



## ARTICLE OPEN ACCESS

# Fixing Epidemic Risk, Fixing Epidemic Spacetime? How Emerging Disease Science Writes Wild Stories About Pandemic Origins

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## ABSTRACT

Using the US Agency for International Development (USAID) PREDICT program as a case study, I argue that mainstream emerging disease science (re)produces Orientalist imaginaries of pandemic risk through narratives about wildlife infections as inherently “backwards” in epidemic time relative to human and domestic animal infections. This temporal imaginary leverages a broad distinction between wild and domestic animals to naturalize Western geographies as low-risk and human spaces and populations in the Global South as the global origin for pandemic threats. To examine these narratives, I draw on nearly 50 scientific publications produced by PREDICT, one of the largest global viral discovery and pandemic risk assessment programs to date. In doing so, I illustrate how ideas about epidemic time help (re)produce Orientalist spatial imaginaries as well as how wildness not only marks animals as belonging to a different *place* than (certain) humans, but also to a different time.

## 1 | Introduction

Something the weather people would give their right arm to be able to [do is go] over Niger, take an action and actually change the meteorologic conditions that would prevent anything spilling out into the Atlantic. So we can learn from the weather forecasting, but we aren't constrained by it. We have the ability to be much more powerful.

(PREDICT practitioner, interview with author)

While scientists and policymakers did not use the language of “emerging infectious diseases” (EID) until the late 20th century (Cooper 2008; Holmberg 2024), practitioners from Europe and its settler colonies have long treated infectious disease risk as a threat inherently external to Western populations and geographies. During the 19th century, Europeans fixated on qualities

of “Eastern” landscapes, populations, and cultures as causes of pandemic cholera emergence—even as these early pandemics closely tracked the movements of colonial troops and resource extraction (Arnold 1986; Downs 2021; Raza Kolb 2020; White 2023). Scientists and lawmakers developing new tools to study and regulate infectious disease spread codified this viewpoint into their scientific reports and the world's first international disease control legislation (Downs 2021; Raza Kolb 2020; White 2023). White (2023) calls this perspective and set of discourses *epidemic Orientalism*.

Like Said's original formulation, epidemic Orientalism identifies a way that Europe and its settler colonies have defined themselves “in opposition to the rest of the world” that they target for “exploitation, extraction, and knowledge creation” (White 2023, 18). Said (1978, 12) describes Orientalism more broadly as a discourse that defines the world as “made up of two unequal halves, Orient and Occident” and leverages this crude

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geographical imaginary within the West's colonial projects as part of exerting control over people and landscapes defined as fundamentally other. Epidemic Orientalism names the role that perceptions about disease threat play in constructing and perpetuating this geographic imaginary. White (2023, 31) explains how, across contexts, epidemic Orientalism [i] represents global geographies in relation to the disease threats they pose to the West; [ii] portrays the presence of infectious disease (in people and places) as an often racialized marker "of innate difference and inferiority"; and [iii] defines Western modernity through a disease-free status to which the rest of the world should strive.

In his history of the WHO's International Health Regulations (IHRs), White (2023) traces different iterations of epidemic Orientalism through infectious disease control legislation from the 19th century to the present, as each update evolves to encode this viewpoint in new ways. Infectious disease science has similarly updated its language and discursive tropes over time, often in tandem with the development of new legal and public health tools to manage outbreaks (Downs 2021; Raza Kolb 2020; Wald 2008). Starting in the late 20th century, US-based scientists and policymakers began mobilizing resources around what they called *emerging infectious diseases* like HIV/AIDS and Ebola as a significant global health security concern (Cooper 2008; Lakoff 2017; Wald 2008). Early proponents characterized EIDs as those "that either have newly appeared in a population or are rapidly expanding their range" (Morse 1993, 10). While not a novel phenomenon, invoking this term in media, pop culture, and policy spaces has served to promote and normalize militarized interventions targeting the Global South, guided by "apolitical" scientific expertise, as the primary means of EID containment and prevention (Cooper 2008; Raza Kolb 2020; Wald 2008). Critical geographers and disease ecologists have long lambasted mainstream EID science for failing to attend to the structural drivers of infectious disease emergence, leaving capitalist drivers of land use change and shifting human–environment interactions underexamined as causes of global pandemic risk (i.e., Chaves et al. 2022; Wallace 2009, 2016; Wallace et al. 2015, 2020). Others have recently called for analyses that interrogate "the structuring conditions of racialization" alongside capitalism and uneven development as central to "contemporary biosecurity initiatives" like EID science (Prouse and Arefin 2026, 68). By what discursive mechanisms does contemporary EID science naturalize Orientalist imaginaries of infectious disease risk? Are there ways that it encodes these ideas without explicitly invoking human or geographic difference?

Using the US Agency for International Development (USAID) PREDICT<sup>1</sup> program as a case study, I argue that epidemic Orientalism in hegemonic EID science relies on understanding wildlife outbreaks as inherently "backwards" in epidemic time relative to infections of people and domestic species. Here *epidemic time* refers to any assumed progression of outbreak stages—for example, in PREDICT's case: [i] animal–animal transmission; [ii] animal infection to people (known as *spillover*); [iii] sustained human–human transmission; [iv] outbreak containment. This temporal imaginary leverages a broad distinction between wild and domestic animals to naturalize spaces and human populations in the Global South as the ultimate progenitor for global pandemic threats and sites

where these threats must be contained. The fixity of these spaces and populations as the global locus of spillover risk in this narrative belies PREDICT scientists' failures to definitively fix them as the most significant spillover threat in their risk models.

USAID issued PREDICT in 2009 "with the goal of establishing a global early warning system for disease emergence that detects, tracks and predicts the emergence of new infectious diseases in high-risk wildlife ... that could pose a major threat to human health" (USAID 2009, 21). Over the next 11 years, USAID would funnel over \$200 million through PREDICT to develop scientific and state capacities in over 30 countries across Asia, Africa, and Latin America to identify where in the world animal viruses were most likely to spill over and infect people. It did this by building laboratory capacity, training scientists and government officials, testing animals and people for novel viruses, and modeling relative spillover risks across a range of contexts. PREDICT scientists ran over a million laboratory tests looking for novel viruses of concern over the 11 years the program ran (PREDICT Consortium 2020). At \$75 million, the first 5-year PREDICT grant was orders of magnitude larger than other public grants available to EID scientists at the time.<sup>2</sup> Historically, only the Rockefeller Foundation's 20-year effort to systematically catalog global arboviruses in the late 20th century likely compares or supersedes it in scope (Holmberg 2024). The Biden administration canceled DEEP VZN, PREDICT's intended successor program, in 2024 before scientists could begin testing samples (Willman 2023).

USAID developed PREDICT partially in response to the 2005 IHRs' new (and unfunded) mandate that all countries must be able to detect and report infectious disease outbreaks of international concern within their borders (Holmberg 2024). Start-up funding for the Emerging Pandemic Threats unit that operated PREDICT within USAID came from supplemental funding bills for the US-led Global War on Terror, separate from the regular USAID budget and outside the agency's "normal chain of command" (PREDICT practitioner, interview with author; Holmberg 2024).<sup>3</sup> The phrase "global health security" occurs over 70 times in PREDICT's final program report from 2020 (titled "PREDICT: Advancing Global Health Security at the Frontiers of Disease Emergence"). In contrast, language referencing "health equity," "equity," or "health inequalities" appears a mere three times. However, even with its explicit security focus, PREDICT also eventually fell casualty to the US government's intensifying redistribution of resources toward more spectacular modes of resource extraction, policing, imperial violence, and border security. While the program remained active through 2020, the Trump administration functionally froze much of PREDICT's funding in 2018 and formally announced the program's end in fall 2019 (McNeil Jr 2019).<sup>4</sup> These cuts came amid major EID events, including what was then the world's second-deadliest ever Ebola outbreak in Kivu (Maxmen 2020) and the onset of the COVID-19 pandemic. More recently, public health experts have condemned USAID's dismantling as a significant contributor to the late detection of another deadly Ebola outbreak in the DRC (Schreiber 2026). These cuts and their deadly consequences suggest a very real tension between trying to reconcile our responses to deadly new infectious disease outbreaks with the priorities of the same colonial and imperial

security apparatuses that often spur those outbreaks to begin with (see Ahuja 2016; Mitchell 2002; Raza Kolb 2020).

Geographic scholarship on biosecurity projects like PREDICT links efforts to secure desired ecological and epidemiological relations with (often racializing) projects of control over human bodies and territory. State actors figure centrally in much of this literature, relying on scientific expertise to develop and deploy key technologies and often working to enroll citizens in biosecurity practices (Barker 2010). Many of these studies link rhetoric about eliminating biological threats with violence against racialized and disabled people (i.e., Ahuja 2016; Braun 2007; Mansfield 2012; Patchin 2021; Prouse and Arefin 2026; White 2023). Others also highlight how species difference and violence against nonhumans articulates with violence against people in biosecurity (Ahuja 2016; Collard 2012a, 2012b; Kim 2015; Lunstrum et al. 2021; Plumwood 1995; Wallace 2009). Meanwhile, others have problematized how particular bodies are figured as unhealthy, risky (and often racialized) sites of global health intervention (i.e., Ahuja 2016; Benton 2016; Raza Kolb 2020; White 2023). Lioba Hirsch (2024, 2) indicts the international West African Ebola outbreak response as exemplary of how medical humanitarianism defaults to “treat[ing] Black bodies, not people.” In conversation with this work, I detail how PREDICT’s spillover risk assessments mobilized a broad distinction between domestic animals and wildlife to define non-Western geographies as a continuous global health security threat requiring intervention and control.

Ruth Wilson Gilmore (2002) describes violent control over abstracted, hierarchical human and spatial difference as a core feature of racism. She emphasizes how this selects dehumanized populations for premature death, calling racism a “death-dealing displacement of difference into hierarchies that organize relations between the planet’s sovereign territories” (Gilmore 2002, 16). Sylvia Wynter’s (2003) work connecting the development of racism to scientific descriptions of geographic and biological difference is particularly helpful for understanding how PREDICT’s spillover risk calculation reinforced racist hierarchies. She describes race as a secularized (naturalized) hierarchy that classifies different groups of people as more or less human in relation to a description of “Man” represented as universal (Wynter 2003). Wynter (2003) outlines how Europeans’ beliefs about the *places* they colonized starting in the 15th century were central to the development of ideas about race. With the invention of race, Europeans appealed to “natural law” to explain perceived differences between human populations (and their degree of assumed inferiority relative to a bourgeois Western ideal) and the geographies where people lived (Wynter 2003).

Others have elaborated on how racist discourses define white populations and spaces in contrast to nature, animals, and people of the global majority who they violently naturalize/animalize as closer to nature (Ahuja 2016; Belcourt 2015; Kim 2015). While PREDICT materials do not use explicitly racial terminology, this makes securing spillover risk within non-Western geographies and portraying these geographies (and populations within them) as naturally more diseased, closer to nature, and backward in time—as PREDICT did—as a racializing project. White and others emphasize that efforts

to “modernize” or improve the health of populations most affected by infectious diseases reify racist power differentials when they operate according to this logic (Ahuja 2016; Raza Kolb 2020; White 2023). Lioba Hirsch (2024, 6) emphasizes that the “traces of enslavement and colonialism” pervade international outbreak responses within global health in the present, defaulting to modes of care that degrade Black agency. Here I also follow scholars of racial capitalism like Gilmore (2002, 2007) and White (2023) in treating racism as co-constitutive with (and not reducible or merely auxiliary to) the capitalist drivers of disease emergence that critical geographers and disease ecologists have elaborated elsewhere (i.e., Chaves et al. 2022; Kaup and Austin 2025; Wallace and Wallace 2016).

The ways PREDICT deployed computational tools to translate data on viral disease ecologies into spillover risk metrics offer insight into how different variables became legible (and illegible) as potential drivers of zoonotic spillover risk. In this context, “risk” encompasses both the likelihood and severity of a potential spillover event (i.e., outbreak size, severity of health outcomes), calculated for the purpose of eliminating, minimizing, or mitigating it (Luhmann 2002; Reith 2004). While the design of some risk computations restricted them to locating spillover hotspots in parts of Africa, Asia, and Latin America where PREDICT worked, others presented findings that potentially complicated this geographic imaginary. Choices about what to include and exclude from risk analysis restrict the possible actions that risk analysis can potentially suggest, and this means risk assessments encode political values in their design. For example, anthropologist Joseph Dumit (2012) outlines how the US pharmaceutical industry has used its influence over the design of clinical trials to prioritize studies that assess how increasing pharmaceutical consumption at the population level can reduce health risks rather than those that investigate interventions that might reduce overall pharmaceutical intake (such as studies of taking patients off medications). When Dumit (2012, 208) argues that companies’ “valuing of clinical research in terms of how many pill prescriptions will result means that basically the only facts of health we have these days are about the value of more medicine,” this connects specific forms of risk calculation with a normalization of restricted health management options that benefits companies’ bottom line. Here, the prominence of scientific risk assessments examining some drivers of health and not others produces a form of common sense about health as defined through medicated risk reduction. This does not make these risk calculations wrong, but it does mean their design only makes them useful for informing a limited range of choices. It also can bolster discourse suggesting that the options risk calculations assess are the best or only ones.

In what follows, I trace how PREDICT scientists narrated their findings on spillover risk across models assessing genetic and ecological relations; evolution and ecological plasticity; biodiversity; and features of geography and demography. I start by outlining how PREDICT’s viral discovery studies repeatedly associated animal (and especially wildlife) contact with spillover risk. I then detail how PREDICT scientists refracted evidence of the substantial spillover risks associated with domestic animals back onto wildlife through assumptions that domestic animal viruses originate with wild animals. Finally, I outline how PREDICT’s spatial models located the risky wildlife contact it

blamed for starting outbreaks in non-Western geographies, explicitly linking ideas about wildlife outbreaks as “backwards” in epidemic time to an Orientalist spatial imaginary.

For this analysis, I examine PREDICT’s general risk assessments (18 total; across all virus families) and coronavirus papers (33 total; as exemplary of its numerous family-level studies). Although PREDICT scientists conducted viral discovery across over two dozen virus families, coronaviruses were the only viral family to receive priority for the duration of the program. They also comprised a significant fraction of the total tests PREDICT ran, and only paramyxoviruses<sup>5</sup> outnumbered coronaviruses when practitioners tallied the total number of novel viruses they found by family at the program’s end. Thus, although every viral family PREDICT studied has different ecologies, human histories, and geographies—and PREDICT’s findings on coronavirus diversity are therefore not interchangeable with its findings on, say, influenza or filovirus diversity—PREDICT’s coronavirus research helpfully spans the length of the program and represents a significant fraction of its findings on viral diversity.<sup>6</sup>

## 2 | Animal (Wildlife) Contact as Risk Factor

PREDICT’s models often treated contact with wildlife disease as a primary driver of human EIDs. While this scientific writing does not reference human difference, geographers and others have highlighted how pathologizing certain ways of living with and relating to animals readily serves racist and colonial power differentials between people (i.e., Ahuja 2016; Belcourt 2015; Chen 2012; Kim 2015). Ahuja (2016) emphasizes that the US government has long asserted political power through medical and sanitary interventions targeting people of the global majority through their interspecies relations. More generally, Hirsch (2024, 13) stresses that the “medical and health-related sciences” should always be understood as tools of political intervention within global public health contexts. With most wildlife diversity concentrated in the Global South, singling out human contact with wildlife as especially risky primed much of PREDICT’s science to reiterate Orientalist narratives about non-Western geographies as the global locus of spillover risk.

In PREDICT publications, wildlife contact first emerges as risky through phylogenetic measures of relatedness between novel viruses and known zoonotic viruses. Phylogenetics involves what many people outside biology might recognize as “family trees” showing how species have evolved to become different from one another over time. However, unlike a qualitative family tree that merely illustrates direct ancestors and indirect degrees of relatedness, phylogenetics quantifies the genetic change separating different taxa to identify closest relatives and/or likely ancestors. In many ways, phylogenetic models and diagrams are quantitative origin stories.

PREDICT’s coronavirus papers often highlight degrees of relatedness between novel viruses they find and MERS and SARS(-CoV-1 and -CoV-2). These discussions always also implicate the viral ecologies, geographies, and human activities most directly associated with them. For example, PREDICT conducted five studies of MERS virus origins and MERS-related CoVs in north Africa and southwest Asia (Alagaili et al. 2014;

Ali et al. 2017; Anthony, Gilardi, et al. 2017; Briese et al. 2014; Memish et al. 2013). In one study of MERS in Egyptian mammals, PREDICT scientists use evidence of widespread and genetically diverse MERS lineages in dromedary camels to argue for “the initiation of an active surveillance study on humans, particularly those that are at higher risk of exposure to MERS-CoV infections such as camel traders and abattoir workers” (Ali et al. 2017, 7). Genetic trees thus make legible a gradient of potential spillover risk in relation to known zoonotic coronaviruses. This relational risk gradient then implicates a wide range of people and animal hosts at points where they come into contact and marks them for intensified biosecurity via additional surveillance, sanitation, and PPE. To the extent that PREDICT only mapped phylogenies for viruses sampled from parts of Africa, Asia, and Latin America, the people and animals its phylogenies marked for surveillance and sanitizing interventions were inevitably restricted to these geographies. It naturalized these geographies as sites of pathogenic relations with dangerous viruses.

Measures of relatedness between viral host species also emerged repeatedly as risk factors for viral sharing and spillover. Most PREDICT studies of how viruses move between different host species emphasized mammals—a choice backed both by scientific consensus (given existing data) and genetic relatedness (since people are also mammals). One team from the EcoHealth Alliance (Olival et al. 2017) even identified phylogenetic proximity (relatedness) to people as the number one predictor of how many zoonotic viruses a mammal species could host. Emphasizing host species’ phylogenetic proximity to people as a predictor of zoonotic spillover risk matters because most mammal species—and especially the primates and great apes who share the most in common with us genetically—are not domesticated. This draws attention to contact with wild mammals (specifically) as a key threat and wildlife surveillance<sup>7</sup> as a central component of spillover mitigation. Implicit to this focus is also a geographic imaginary: the places where monkeys and great apes live as well as hotspots of mammalian biodiversity. In this spillover risk gradient, tropical non-Western geographies and the people who reside there emerge as a “natural” and relatively intractable source of infectious disease threat requiring continuous management for the sake of global health security.

PREDICT also frequently discussed biodiversity—of viruses and wildlife—as a key spillover risk. These discussions often centered on the risks posed by different kinds and arrangements of biodiversity. On the one hand, PREDICT scientists report far greater *total* viral diversity in wild animals (Bogich et al. 2012; Kreuder-Johnson et al. 2015). They also defend the program’s focus on wild animal viruses by noting that these “are responsible for nearly all recent pandemics” and have had increasing impacts globally over the last few decades (Allen et al. 2017). PREDICT scientists also often treat high viral biodiversity as a proxy for spillover risk, where they understand this diversity as increasing the probability that one of the animal viruses present may be able to infect people. In these analyses, the effects biodiversity has on viral evolution and ecological plasticity also play a key role in rendering wildlife contact “risky” and in need of intensified management.

For example, several PREDICT publications express concern over the spillover risks potentially generated by viral

recombination, which is when two different kinds of viruses infect the same animal and generate a completely new virus sharing genes from both predecessors. By combining traits from two viruses, recombination potentially allows a new virus to suddenly do things that neither parent could alone. It changes many parts of the viral genome all at once. In contrast, regular random mutations only change one (or a few) base pairs at a time. Scientists can identify evidence of past recombination events in a virus's evolutionary history using phylogenetics. Coronavirus studies conducted in Thailand and Cambodia during PREDICT's COVID-19 extension period consistently found evidence of past recombination events in wildlife coronaviruses related to SARS-CoV-2 (Delaune et al. 2021; Wacharapluesadee et al. 2021; Zhu et al. 2022). They and other PREDICT teams (Ge et al. 2013; Wacharapluesadee et al. 2015; Wells et al. 2021) underscore the capacity for recombination to suddenly increase the number of host species viruses can infect, increasing the risk of spillover into humans. In these studies, the risks posed by past and potential future recombination events almost always mandate more wildlife virus surveillance. Without work to connect ecological change to its political and economic contexts, these studies can readily portray molecular markers of spillover risk as “natural” features of pathogenic ecologies in need of containment, sanitation, and control.

Multiple PREDICT teams also identify “high host plasticity” as a key risk factor for zoonotic spillover (Johnson et al. 2020; Kreuder-Johnson et al. 2015; Olival et al. 2017).<sup>8</sup> Viral host plasticity is a property of viruses and measures a specific form of biodiversity: the “count of host orders and ecological groups” (i.e., both the genetic and ecological diversity of host species) that a given virus can infect (Kreuder-Johnson et al. 2015). It is a metric of how much a virus has been observed infecting different kinds of organisms—the extent to which its host range is *plastic*. To make sense of the finding that this property correlates with higher zoonotic spillover risk, PREDICT practitioners explain that “[h]uman practices facilitating heightened contact between taxonomically diverse animal hosts has likely facilitated selection of viruses with high host plasticity and sharing of zoonotic diseases” (Kreuder-Johnson et al. 2015, 4). In this narrative, plasticity therefore implicates particular “human practices” as risky. Managing this viral property then requires managing risky practices to produce ‘proper’ orderings of (and interactions with) animal biodiversity, including but not limited to: protecting wildlife habitat (i.e., parks); donning PPE; excluding wildlife from human homes, food, and drinking water; prohibiting wild game hunting; and controlling contact between wildlife and domestic species (especially high-intensity livestock production) (Johnson et al. 2020; Roth 2022).

Elsewhere, PREDICT practitioners detail how habitat loss and direct animal exploitation (i.e., poaching) that degrades biodiversity by reducing wild animal populations also increases how many viruses threatened mammal species tend to share with people (Johnson et al. 2020). They also note that in some contexts, biodiversity may have a protective effect against zoonotic spillover (Johnson et al. 2020). This highlights the key role people play in mediating the zoonotic spillover risk associated with biodiversity in PREDICT's scientific findings. Depending on how people *order* and manage it, high concentrations of animal

diversity can thus alternately exacerbate or protect against zoonotic spillover risk in different contexts.

Similarly, some of this work also implicates certain conservation practices in zoonotic disease emergence, especially those that trouble distinctions between wild and domestic space. Writing on the relationship between mammal populations and viral spillover risk, one PREDICT team found that threatened species for whom population reductions were directly observed (because they are so intensively monitored)—for example, species like sea otters or African elephants—hosted over twice as many zoonotic viruses on average compared to other IUCN-listed mammals. They note that “intensive and often hands-on wildlife management” as well as ecotourism “can increase opportunities for pathogen transmission from animals to humans” (Johnson et al. 2020, 5). In PREDICT findings and elsewhere, animal sanctuaries and zoos recur as sites with especially high microbial biodiversity and zoonotic disease risk. Technologies of liberal environmental governance momentarily become legible as drivers of spillover risk here.<sup>9</sup> Regardless, for PREDICT, recognizing these conservation spaces as risky requires adding more biosecurity rather than re-thinking securitized conservation practices: more surveillance, PPE, sanitation, and extra-careful management of human–animal contact. (Notably, discussions of wildlife trade as a source of zoonotic disease risk in PREDICT materials highlight pet trade and animal consumption rather than wildlife trade's well-documented connections to captive animal display and ecotourism.)<sup>10</sup> Biodiversity hotspots concentrated in Africa, Asia, and Latin America remain the assumed locus of spillover risk and site where it must be managed through intensified control techniques. By centering animal (especially wildlife) contact and omitting structural drivers of disease emergence from its risk assessments, PREDICT therefore presented geographies in the Global South and viruses, animals, and human behaviors within these geographies as the primary sites for intervention to prevent pandemic spillover.

### 3 | Placing Wildlife Viruses “Backwards” in Time

In their studies of risky animal contact, PREDICT scientists frequently grappled with the different kinds of risks posed by contact with wild versus domestic species. Elsewhere, geographers have highlighted how ideas about animals’ “wildness,” as contrasted to “domesticity,” shape their value as commodities and in conservation (i.e., Barua 2016; Collard 2020; Holmberg 2022). Here wildness signals something else as well: animals who not only belong to a different *place* than (certain) humans, but also to a different *time* (in multiple senses of the word). Ideas about epidemic time, where scientists understood domestic animal infections as inherently downstream from wildlife infections, played a key role in how PREDICT refracted the risks of domestic animal contact back onto wildlife. Meanwhile, popular media narratives about EIDs have long associated spillover with “primitive” spaces and practices locked “backwards” in developmental time relative to the so-called West (Wald 2008). In this scientific narrative, distinguishing wild and domestic animals therefore functions to help frame wildlife contact as backwards in both epidemic and developmental time from Western modernity.

This matters because PREDICT at times confronted evidence that domestic animal contact has substantial spillover risks. Using network analysis, PREDICT teams determined that domestic animal viruses can on average infect many more host species than wild animal viruses (Kreuder-Johnson et al. 2015), which usually only have the capacity to infect a single species (Pandit et al. 2022). They report elsewhere that only 12 species of domestic animals host half of *all* known zoonotic viruses (Johnson et al. 2020). Additionally, they highlight the “dubious non-domestication status” of the house mouse (*Mus musculus*) and black rat (*Rattus rattus*) as helping explain why these two species are “[t]he only wild animals among the top 10 species with detected zoonotic viruses” in their analysis (Johnson et al. 2020, 7). Even without considering structural drivers of disease emergence, these findings could implicate spaces where people live and work in close proximity to a small number of globally distributed species as concentrated sites of spillover risk. This differs drastically from the geographies produced by centering wildlife contact as the primary source of spillover risk. Findings on the risks domestic species pose therefore potentially challenge Orientalist narratives about spillover risk concentrated in the Global South. They also suggest different interventions, like regulation targeting pathogenic practices in the agriculture industry rather than surveillance and sanitation targeting rural people’s interactions with wildlife (Hinchliffe et al. 2024).

PREDICT practitioners offer several different narratives to explain these findings. Writing in 2015, Kreuder-Johnson et al. note how the “breeding and keeping of taxonomically diverse domesticated species in regular close contact with people for centuries” has selected for viruses especially prone to spillover into humans (Kreuder-Johnson et al. 2015, 5). They explain that “For the many viruses shared by wildlife and domestic animals, domesticated species play a critical role in facilitating direct contact with people, as well as amplification of disease transmission in intensive animal production facilities” (Kreuder-Johnson et al. 2015, 5). In this narrative, practitioners present spillover risk as distributed across wildlife and domestic species but amplified in alarming ways by the particular ecological conditions produced by domestication and especially industrial animal agriculture. According to this logic, the most effective way to reduce global spillover risk would be to eliminate or significantly restrict the activities of “intensive animal production facilities” that “play a critical role” in amplifying animal outbreaks and facilitating human contact with infected animals.

Five years later, a different PREDICT team framed the spillover risks of domestication in different terms. While Johnson et al. (2020, 7) find that 50% of known zoonotic viruses infect a small number of domestic species, they “postulate that wild mammals were the original host for the majority of [zoonotic] viruses, sharing viruses with domesticated species over centuries of coevolution and domestication.” What matters in this telling is not the extent to which different ecologies amplify spillover risk but which species might be considered the ultimate origin for viruses that eventually infect humans. This pattern recurs in other PREDICT publications as well. For example, despite extensive evidence (including from PREDICT studies [see Alagaili et al. 2014; Ali et al. 2017; Briese et al. 2014]) of MERS’ endemicity in domestic camels, PREDICT scientists describe MERS as

an EID of wildlife origin (based on the assumption that camels originally contracted it from bats [Anthony, Gilardi, et al. 2017]). PREDICT’s imagined spillover chronology means that findings on zoonotic risk from domestic species often still implicate wildlife contact. Johnson et al. (2020, 7) admit, for example, that “directionality in historical transmission of viruses between wild mammals and their domesticated kin can only be inferred.” The spillover chronology from wildlife to domestic species is assumed. Furthermore, the team finds that for primates, bats, and rodents (the primary host taxa PREDICT sampled), zoonotic viruses infecting them tend to infect humans directly without mediation by domestic species or other wildlife. Past findings on the significance of domestic species as amplifiers of wildlife virus spillover risk therefore do not apply to the animal taxa PREDICT targeted. Highlighting contact with different animals as drivers of global spillover risk implies different geographies of spillover risk and different interventions to manage, contain, or eliminate it. When Johnson et al. or other PREDICT teams refract domestic animal risk back onto wildlife contact, they thus (implicitly) shift the locus of infectious disease management from sites like global “intensive animal production facilities” to rural human–wildlife interfaces concentrated in the Global South.

#### 4 | Spatializing Spillover Risk From Wildlife Contact

However, PREDICT also made Orientalist spatial imaginaries explicit through efforts to map spillover risk and its geographic predictors. PREDICT scientists’ geographic models link ideas about inherently risky wildlife contact and epidemic time to a spatial imaginary that locates the risky contact responsible for starting outbreaks in non-Western geographies. The variables scientists chose (and chose not) to consider as potential spillover risk drivers played a central role in shaping what these models ultimately made legible as points of intervention to prevent pandemics. Disease ecologists, geographers, and others have long highlighted the role that factors like industrializing agriculture, concentrations and reconfigurations of land ownership in the Global South, and (neoliberal) economic and development policies play in amplifying spillover risk (i.e., Craddock and Hinchliffe 2015; Mitchell 2002; Wallace et al. 2015). This work links proximate drivers of disease emergence like land use change, demographic shifts, and biodiversity loss to structural inequalities and histories of colonial exploitation. PREDICT’s spatial risk analyses omit this context. Instead, they treat spillover risk as a purely technical problem of geography and demography (Mitchell 2002).

For example, PREDICT scientists often observed positive spatial correlations between mammalian biodiversity and zoonotic spillover risk. Overall biodiversity, especially among mammals, recurs as a core geographic predictor of EID risk in PREDICT analyses (Allen et al. 2017; Bogich et al. 2012; Johnson et al. 2020; Olival et al. 2017). In one early study, PREDICT leadership (Bogich et al. 2012, 613) argues that “The strong relationship between high wildlife host biodiversity—primarily found in low-latitude developing countries—and EID events caused by zoonotic pathogens from wildlife ... suggests that these geographic regions will continue to be a key source of novel EIDs

in the future.” This framing helps illustrate how fixating on the spillover risks posed by wildlife biodiversity helps naturalize “low-latitude developing countries” outside the West as an intractable hotspot for spillover risk: one that “will continue to be a key source of novel EIDs in the future.” Other PREDICT analyses specifically highlight bat biodiversity as a predictor of viral biodiversity (and by extension, spillover risk) (Anthony, Johnson, et al. 2017; Brierley et al. 2016). As a taxon, bats are also extraordinarily diverse, comprising more than one-fifth of all mammal species globally (Lacroix et al. 2017). This correlation between mammal diversity and viral diversity emerges from the fact that all mammal species have viruses adapted to infect them, and the overwhelming majority of viruses infect only one host. Here, wild animal biodiversity hotspots thus transmute via algorithm into hotspots for spillover risk—hotspots concentrated in Africa, Asia, and Latin America (see also Roth and Mitman 2024). Studies associating places with high wildlife diversity with higher spillover risk were one of the primary ways that discourse about wildlife contact mediated epidemic Orientalism under PREDICT.

PREDICT repeatedly framed land use and population as spatial proxies for wildlife contact driving spillover risk. Land use change associated with agricultural intensification, deforestation, mining, urbanization, and the construction of large infrastructure like dams recurs as a core driver of zoonotic spillover in PREDICT studies. In their study of global EID hotspots, Allen et al. (2017, 4) report that “EID events are best predicted by the distribution of tropical forested regions, higher mammalian species richness, and variables relating to shifts in agricultural land use; and appear to occur more often in tropical regions” (emphasis added). On its own, this reference to “shifts in agricultural land use” could potentially implicate a wide range of global geographies. However, in context it suggests that risks due to land use change are amplified where this change abuts “tropical forested regions” and/or occurs “in tropical regions.” So-called “tropical regions” are thus implicated at least twice as the source of especially risky land use change (arguably, three times via the reference to mammalian biodiversity). Though published 25 years later, this narrative recalls Richard Preston’s (1992) early writing in the *New Yorker* on disease emergence as “a natural consequence of the ruin of the tropical biosphere.”

PREDICT often treated population metrics as a proxy for human incursion into ecosystems and high-risk human–animal interfaces. For example, Brierley et al. (2016) find that human population density predicts the spatial distribution of spillover risk indicators in bats. From this they conclude, “Global risk patterns linking anthropogenic drivers and zoonotic viruses suggest that *as human populations continue to expand into pristine and potentially virus-rich habitat*, particularly in *tropical hotspots*, the threat of zoonoses will increase” (Brierley et al. 2016, 10, emphasis added). Spatial correlations again translate into a narrative about human bodies encroaching into “pristine,” biodiverse ecosystems concentrated in “tropical hotspots.” Elsewhere, Olival et al. (2017) find urbanization metrics more effective at predicting the spatial distribution of spillover risk than population size or density. However, they understand all these variables as potential “proxies for human–wildlife contact” (Olival et al. 2017, 653). Comments like “demographic growth remains a key point

for natural environment evolution, forest loss and dynamics of urban and suburban expansion” (Lacroix et al. 2017, 16) implicated in disease emergence—reiterating narratives of human population and urbanization as drivers of disease emergence—also recur in PREDICT publications that do not directly test the effects of these variables.

This again recalls a key narrative trope from early EID literature like Preston’s 1992 article that introduced the concept of EIDs to the general public for the first time (see Holmberg 2024). In this piece, Preston (1992, 48) quotes virologist Karl Johnson blaming spillover on “a human population explosion and a human invasion of tropical habitats,” where “There are just too many people entering too many ecosystems and violating them. People stumble into something and get sick.” While PREDICT’s publications couch this narrative in more scientific language, the message remains essentially unchanged decades later: excess human life boiling over and desecrating biodiverse ecosystems in the tropics. The way this discourse deploys narratives about population growth and land use change to fix non-Western geographies as the assumed source of spillover risk leaves little doubt to whose existence is assumed excessive, and by extension, a global health security threat.

At the same time, PREDICT teams frequently confronted the inherent indeterminacy of population metrics as stand-ins for complex, context-dependent socio-ecological processes and struggled with how to make sense of them. For example, Allen et al. (2017) identify human population density as the second-best predictor for the spatial distribution of EID events (after the distribution of tropical forests) in their preferred model. However, in their preferred model this correlation is negative: spillover risk *decreases* as human population density goes up! This presents a problem, as:

Theoretically, population has a baseline multiplicative effect on human disease events—of which EID events are a subclass—and their detection is modulated by reporting effort. Reporting effort appears to be associated with urbanization, but reporting effort and urbanization are also both products of human population.

(Allen et al. 2017, 6)

As indicated here, Allen et al.’s (2017) preferred model factors “reporting effort,”<sup>11</sup> a metric of how much effort researchers have exhausted studying infectious diseases in a given location, out of spillover risk. It assumes that in high-income regions like North America and Europe, a higher fraction of spillover events will be detected because policymakers and scientists devote more resources to studying infectious diseases there. Factoring reporting effort out of spillover risk supposes that EID events are under-reported in places with disproportionately fewer infectious disease studies. It results in down-weighting spillover risk in many Western countries and up-weighting spillover risk elsewhere. In the above passage, Allen et al. (2017) posit associations between reporting effort and urbanization as a possible explanation for why factoring

reporting effort *out* produces a supposedly nonsensical result where higher population density correlates to lower spillover risk. Forced to choose between leaving reporting bias in the model and leaving questions about a perplexing result unresolved, they choose the latter, explaining: “We did not attempt to fully disentangle these factors, instead using our measure of reporting effort to present a map of emerging disease hotspots with bias ‘factored out’” (Allen et al. 2017, 6).

Elsewhere, Olival et al. (2017, 648) admit that across different geospatial models they tried for predicting spillover risk from mammals, “A single global metric of human–wildlife contact” never emerged. However, for them, limits to what geospatial regression with population metrics can do simply indicate “the need for controlled field experiments and human behavioural risk studies to uncover the mechanisms underlying this risk” (Olival et al. 2017, 648). In an intriguing pivot, Bogich et al. (2012, 612) also discuss population density as one of “the strongest correlates with the supply of qualified medical staff, healthcare facilities, diagnostics, and treatments” that are critical to *reducing* infectious disease risk globally. This is despite the fact that they identify population density as a significant spillover risk driver and also frame it as an indicator of human incursion into ecosystems. “Population” can, apparently, indicate risky incursions into pristine ecosystems and the likely presence of robust health care infrastructure equally well. Finally, Brierley et al. (2016, 9) stress that their finding that human population density correlates with indicators of spillover risk in bats “should be interpreted as broad ecological associations rather than suggesting specific causal mechanisms.” They concede that “human population density could represent a variety of factors known to influence opportunity for transmission,” like travel, connectivity between regions, and processes like urbanization influencing viral richness (Brierley et al. 2016, 9). This reflection complicates an otherwise straightforward narrative about people stumbling into unspoiled tropical ecosystems and getting sick. In grappling over how to interpret their findings, Brierley et al. thus confront profound limits to what a metric of human bodies distributed over space can actually tell researchers about even proximate spillover risk drivers (much less context-specific and power-laden socio-ecological processes).

PREDICT’s focus on geography and demography as supposedly natural determinants of socio-ecological crisis outside the West has a long history in development (and USAID’s work specifically), predating the rollout of PREDICT by decades. Political scientist Timothy Mitchell (2002, 209) explains how during the mid-late 20th century, development actors like the World Bank and USAID deployed Orientalist narrative tropes and calculative techniques to reduce Egypt’s social, political, and economic problems to a depoliticized “problem of geography versus demography.” Through measurements of demographic change and “natural” limits to food production and land availability, they presented problems like poverty and food insecurity as “natural rather than political” so “questions of social inequality and powerlessness disappeared into the background” (Mitchell 2002, 221–222). Development actors could then safely attend to socio-economic issues as technical problems of efficient natural resource management without asking “at whose cost efficiencies were to be made, or in

whose hands control was to be strengthened” (Mitchell 2002, 230). Reports bolstered this framing with Orientalist narrative tropes touting imagery of an ancient Nile River delta and agricultural system fixed in time, crowded by “rapidly multiplying millions of inhabitants” (Mitchell 2002, 209). Mitchell (2002, 242–243) explains how this discourse serves to present Western development “as a detached center of rationality and intelligence” in contrast to the “non-West,” where:

The West possesses the expertise, technology, and management skills that the non-West is lacking. This lack is what has caused the problems of the non-West. Questions of power and inequality, whether on the global level of international grain markets, state subsidies, and the arms trade, or the more local level of landholding, food supplies, and income distribution, will nowhere be discussed.

Here Mitchell describes how development discourse defines the relationship between the West and non-West in technical, depoliticized terms that obscure questions of power. He emphasizes the special utility of population and land use metrics to this project. The features of development discourse that Mitchell describes bear an eerie resemblance to many aspects of PREDICT’s statements decades later about demography and geography driving disease emergence. As in Mitchell’s development discourse, PREDICT publications that examined land use and population—even where they acknowledge profound limits on what researchers can conclude from regressions presenting these variables as spillover drivers—skirt questions of power and inequality. Likewise, PREDICT publications often lean into Orientalist narrative tropes that frame risky population growth and land use change as “natural” features of non-Western tropical geographies. This discourse thus defines the so-called West in contrast to non-Western geographies via geographic and demographic indicators of spillover risk.

## 5 | Conclusions

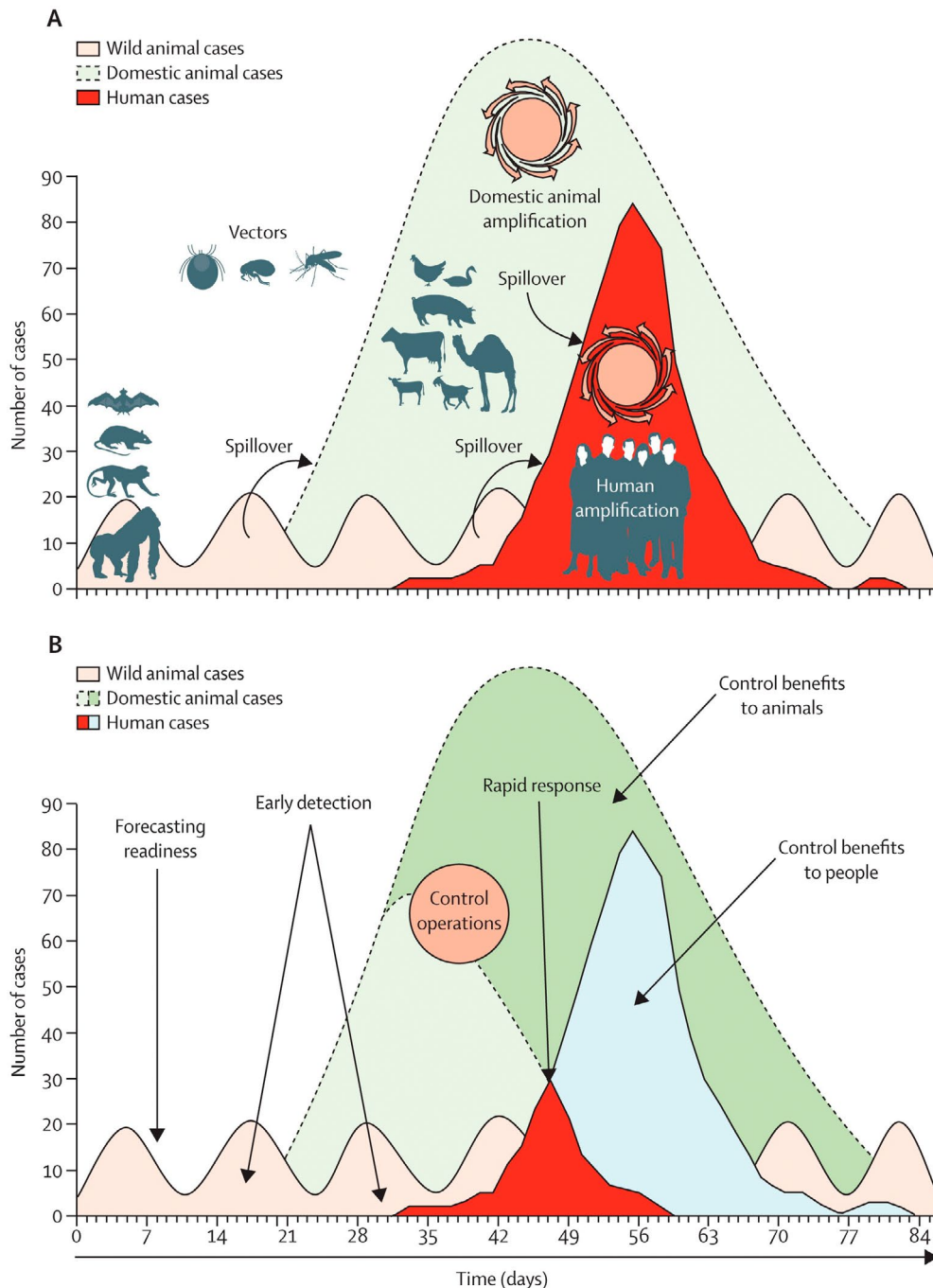
Across its various modeling approaches, PREDICT thus aimed to make targeted actions possible to prevent and mitigate zoonotic spillover. The design of many of these risk models was rooted in a viewpoint and set of discourses that historian Alexandre White calls epidemic Orientalism. PREDICT frequently articulated this geographic imaginary using discourse about wildlife contact as the ultimate progenitor for EIDs in people and domestic species and depoliticizing the drivers of ecological change. In a sense, containing and mitigating the spillover risks posed by wildlife contact became a sort of alibi for epidemic Orientalism under PREDICT. Discourses rooted in epidemic Orientalism define non-Western geographies through the infectious disease threats they pose to Western populations and global commerce. In EID science, epidemic Orientalism produces a kind of commonsense about spillover hotspots concentrated outside the West and constrains explanations about what drives spillover to proximate features of (human and nonhuman) populations and geography. This translates into policy recommendations targeting these populations and geographies for surveillance and sanitation rather

than addressing political or economic drivers of the shifting disease ecologies implicated in spillover risk.

In using PREDICT to examine how EID science discursively (re)produces epidemic Orientalism without explicitly naming human or geographic difference, I make two key theoretical interventions to critical geography. First, I elaborate on the role ideas about *epidemic time* play in (re)producing the spatial imaginaries of Orientalism and epidemic Orientalism in particular,

as illustrated in Figure 1, an infographic from a PREDICT *Lancet* publication (Karesh et al. 2012).

In this conception of epidemic time touted by PREDICT, viruses that cause human pandemics exist “later” in epidemic time relative to their wildlife virus progenitors. This in turn reinforces an Orientalist *spatial* imaginary of human–wildlife contact concentrated in the Global South as the primary origin for global pandemic threats. However, this conception of



**FIGURE 1** | Infographic from Karesh et al. (2012). Y-axes display hypothetical “number of cases” while X-axes display time in days, and each differently-colored curve represents infections of a hypothetical infectious disease in wildlife (pink), domestic animals (green), and humans (blue/red). Arrows in the top figure indicate spillover events between groups, and in the bottom figure they indicate potential points of early intervention and the resulting infections averted. (Reprinted from *The Lancet*, Volume 380, Issue 9857, W. Karesh, A. Dobson, J. Lloyd-Smith, et al., “Ecology of Zoonoses: Natural and Unnatural Histories”, Pages 1936–1945, © 2012 Elsevier Ltd., with permission from Elsevier).

epidemic time is not a given. Disease ecologists working in other contexts have long emphasized the threat infectious diseases originating with people and domestic animals pose to wildlife and centered more multidirectional conceptions of epidemic time (see AHEAD 2003; Sanderson 2004; Schwabe 1984). While the PREDICT illustration above accounts for multidirectional spread, it centers a conception of epidemic time where most human outbreaks ultimately originate with wildlife contact. Said (1978) and White (2023) both emphasize how Orientalism hinges on a definition of the so-called West as “modern” or “developed” and the rest of the world as “primitive” or “backwards” in developmental time by comparison. By scientific sleight of hand, PREDICT’s emerging disease science layers a second temporal imaginary onto this: naturalizing human pandemics as temporally downstream from wildlife infections. This scientifically coded narrative about outbreak progression simultaneously naturalizes and obscures the Orientalist spatial imaginary it encodes, buttressing older Orientalist temporal imaginaries of “modern” versus “primitive” regions and populations. Through a close reading of PREDICT’s scientific publications on novel coronaviruses and general spillover risk, I detail how the program naturalized this conception of epidemic time in its science—even as it confronted findings that potentially complicated or confounded Orientalist imaginaries of global spillover risk.

By detailing how distinctions between domestic and wild animals mediate epidemic Orientalism in PREDICT’s science, I also build on critical geographic insights about how the (re)production of nonhuman difference reinforces human power differentials. Collard and Dempsey (2017) argue that capitalist accumulation relies upon its ability to inscribe social difference on nonhuman nature so that it can exploit different species and populations in different ways. Kim (2015) highlights how racism similarly relies upon notions of species difference to violently dehumanize people through discursive associations between particular human and animal populations. Elsewhere, critical scholars have detailed how gendered, racial, and colonial violence against humans intersects with violence targeting particular animals for eradication or capitalist exploitation (i.e., Ahuja 2016; Belcourt 2015; Collard 2012a, 2012b; Gillespie 2014; Plumwood 1995). This includes analyses of how “wildness” and “domesticity” shape normative ideas about the spaces where different people and animals belong and how they should interact in shared spaces, with implications for colonial, gendered, and racial power differences between people (i.e., Belcourt 2015; Collard 2020; Haraway 2008). Building on these insights, I highlight how in EID science wildness not only marks animals as belonging to a different *place* than (certain) humans, but also to a different *time*.

In demonstrating how this wild/domestic animal distinction helps (re)produce Orientalist distinctions between human geographies and populations, I also illustrate how EID discourse that purports to define non-Western geographies, populations, and ecologies as pathological is (in some ways) not really about these people or places at all. Numerous scholars have detailed how biosecurity projects violently pathologize different groups of people from the global majority as sub/nonhuman to varying degrees through presumed associations with particular microbes or animals (i.e., Ahuja 2016; Kim 2015; Raza Kolb 2020;

White 2023). However, as Said (1978) emphasizes, Orientalism is first and foremost a self-definition by the West. Accordingly, PREDICT’s EID science leverages its distinction between wild and domestic species primarily in service to defining *Western* geographies and populations through their relative low-risk status.

When PREDICT portrayed wildlife contact as the ultimate progenitor for infectious diseases of people and domestic animals, it thus simultaneously displaced attention from the political economic drivers of pandemic spillover and the racist power differentials this science reinforced. In doing so, it framed ecological change as a purely technical problem of geography, demography, and “proper” conduct toward animals. When EID risk and ecological change are understood this way, controlling human disease means controlling contact with wildlife disease, and controlling contact with wildlife disease means controlling the land wildlife inhabit and human populations most proximate to them. This contrasts with approaches that might seek to address political or economic drivers of environmental degradation (like transnational debt or land dispossession). Under PREDICT, discourse on risk from contact with wildlife disease and human behaviors associated with this contact therefore played a crucial role in rendering non-Western geographies “backwards” in epidemic time, fixing them as the source of zoonotic spillover risk, and promoting interventions designed to secure ecologies in the Global South in the name of global health security. In other words, fixating on the spillover risks posed by wildlife contact in EID science buttresses human power differences that it also obscures.

Here, I want to emphasize that by problematizing PREDICT’s discursive framing of spillover risk, I am not attempting to assess the accuracy or rigor of the scientific work PREDICT scientists conducted. PREDICT findings about spillover risk factors like high host plasticity have the potential to productively inform a range of public health protections with different implications. At the same time, critical evolutionary biologists and disease ecologists elsewhere have explicitly linked their findings on proximate drivers of disease emergence to neoliberal policy changes and industrial agriculture expansion (e.g., see Gao et al. 2026; Sayseng and Chuang 2025). Others have begun deploying statistical tools to illustrate the utility of accounting for structural drivers of disease emergence in epidemiological models alongside the deep inadequacy of models which only consider proximate drivers (i.e., Koren and Chaves 2025). A different disease ecology has always been possible, and in some places, scientists (primarily based in the Global South, and almost certainly working with grants orders of magnitude smaller than PREDICT) are already conducting this work.

Relatedly, I am not suggesting that no one should devote resources toward mitigating EID risk in under-resourced communities. Access to high-quality PPE and sanitation, especially in healthcare and conservation settings where people engage in intensive forms of care, can save lives. People also often don’t *want* rodents, bats, or primates in their homes and workplaces, both because of disease risk and the mayhem these animals are notorious for causing. Understanding how different kinds of diseases spread between animals and people and how to prevent or mitigate it matters. However, this frontline work needs

to serve joint political, economic, and ecological changes that refuse the liberal environmental insistence that rolling out just a little more sanitation, more surveillance, and more *order* with the tools of One Health can indefinitely defer the threat of zoonotic outbreak.

PREDICT scientists attending carefully to their empirical findings often had to grapple with mutable microbial ecologies and indeterminacies in the meaning of model results that presented a much less seamless picture of the directionality of disease spread than the one above. From study to study, different PREDICT teams framed animals' domesticity and wildness differently in relation to zoonotic disease threat. While Kreuder-Johnson et al. (2015) note the significance of domestic animal populations as zoonotic disease amplifiers, others find reasons to discount the spillover risks domestic species pose relative to wildlife. Biodiversity and genetic relatedness (of viruses, of host species) recur as predictors of spillover risk in many PREDICT studies. However, in other cases attention to evolutionary processes like recombination and context-specific ecologies complicate straightforward narratives about risky genetic relations and diversity hotspots. Finally, researchers who tested population metrics as correlated with or driving spillover risk frequently had to confront the profound indeterminacy of what they could conclude from geospatial regressions on these variables. While researchers often still found ways to frame their findings that naturalized non-Western geographies as the global locus of spillover risk, these moments of indeterminacy matter. Even when the design of computations limits them in advance to asking which people, wildlife, and places in the Global South to target for spillover prevention, attention to context-specific ecologies, evolutionary change, and virus-host networks can still (at times) trouble Orientalist framings of spillover risk.

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## Conflicts of Interest

The author declares no conflicts of interest.

## Data Availability Statement

Research data are not shared.

## Endnotes

- <sup>1</sup> Not an acronym.
- <sup>2</sup> US government NIH and NSF grants averaged sums in the five-figure range over 5 years, for example.
- <sup>3</sup> Public policy expert Linda Bilmes (2018, 460) calls this US government funding strategy for the Global War on Terror the "ghost budget," as "Unlike the regular defense and domestic budgets, these types of appropriations were not subject to congressional budget caps; therefore Congress did not have to choose between war funding and other spending."
- <sup>4</sup> The details of the political intrigue and controversies that dogged PREDICT in its final years (and which continue to haunt its leadership) are outside the scope of this article. The FBI and Jeffrey Epstein both make cameos. People stalked and threatened one of my interviewee's grandkids. And no, overwhelmingly, the evidence does *not* indicate a PREDICT or any other laboratory leak-based origin for the COVID-19 pandemic (see Pekar et al. 2022; Worobey et al. 2022).
- <sup>5</sup> Paramyxoviruses include human pathogens like measles and mumps as well as zoonotic viruses like Nipah and Hendra virus.
- <sup>6</sup> In total, PREDICT scientists tested over 88,000 individual animals and people for coronaviruses, confirming positives in less than 5% and identifying 177 distinct known and unknown coronaviruses (PREDICT Consortium 2020, 47).
- <sup>7</sup> Surveilling wildlife viruses entails everything from sampling feces (minimally invasive) to darting and anaesthetizing animals to sample blood, saliva, and other tissues (highly invasive, requires a trained veterinarian) and then testing samples at a nearby laboratory.
- <sup>8</sup> Note that Olival et al. (2017) use the term "phylogenetic host breadth" rather than host plasticity.
- <sup>9</sup> By liberal environmental governance, I mean governance strategies designed to mitigate the environmental harms of capitalist extraction and pollution while also protecting (capitalist) markets and economic growth (O'Connor 1998; Polanyi 1944).
- <sup>10</sup> Although this may be partially explained by the high volume of wild animals poached for the pet trade and food, conservation organizations like zoos and aquariums (and industry groups representing them) that exhibit animals often downplay the permeability between their work and the for-profit wildlife trade (see Braverman 2013; Collard 2020; Holmberg 2022).
- <sup>11</sup> In this study, the authors measure reporting effort by how often a location is mentioned in biomedical literature on infectious disease outbreaks.

## References

- AHEAD. 2003. *AHEAD Invitees' Briefing Packet*. Animal Health for the Environment and Development.
- Ahuja, N. 2016. *Bioinsecurities: Disease Interventions, Empire, and the Government of Species*. Duke University Press.
- Alagaili, A., T. Briese, N. Mishra, et al. 2014. "Middle East Respiratory Syndrome Coronavirus Infection in Dromedary Camels in Saudi Arabia." *MBio* 5, no. 2: e00884-14. <https://doi.org/10.1128/mBio.00884-14>.
- Ali, M., R. El-Shesheny, A. Kande, et al. 2017. "Cross-Sectional Surveillance of Middle East Respiratory Syndrome Coronavirus

- (MERS-CoV) in Dromedary Camels and Other Mammals in Egypt, August 2015 to January 2016." *Eurosurveillance* 22, no. 11: 30487. <https://doi.org/10.2807/1560-7917.ES.2017.22.11.30487>.
- Allen, T., K. Murray, C. Zambrana-Torrel, et al. 2017. "Global Hotspots and Correlates of Emerging Zoonotic Diseases." *Nature Communications* 8: 1124. <https://doi.org/10.1038/s41467-017-00923-8>.
- Anthony, S., K. Gilardi, V. Menachery, et al. 2017. "Further Evidence for Bats as the Evolutionary Source of Middle East Respiratory Syndrome Coronavirus." *MBio* 8, no. 2: e00373-17. <https://doi.org/10.1128/mBio.00373-17>.
- Anthony, S., C. Johnson, D. Greig, et al. 2017. "Global Patterns in Coronavirus Diversity." *Virus Evolution* 3, no. 1: vex012. <https://doi.org/10.1093/ve/vex012>.
- Arnold, D. 1986. "Cholera and Colonialism in British India." *Past & Present* 113: 118–151.
- Barker, K. 2010. "Biosecure Citizenship: Politicising Symbiotic Associations and the Construction of Biological Threat." *Transactions of the Institute of British Geographers* 35, no. 3: 350–363. <https://doi.org/10.1111/j.1475-5661.2010.00386.x>.
- Barua, M. 2016. "Lively Commodities and Encounter Value." *Environment and Planning D: Society and Space* 34, no. 4: 725–744. <https://doi.org/10.1177/0263775815626420>.
- Belcourt, B. R. 2015. "Animal Bodies, Colonial Subjects: (Re)Locating Animality in Decolonial Thought." *Societies* 5, no. 1: 1–11. <https://doi.org/10.3390/soc5010001>.
- Benton, A. 2016. "Risky Business: Race, Nonequivalence and the Humanitarian Politics of Life." *Visual Anthropology* 29, no. 2: 187–203. <https://doi.org/10.1080/08949468.2016.1131523>.
- Bilmes, L. J. 2018. "The Fiscal Opacity Cycle: How America Hid the Costs of the Wars in Iraq and Afghanistan." In *Toward a Just Society: Joseph Stiglitz and Twenty-First Century Economics*, edited by M. Guzman, 457–478. Columbia University Press.
- Bogich, T., K. Olival, P. Hosseini, et al. 2012. "Using Mathematical Models in a Unified Approach to Predicting the Next Emerging Infectious Disease." In *New Directions in Conservation Medicine: Applied Cases in Ecological Health*, edited by A. A. A. guirre, R. S. Ostfield, and P. Daszak, 607–618. Oxford University Press.
- Braun, B. 2007. "Biopolitics and the Molecularization of Life." *Cultural Geographies* 14, no. 1: 6–28. <https://doi.org/10.1177/1474474007072817>.
- Braverman, I. 2013. *Zooland: The Institution of Captivity*. Stanford University Press.
- Brierley, L., M. Vonhof, K. Olival, et al. 2016. "Quantifying Global Drivers of Zoonotic Bat Viruses: A Process-Based Perspective." *American Naturalist* 187, no. 2: E53–E64. <https://doi.org/10.1086/684391>.
- Briese, T., N. Mishra, K. Jain, et al. 2014. "Middle East Respiratory Syndrome Coronavirus Quasispecies That Include Homologues of Human Isolates Revealed Through Whole-Genome Analysis and Virus Cultured From Dromedary Camels in Saudi Arabia." *MBio* 5, no. 3: e01146-14. <https://doi.org/10.1128/mBio.01146-14>.
- Chaves, L. F., J. Velasquez Runk, L. R. Bergmann, and N. L. Gottdenker. 2022. "Reifications in Disease Ecology 1: Demystifying Land Use Change in Pathogen Emergence." *Capitalism Nature Socialism* 34, no. 2: 23–39. <https://doi.org/10.1080/10455752.2022.2144397>.
- Chen, M. 2012. *Animacies: Biopolitics, Racial Matter, and Queer Affect*. Duke University Press.
- Collard, R. C. 2012a. "Cougar Figures, Gender, and the Performances of Predation." *Gender, Place and Culture* 19, no. 4: 518–540. <https://doi.org/10.1080/0966369X.2011.610179>.
- Collard, R. C. 2012b. "Cougar–Human Entanglements and the Biopolitical Un/Making of Safe Space." *Environment and Planning D: Society and Space* 30, no. 1: 23–42. <https://doi.org/10.1068/d19110>.
- Collard, R. C. 2020. *Animal Traffic: Lively Capital in the Global Exotic Pet Trade*. Duke University Press.
- Collard, R. C., and J. Dempsey. 2017. "Capitalist Natures in Five Orientations." *Capitalism Nature Socialism* 28, no. 1: 78–97. <https://doi.org/10.1080/10455752.2016.1202294>.
- Cooper, M. 2008. *Life as Surplus: Biotechnology and Capitalism in the Neoliberal Era*. University of Washington Press.
- Craddock, S., and S. Hinchliffe. 2015. "One World, One Health? Social Science Engagements With the One Health Agenda." *Social Science & Medicine* 129: 1–4. <https://doi.org/10.1016/j.socscimed.2014.11.016>.
- Delaune, D., V. Hul, E. Karlsson, et al. 2021. "A Novel SARS-CoV-2 Related Coronavirus in Bats From Cambodia." *Nature Communications* 12: 6563. <https://doi.org/10.1038/s41467-021-26809-4>.
- Downs, J. 2021. *Maladies of Empire: How Colonialism, Slavery, and War Transformed Medicine*. Harvard University Press.
- Dumit, J. 2012. *Drugs for Life: How Pharmaceutical Companies Define Our Health*. Duke University Press.
- Gao, Y., N. Añez, and L. F. Chaves. 2026. "High Spatial Resolution Ensemble Species Distribution Modeling of *Rhodnius prolixus*, Vector of Chagas Disease, in Western Venezuela." *GeoHealth* 10, no. 5: e2025GH001628. <https://doi.org/10.1029/2025GH001628>.
- Ge, X., J. Li, X. Yang, et al. 2013. "Isolation and Characterization of a Bat SARS-Like Coronavirus That Uses the ACE2 Receptor." *Nature* 503, no. 7477: 535–538. <https://doi.org/10.1038/nature12711>.
- Gillespie, K. 2014. "Sexualized Violence and the Gendered Commodification of the Animal Body in Pacific Northwest US Dairy Production." *Gender, Place and Culture* 21, no. 10: 1321–1337. <https://doi.org/10.1080/0966369X.2013.832665>.
- Gilmore, R. W. 2002. "Fatal Couplings of Power and Difference: Notes on Racism and Geography." *Professional Geographer* 54, no. 1: 15–24. <https://doi.org/10.1111/0033-0124.00310>.
- Gilmore, R. W. 2007. *Golden Gulag: Prisons, Surplus, Crisis, and Opposition in Globalizing California*. University of California Press.
- Haraway, D. J. 2008. *When Species Meet*. University of Minnesota Press.
- Hinchliffe, S., A. Blanchette, K. Chan, et al. 2024. "Understanding the Roles of Economy and Society in the Relative Risks of Zoonosis Emergence From Livestock." *Royal Society Open Science* 11, no. 7: 231709. <https://doi.org/10.1098/rsos.231709>.
- Hirsch, L. A. 2024. *Antiblackness and Global Health*. Pluto Press.
- Holmberg, M. 2022. "Constructing Captive Ecology at the Aquarium: Hierarchy, Care, Violence, and the Limits of Control." *Environment and Planning E: Nature and Space* 5, no. 2: 861–880. <https://doi.org/10.1177/25148486211014508>.
- Holmberg, M. 2024. "On Viral Terrain: The Shifting Imaginaries of Geography and Infectious Disease Risk in USAID's PREDICT." PhD thesis, University of British Columbia.
- Johnson, C., P. Hitchens, P. Pandit, et al. 2020. "Global Shifts in Mammalian Population Trends Reveal Key Predictors of Virus Spillover Risk." *Proceedings of the Royal Society B: Biological Sciences* 287: 20192736. <https://doi.org/10.1098/rspb.2019.2736>.
- Karesh, W., A. Dobson, J. Lloyd-Smith, et al. 2012. "Ecology of Zoonoses: Natural and Unnatural Histories." *Lancet* 380, no. 9857: 1936–1945. [https://doi.org/10.1016/S0140-6736\(12\)61678-X](https://doi.org/10.1016/S0140-6736(12)61678-X).
- Kaup, B. Z., and K. Austin. 2025. *The Pathogens of Finance: How Capitalism Breeds Vector-Borne Disease*. University of California Press.

- Kim, C. J. 2015. *Dangerous Crossings: Race, Species, and Nature in a Multicultural Age*. Cambridge University Press.
- Koren, O., and L. F. Chaves. 2025. "The Land-Use Land-Cover Change–Emerging Infectious Disease Nexus Reconsidered." *Bioscience*: biaf045. <https://doi.org/10.1093/biosci/biaf045>.
- Kreuder-Johnson, C., P. Hitchens, T. Smiley-Evans, et al. 2015. "Spillover and Pandemic Properties of Zoonotic Viruses With High Host Plasticity." *Scientific Reports* 5: 14830. <https://doi.org/10.1038/srep14830>.
- Lacroix, A., V. Duong, V. Hul, et al. 2017. "Genetic Diversity of Coronaviruses in Bats in Lao PDR and Cambodia." *Infection, Genetics and Evolution* 48: 10–18. <https://doi.org/10.1016/j.meegid.2016.11.029>.
- Lakoff, A. 2017. "A Fragile Assemblage: Mutant Bird Flu and the Limits of Risk Assessment." *Social Studies of Science* 47, no. 3: 376–397. <https://doi.org/10.1177/0306312716666420>.
- Luhmann, N. 2002. *Risk: A Sociological Theory*. Routledge.
- Lunstrum, E., N. Ahuja, B. Braun, R. Collard, P. J. Lopez, and R. W. Y. Wong. 2021. "More-Than-Human and Deeply Human Perspectives on COVID-19." *Antipode* 53, no. 5: 1503–1525. <https://doi.org/10.1111/anti.12730>.
- Mansfield, B. 2012. "Environmental Health as Biosecurity: 'Seafood Choices', Risk, and the Pregnant Woman as Threshold." *Annals of the Association of American Geographers* 102, no. 5: 969–976. <https://doi.org/10.1080/00045608.2012.657496>.
- Maxmen, A. 2020. "World's Second-Deadliest Ebola Outbreak Ends in Democratic Republic of the Congo." *Nature*, June 26. <https://doi.org/10.1038/d41586-020-01950-0>.
- McNeil, D. G., Jr. 2019. "Scientists Were Hunting for the Next Ebola. Now the US Has Cut Off Their Funding." *The New York Times*, October 25. <https://www.nytimes.com/2019/10/25/health/predict-usaid-virus-es.html>.
- Memish, Z., N. Mishra, K. Olival, et al. 2013. "Middle East Respiratory Syndrome Coronavirus in Bats, Saudi Arabia." *Emerging Infectious Diseases* 19, no. 11: 1819–1823. <https://doi.org/10.3201/eid1911.131172>.
- Mitchell, T. 2002. *Rule of Experts: Egypt, Techno-Politics, Modernity*. University of California Press.
- Morse, S. S., ed. 1993. *Emerging Viruses*. Oxford University Press.
- O'Connor, J. 1998. *Natural Causes: Essays in Ecological Marxism*. Guilford Press.
- Olival, K., P. Hosseini, C. Zambrana-Torrel, et al. 2017. "Host and Viral Traits Predict Zoonotic Spillover From Mammals." *Nature* 546, no. 7660: 646–650. <https://doi.org/10.1038/nature22975>.
- Pandit, P., S. Anthony, T. Goldstein, et al. 2022. "Predicting the Potential for Zoonotic Transmission and Host Associations for Novel Viruses." *Communications Biology* 5: 844. <https://doi.org/10.1038/s42003-022-03797-9>.
- Patchin, P. M. 2021. "For the Sake of the Child: The Economization of Reproduction in the Zika Public Health Emergency." *Transactions of the Institute of British Geographers* 46, no. 1: 2–14. <https://doi.org/10.1111/tran.12384>.
- Pekar, J., A. Magee, E. Parker, et al. 2022. "The Molecular Epidemiology of Multiple Zoonotic Origins of SARS-CoV-2." *Science* 377, no. 6609: 960–966. <https://doi.org/10.1126/science.abp8337>.
- Plumwood, V. 1995. "Human Vulnerability and the Experience of Being Prey." *Quadrant* 39, no. 3: 29–34.
- Polanyi, K. 1944. *The Great Transformation*. Beacon Press.
- PREDICT Consortium. 2020. *PREDICT: Advancing Global Health Security at the Frontiers of Disease Emergence*. One Health Institute, University of California. [https://ohi.vetmed.ucdavis.edu/sites/g/files/dgvnsk5251/files/inline-files/PREDICT%20LEGACY%20-%20FINAL%20FOR%20WEB%20-compressed\\_0.pdf](https://ohi.vetmed.ucdavis.edu/sites/g/files/dgvnsk5251/files/inline-files/PREDICT%20LEGACY%20-%20FINAL%20FOR%20WEB%20-compressed_0.pdf).
- Preston, R. 1992. "Crisis in the Hot Zone: Lessons From an Outbreak of Ebola." *The New Yorker*. October 26. <https://www.newyorker.com/magazine/1992/10/26/crisis-in-the-hot-zone>.
- Prouse, C., and M. R. Arefin. 2026. "Geographies of Biosecurity: Racialized Vulnerability, Colonial Extraction, and a New Critical Consciousness." *Progress in Human Geography* 50, no. 1: 63–82. <https://doi.org/10.1177/03091325251358410>.
- Raza Kolb, A. F. 2020. *Epidemic Empire: Colonialism, Contagion, and Terror, 1817–2020*. University of Chicago Press.
- Reith, G. 2004. "Uncertain Times: The Notion of 'Risk' and the Development of Modernity." *Time & Society* 13, no. 2–3: 383–402. <https://doi.org/10.1177/0961463X04045672>.
- Roth, E. 2022. "How to Live Safely With Bats? Ignorance(s) in Post-Ebola Risk Communication (Guinea, Sierra Leone)." *Sources: Materials & Fieldwork in African Studies* 4: 39–67. <https://doi.org/10.4000/11tar>.
- Roth, E., and G. Mitman. 2024. "The Value of Fragments: Making a Hotspot in Mount Nimba, Liberia." *Springs: The Rachel Carson Center Review*, June 13. <https://doi.org/10.5282/rcc-springs-6716>.
- Said, E. W. 1978. *Orientalism*. Random House.
- Sanderson, S. 2004. "Introduction—Dr. Steven Sanderson." Paper presented to "One World, One Health: Building Interdisciplinary Bridges", New York, September 29. [https://www.oneworldonehealth.org/sept2004/presentations/intro\\_sanderson.html](https://www.oneworldonehealth.org/sept2004/presentations/intro_sanderson.html).
- Sayseng, J. O., and T. Chuang. 2025. "Integrating Citizen Science and Remote Sensing Data to Identify Key Environmental Factors Influencing H5N1 Avian Influenza Virus Potential Spillover Risk in The Philippines." *GeoHealth* 9, no. 8: e2025GH001405. <https://doi.org/10.1029/2025GH001405>.
- Schreiber, M. 2026. "US Is 'Simply Choosing Not to Stop' Ebola Outbreak After Massive Public Health Cuts, Experts Say." *The Guardian*, May 21. <https://www.theguardian.com/world/2026/may/21/ebola-outbreak-public-health>.
- Schwabe, C. W. 1984. *Veterinary Medicine and Human Health*. 3rd ed. Williams & Wilkins.
- USAID. 2009. *PREDICT—Request for Applications (RFA)—RFA Number: USAID/M/OAA/GH/HSR-09-878*. Avian and Pandemic Influenza and Zoonotic Disease Program, US Agency for International Development. <https://grants.gov/search-results-detail/47965>.
- Wacharapluesadee, S., P. Duengkae, A. Rodpan, et al. 2015. "Diversity of Coronavirus in Bats From Eastern Thailand." *Virology Journal* 12: 57. <https://doi.org/10.1186/s12985-015-0289-1>.
- Wacharapluesadee, S., C. W. Tan, P. Maneeorn, et al. 2021. "Evidence for SARS-CoV-2 Related Coronaviruses Circulating in Bats and Pangolins in Southeast Asia." *Nature Communications* 12: 972. <https://doi.org/10.1038/s41467-021-21240-1>.
- Wald, P. 2008. *Contagious: Cultures, Carriers, and the Outbreak Narrative*. Duke University Press.
- Wallace, R. G. 2009. "Breeding Influenza: The Political Virology of Offshore Farming." *Antipode* 41, no. 5: 916–951. <https://doi.org/10.1111/j.1467-8330.2009.00702.x>.
- Wallace, R. G. 2016. *Big Farms Make Big Flu: Dispatches on Influenza, Agribusiness, and the Nature of Science*. NYU Press.
- Wallace, R. G., L. Bergmann, R. Kock, et al. 2015. "The Dawn of Structural One Health: A New Science Tracking Disease Emergence Along Circuits of Capital." *Social Science & Medicine* 129: 68–77. <https://doi.org/10.1016/j.socscimed.2014.09.047>.

Wallace, R. G., A. Liebman, L. F. Chaves, and R. Wallace. 2020. "COVID-19 and Circuits of Capital: New York to China and Back." *Monthly Review*, March 27. <https://monthlyreview.org/articles/covid-19-and-circuits-of-capital/>.

Wallace, R. G., and R. Wallace, eds. 2016. *Neoliberal Ebola: Modeling Disease Emergence From Finance to Forest and Farm*. Springer.

Wells, H., M. Letko, G. Lasso, et al. 2021. "The Evolutionary History of ACE2 Usage Within the Coronavirus Subgenus Sarbecovirus." *Virus Evolution* 7, no. 1: veab007. <https://doi.org/10.1093/ve/veab007>.

White, A. I. R. 2023. *Epidemic Orientalism: Race, Capital, and the Governance of Infectious Disease*. Stanford University Press.

Willman, D. 2023. "The US Quietly Terminates a Controversial \$125m Wildlife Virus Hunting Programme Amid Safety Fears." *BMJ* 382: 2002. <https://doi.org/10.1136/bmj.p2002>.

Worobey, M., J. Levy, L. Malpica Serrano, et al. 2022. "The Huanan Seafood Wholesale Market in Wuhan Was the Early Epicenter of the COVID-19 Pandemic." *Science* 377, no. 6609: 951–959. <https://doi.org/10.1126/science.abp8715>.

Wynter, S. 2003. "Unsettling the Coloniality of Being/Power/Truth/Freedom: Towards the Human, After Man, Its Overrepresentation—An Argument." *CR: The New Centennial Review* 3, no. 3: 257–337. <https://doi.org/10.1353/ncr.2004.0015>.

Zhu, F., V. Duong, X. Lim, et al. 2022. "Presence of Recombinant Bat Coronavirus GCCDC1 in Cambodian Bats." *Viruses* 14, no. 2: 176. <https://doi.org/10.3390/v14020176>.