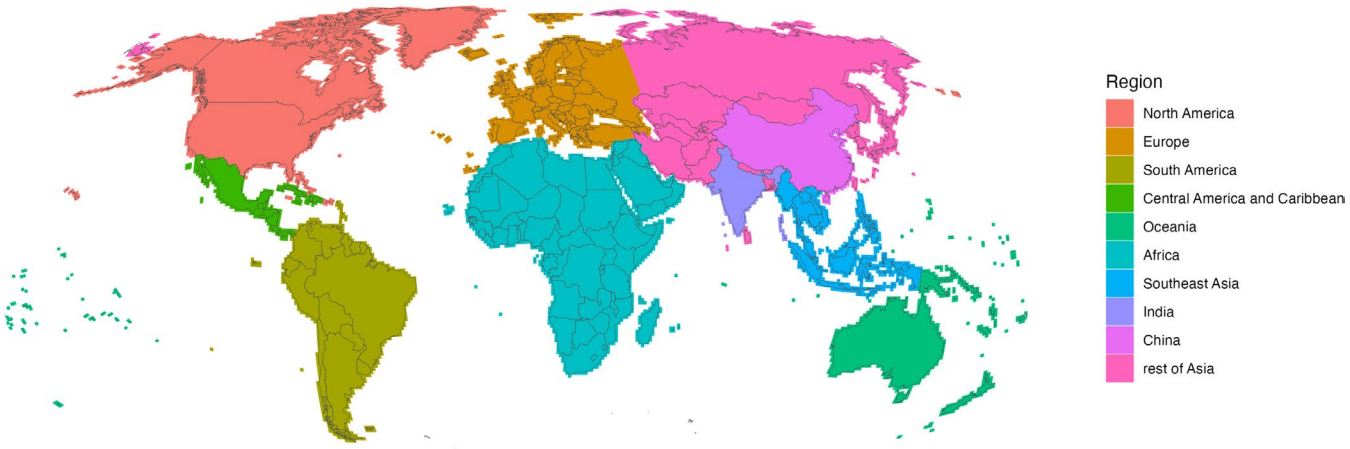

FIGURE 5 | Global regions used for analysis. Map lines delineate study areas and do not necessarily depict accepted national boundaries.

Author Contributions

Rohan D. Simkin: conceptualization, data curation, formal analysis, methodology, visualization, writing – original draft, writing – review and editing. **Barbara A. Han:** conceptualization, supervision, writing – original draft, writing – review and editing. **Volker C. Radeloff:** conceptualization, data curation, methodology, writing – original draft, writing – review and editing. **Shannon LaDeau:** conceptualization, writing – original draft, writing – review and editing. **Franz Schug:** conceptualization, data curation, methodology, writing – original draft, writing – review and editing. **Karen C. Seto:** conceptualization, formal analysis, methodology, supervision, visualization, writing – original draft, writing – review and editing.

Acknowledgments

We thank John Wilshire, Ajay Ranipeta, Julie Portman, and Walter Jetz from the Map of Life team for support with biodiversity data and Walter Jetz for guidance on its analysis. We acknowledge funding from the E.O. Wilson Biodiversity Foundation through the Half-Earth Project which supported biodiversity data provision from the Map of Life team. Dr. Adrian Castellanos prepared the host–pathogen association data used in this analysis. This work was funded by NASA LCLUC MuSLI grant 80NSSC21K0310; NASA IDS grant 80NSSC24K0303; NASA grant 22-SMDSS22-0042; US Department of Agriculture McIntire Stennis award WIS03072 and NSF DEB-1717282.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data and code that supports the findings of this study are freely available at https://doi.org/10.5281/zenodo.14624578. Mammal expert range map polygons were obtained from Map of Life at https://doi.org/10.48600/mol-48vz-p413. Species habitat and elevation preferences were obtained from Map of Life at https://doi.org/10.48600/0341-nf74. Global WUI raster data were obtained from Zenodo at https://zenodo.org/records/7941460. The ALOS Global Digital Surface Model "ALOS World 3D - 30m (AW3D30)" v3.2 digital elevation model (DEM) data were obtained from the Japan Aerospace Exploration Agency at https://www.eorc.jaxa.jp/ALOS/en/dataset/aw3d30/aw3d30_e.htm. GHSL population data were obtained from the European Commission, Joint Research Centre (JRC) at https://doi.org/10.2905/AB7AD451-5ED5-44A6-A4D0-9F7A4E848CEE. The CCI Landcover dataset was obtained from ESA at https://www.esa-landcover-cci.org/ (v2.1.1). Continent and country boundaries used to demarcate regions and for visualizations were obtained from Natural Earth at https://www.naturalearthdata.com/. Spatial data for the Equi7Grid projection were obtained from Dryad at https://doi.org/10.5281/zenodo.1048530 and Github at https://github.com/TUW-GEO/Equi7Grid/tree/v0.2.4.

References

Albery, G. F., D. J. Becker, L. Brierley, et al. 2021. "The Science of the Host–Virus Network." *Nature Microbiology* 6, no. 12: 1483–1492. https://doi.org/10.1038/s41564-021-00999-5.

Albery, G. F., C. J. Carlson, L. E. Cohen, et al. 2022. "Urban-Adapted Mammal Species Have More Known Pathogens." *Nature Ecology & Evolution* 6, no. 6: 794–801. https://doi.org/10.1038/s41559-022-01723-0.

Alirol, E., L. Getaz, B. Stoll, F. Chappuis, and L. Loutan. 2011. "Urbanisation and Infectious Diseases in a Globalised World." *Lancet Infectious Diseases* 11, no. 2: 131–141. https://doi.org/10.1016/S1473-3099(10)70223-1.

Balmford, A., J. M. H. Green, M. Anderson, et al. 2015. "Walk on the Wild Side: Estimating the Global Magnitude of Visits to Protected Areas." *PLoS Biology* 13, no. 2: e1002074. https://doi.org/10.1371/journal.pbio.1002074.

Bar-Massada, A., V. C. Radeloff, and S. I. Stewart. 2014. "Biotic and Abiotic Effects of Human Settlements in the Wildland–Urban Interface." *Bioscience* 64, no. 5: 429–437. https://doi.org/10.1093/biosci/biu039.

Barnes, A. N., J. Mumma, and O. Cumming. 2018. "Role, Ownership and Presence of Domestic Animals in Peri-Urban Households of Kisumu, Kenya." *Zoonoses and Public Health* 65, no. 1: 202–214. https://doi.org/10.1111/zph.12429.

Bauer-Marschallinger, B., D. Sabel, and W. Wagner. 2014. "Optimisation of Global Grids for High-Resolution Remote Sensing Data." *Computers & Geosciences* 72: 84–93. https://doi.org/10.1016/j.cageo.2014.07.005.

Bento-Gonçalves, A., and A. Vieira. 2020. "Wildfires in the Wildland-Urban Interface: Key Concepts and Evaluation Methodologies." *Science of the Total Environment* 707: 135592. https://doi.org/10.1016/j.scitotenv.2019.135592.

Bethony, J., S. Brooker, M. Albonico, et al. 2006. "Soil-Transmitted Helminth Infections: Ascariasis, Trichuriasis, and Hookworm." *Lancet* 367, no. 9521: 1521–1532. https://doi.org/10.1016/S0140-6736(06)68653-4.

Bisanzio, D., M. P. Fernández, E. Martello, R. Reithinger, and M. A. Diuk-Wasser. 2020. "Current and Future Spatiotemporal Patterns of

Lyme Disease Reporting in the Northeastern United States." *JAMA Network Open* 3, no. 3: e200319. https://doi.org/10.1001/jamanetworkopen.2020.0319.

Bradley, C. A., and S. Altizer. 2007. "Urbanization and the Ecology of Wildlife Diseases." *Trends in Ecology & Evolution* 22, no. 2: 95–102. https://doi.org/10.1016/j.tree.2006.11.001.

Burthe, S. J., S. M. Schäfer, F. A. Asaaga, et al. 2021. "Reviewing the Ecological Evidence Base for Management of Emerging Tropical Zoonoses: Kyasanur Forest Disease in India as a Case Study." *PLoS Neglected Tropical Diseases* 15, no. 4: e0009243. https://doi.org/10.1371/journal.pntd.0009243.

Chala, B., and F. Hamde. 2021. "Emerging and Re-Emerging Vector-Borne Infectious Diseases and the Challenges for Control: A Review." *Frontiers in Public Health* 9: 715759. https://doi.org/10.3389/fpubh.2021.715759.

Chumo, I., C. Kabaria, A. Shankland, and B. Mberu. 2023. "Drivers of Vulnerability to Health and Wellbeing Challenges in Informal Settlements." *Frontiers in Sustainable Cities* 5. https://doi.org/10.3389/frsc.2023.1057726.

Connolly, C., R. Keil, and S. H. Ali. 2020. "Extended Urbanisation and the Spatialities of Infectious Disease: Demographic Change, Infrastructure and Governance." *Urban Studies* 58, no. 2: 245–263. https://doi.org/10.1177/0042098020910873.

Despommier, D., B. R. Ellis, and B. A. Wilcox. 2006. "The Role of Ecotones in Emerging Infectious Diseases." *EcoHealth* 3, no. 4: 281–289. https://doi.org/10.1007/s10393-006-0063-3.

Ecke, F., B. A. Han, B. Hörnfeldt, et al. 2022. "Population Fluctuations and Synanthropy Explain Transmission Risk in Rodent-Borne Zoonoses." *Nature Communications* 13, no. 1: 7532. https://doi.org/10.1038/s41467-022-35273-7.

Espinosa, R., D. Tago, and N. Treich. 2020. "Infectious Diseases and Meat Production." *Environmental and Resource Economics* 76, no. 4: 1019–1044. https://doi.org/10.1007/s10640-020-00484-3.

European Space Agency. 2017. "Land Cover CCI Product User Guide Version 2.0." www.esa-landcover-cci.org.

Evans, M. V., S. Bhatnagar, J. M. Drake, C. C. Murdock, and S. Mukherjee. 2022. "Socio-Ecological Dynamics in Urban Systems: An Integrative Approach to Mosquito-Borne Disease in Bengaluru, India." *People and Nature* 4, no. 3: 730–743. https://doi.org/10.1002/pan3.10311.

Faria, N. R., M. U. G. Kraemer, S. C. Hill, et al. 2018. "Genomic and Epidemiological Monitoring of Yellow Fever Virus Transmission Potential." *Science* 361, no. 6405: 894. https://doi.org/10.1126/science.aat7115.

Forest Service, B. o. I. A., Bureau of Land Management, Fish and Wildlife Service & National Park Service. 2001. "Urban Wildland Interface Communities Within the Vicinity of Federal Lands That are at High Risk From Wildfire." *Federal Register* 66, no. 3: 751–777.

GIDEON. 2023. "Global Infectious Diseases and Epidemiology Online Network." www.gideononline.com.

Goldstein, J. E., I. Budiman, A. Canny, and D. Dwipartidrisa. 2022. "Pandemics and the Human-Wildlife Interface in Asia: Land Use Change as a Driver of Zoonotic Viral Outbreaks." *Environmental Research Letters* 17, no. 6: 63009. https://doi.org/10.1088/1748-9326/ac74d4.

Guernier, V., M. E. Hochberg, and J.-F. Guégan. 2004. "Ecology Drives the Worldwide Distribution of Human Diseases." *PLoS Biology* 2, no. 6: e141. https://doi.org/10.1371/journal.pbio.0020141.

Halliday, J. E. B., K. J. Allan, D. Ekwem, S. Cleaveland, R. R. Kazwala, and J. A. Crump. 2015. "Endemic Zoonoses in the Tropics: A Public Health Problem Hiding in Plain Sight." *Veterinary Record* 176, no. 9: 220–225. https://doi.org/10.1136/vr.h798.

Han, B. A., A. M. Kramer, and J. M. Drake. 2016. "Global Patterns of Zoonotic Disease in Mammals." *Trends in Parasitology* 32, no. 7: 565–577. https://doi.org/10.1016/j.pt.2016.04.007.

Hassell, J. M., M. Begon, M. J. Ward, and E. M. Fevre. 2017. "Urbanization and Disease Emergence: Dynamics at the Wildlife-Livestock-Human Interface." *Trends in Ecology & Evolution* 32, no. 1: 55–67. https://doi.org/10.1016/j.tree.2016.09.012.

Haug, S., J. Braveboy-Wagner, and G. Maihold. 2021. "The 'Global South' in the Study of World Politics: Examining a Meta Category." *Third World Quarterly* 42, no. 9: 1923–1944. https://doi.org/10.1080/01436597.2021.1948831.

Hikufe, E. H., C. M. Freuling, R. Athingo, et al. 2019. "Ecology and Epidemiology of Rabies in Humans, Domestic Animals and Wildlife in Namibia, 2011-2017." *PLoS Neglected Tropical Diseases* 13, no. 4: e0007355. https://doi.org/10.1371/journal.pntd.0007355.

Hotez, P. J. 2017. "Global Urbanization and the Neglected Tropical Diseases." *PLoS Neglected Tropical Diseases* 11, no. 2: e0005308. https://doi.org/10.1371/journal.pntd.0005308.

Islam, M. M., P. Dutta, R. Rashid, et al. 2023. "Pathogenicity and Virulence of Monkeypox at the Human-Animal-Ecology Interface." *Virulence* 14, no. 1: 2186357. https://doi.org/10.1080/21505594.2023.2186357.

Jetz, W., J. M. McPherson, and R. P. Guralnick. 2012. "Integrating Biodiversity Distribution Knowledge: Toward a Global Map of Life." *Trends in Ecology & Evolution* 27, no. 3: 151–159. https://doi.org/10.1016/j.tree.2011.09.007.

Jones, K. E., N. G. Patel, M. A. Levy, et al. 2008. "Global Trends in Emerging Infectious Diseases." *Nature* 451, no. 7181: 990–993. https://doi.org/10.1038/nature06536.

Lawson, E. T., J. S. Ayivor, F. Ohemeng, and Y. Ntiamoa-Baidu. 2019. "Avoiding Bites and Scratches? Understanding the Public Health Implication of Human–Bat Interactions in Ghana." *Zoonoses and Public Health* 66, no. 1: 108–116. https://doi.org/10.1111/zph.12540.

Levy, M. Z., C. M. Barbu, R. Castillo-Neyra, et al. 2014. "Urbanization, Land Tenure Security and Vector-Borne Chagas Disease." *Proceedings of the Royal Society B: Biological Sciences* 281, no. 1789: 20141003. https://doi.org/10.1098/rspb.2014.1003.

Mammal Diversity Database. 2022. "Mammal Diversity Database (Version 1.9.1)." https://doi.org/10.5281/zenodo.7358650.

Marsh, C. J., Y. V. Sica, C. J. Burgin, et al. 2022. "Expert Range Maps of Global Mammal Distributions Harmonised to Three Taxonomic Authorities." *Journal of Biogeography* 49, no. 5: 979–992. https://doi.org/10.1111/jbi.14330.

McCarthy, J., and T. A. Moore. 2000. "Emerging Helminth Zoonoses." *International Journal for Parasitology* 30, no. 12: 1351–1359. https://doi.org/10.1016/S0020-7519(00)00122-3.

Neiderud, C.-J. 2015. "How Urbanization Affects the Epidemiology of Emerging Infectious Diseases." *Infection Ecology & Epidemiology* 5: 27060. https://doi.org/10.3402/iee.v5.27060.

Pebesma, E. 2018. "Simple Features for R: Standardized Support for Spatial Vector Data." *R Journal* 10, no. 1: 439–446. https://doi.org/10.32614/RJ-2018-009.

Pebesma, E., and R. Bivand. 2023. *Spatial Data Science: With Applications in R*. London: Chapman and Hall/CRC.

Pesaresi, M. P. 2022. "GHS Built-up Surface Grid, Derived From Sentinel2 and Landsat, Multitemporal (1975–2030)."

Plowright, R. K., C. R. Parrish, H. McCallum, et al. 2017. "Pathways to Zoonotic Spillover." *Nature Reviews. Microbiology* 15, no. 8: 502–510. https://doi.org/10.1038/nrmicro.2017.45.

Plowright, R. K., J. K. Reaser, H. Locke, et al. 2021. "Land Use-Induced Spillover: A Call to Action to Safeguard Environmental, Animal, and Human Health." *Lancet Planetary Health* 5, no. 4: e237–e245. https://doi.org/10.1016/S2542-5196(21)00031-0.

Potter, C., L. Mullen, S. Ssendagire, et al. 2022. "Retrospective Identification of Key Activities in Uganda's Preparedness Measures Related to the 2018–2020 EVD Outbreak in Eastern DRC Utilizing a Framework Evaluation Tool." *PLOS Global Public Health* 2, no. 5: e0000428. https://doi.org/10.1371/journal.pgph.0000428.

Powers, R. P., and W. Jetz. 2019. "Global Habitat Loss and Extinction Risk of Terrestrial Vertebrates Under Future Land-Use-Change Scenarios." *Nature Climate Change* 9: 323–329. https://doi.org/10.1038/s41558-019-0406-z.

R Core Team. 2023. "_R: A Language and Environment for Statistical Computing_Vienna, Austria: R Foundation for Statistical Computing." https://www.R-project.org.

Radeloff, V. C., R. B. Hammer, S. I. Stewart, J. S. Fried, S. S. Holcomb, and J. F. McKeefry. 2005. "The Wildland-Urban Interface in the United States." *Ecological Applications* 15: 799–805.

Radeloff, V. C., D. P. Helmers, H. A. Kramer, et al. 2018. "Rapid Growth of the US Wildland-Urban Interface Raises Wildfire Risk." *Proceedings of the National Academy of Sciences* 115, no. 13: 3314–3319. https://doi.org/10.1073/pnas.1718850115.

Rulli, M. C., M. Santini, D. T. S. Hayman, and P. D'Odorico. 2017. "The Nexus Between Forest Fragmentation in Africa and Ebola Virus Disease Outbreaks." *Scientific Reports* 7, no. 1: 41613. https://doi.org/10.1038/srep41613.

Schiavina, M., S. Freire, A. Carioli, and K. MacManus. 2023. "GHS-POP R2023A–GHS Population Grid Multitemporal (1975–2030)."

Schug, F., A. Bar-Massada, A. R. Carlson, et al. 2023. "The Global Wildland–Urban Interface." *Nature* 621, no. 7977: 94–99. https://doi.org/10.1038/s41586-023-06320-0.

Scinachi, C. A., G. A. C. G. Takeda, L. F. Mucci, and A. Pinter. 2017. "Association of the Occurrence of Brazilian Spotted Fever and Atlantic Rain Forest Fragmentation in the São Paulo Metropolitan Region, Brazil." *Acta Tropica* 166: 225–233. https://doi.org/10.1016/j.actatropica.2016.11.025.

Shah, H. A., P. Huxley, J. Elmes, and K. A. Murray. 2019. "Agricultural Land-Uses Consistently Exacerbate Infectious Disease Risks in Southeast Asia." *Nature Communications* 10, no. 1: 4299. https://doi.org/10.1038/s41467-019-12333-z.

Spengler, J. R., É. Bergeron, and P. E. Rollin. 2016. "Seroepidemiological Studies of Crimean-Congo Hemorrhagic Fever Virus in Domestic and Wild Animals." *PLoS Neglected Tropical Diseases* 10, no. 1: e0004210. https://doi.org/10.1371/journal.pntd.0004210.

Tadono, T., H. Nagai, H. Ishida, et al. 2016. "Generation of the 30 M-Mesh Global Digital Surface Model by ALOS Prism." *ISPRS Arch Photogramm Remote Sensing and Spatial Information Sciences* 4: 157–162. https://doi.org/10.5194/isprs-archives-XLI-B4-157-2016.

UN-Habitat. 2022. "World Cities Report. Retrieved from Nairobi, Kenya."

United Nations Department of Economic and Social Afairs Population Division. 2018. "World Urbanization Prospects: The 2018 Revision, Online Edition."

United Nations Department of Economic and Social Affairs Population Division. 2022. "World Population Prospects 2022 Online Edition."

USDA. 2001. "Urban Wildland Interface Communities Within Vicinity of Federal Lands That Are at High Risk From Wildfire." *Federal Register* 66: 751–777.

Wickham, H. 2023. "httr2: Perform HTTP Requests and Process the Responses. R package Version 1.0.0." https://github.com/r-lib/httr2, https://httr2.r-lib.org.

Wickham, H., M. Averick, J. Bryan, et al. 2019. "Wlcome to the Tidyverse." *Journal of Open Source Software* 4, no. 43: 1686. https://doi.org/10.21105/joss.01686.

Wilson, D. E., and D. M. Reeder, eds. 2005. *Mammal Species of the World: A Taxonomic and Geographic Reference*. 3rd ed. Baltimore, Maryland: Johns Hopkins University Press.

Winck, G. R., R. L. G. Raimundo, H. Fernandes-Ferreira, et al. 2022. "Socioecological Vulnerability and the Risk of Zoonotic Disease Emergence in Brazil." *Science Advances* 8, no. 26: eabo5774. https://doi.org/10.1126/sciadv.abo5774.

World Bank. 2023. "World Bank Country and Lending Groups." https://datahelpdesk.worldbank.org/knowledgebase/articles/906519-world-bank-country-and-lending-groups.

Zanaga, D., R. Van De Kerchove, W. De Keersmaecker, et al. 2020. "ESA WorldCover 10 m 2020 v100 (Version v100) [Data Set]."

Supporting Information

Additional supporting information can be found online in the Supporting Information section.