Agnotology of virology: The origins of Covid-19 and the next zoonotic pandemic

LYLE FEARNLEY
Singapore University of Technology and Design, Singapore

Abstract

Global pandemic preparedness initiatives rely on a vision of viral discovery—to rapidly detect and respond to emerging viruses, or even predict their future emergence. But despite the fact that the emergence of a coronavirus was accurately predicted, and the new virus was also rapidly detected, viral discovery failed to prevent or contain the Covid-19 pandemic. In this paper, I trace the history of the viral discovery paradigm in order to illuminate its 'agnotology'—that is, what is ignored in the production of virological knowledge. I argue that this ignorance is primarily a question of scale: the massive expansion in molecular knowledge of viruses has not been matched by knowledge about the ecology of virus emergence.

Keywords: disease ecology; influenza; SARS-CoV-2; molecularisation; scale; laboratory

In December 2012, the British medical journal *The Lancet* published a paper written by leading researchers on zoonotic emerging diseases. The paper carried an unusually ambitious title: 'Prediction and prevention of the next pandemic zoonosis'. Despite the fact that 'no pathogens have been predicted before their first appearance', the authors argued that 'patterns' in the origin of novel pathogens, combined with advances in virological analysis, communications and computational tools, now 'promise[d] the possibility' of pandemic prediction. By focusing surveillance on high-risk 'hot spots'—primarily zones with intensive human–animal interactions; and by deploying new forms of pathogen surveillance, such as metagenomics—the authors heralded a new era of *viral discovery*.[1] Routinely detecting new viruses in animal reservoirs, such viral discovery programs would enable the development of countermeasures, such as vaccines, before even a single human fell sick. Future outbreaks could become not only rapidly detectable, but even predictable, enabling a shift in global health from *response* to outbreaks of disease toward *pre-emption* of pandemic emergence.

1 Stephen S. Morse et al., 'Prediction and prevention of the next pandemic zoonosis', *Lancet* 380, no. 9857 (1 December 2012): 1956–65, doi.org/10.1016/S0140-6736(12)61684-5.

Exactly seven years later, in December 2019, the Wuhan Institute of Virology in China identified a sample taken from a cluster of atypical severe pneumonia patients as a novel coronavirus. But within weeks of their discovery, the novel coronavirus—now known as SARS-CoV-2—had spread across China and beyond China's borders. All too quickly, the pandemic was upon us.

By now, we are used to narrating the Covid-19 pandemic as a dramatic failure of science and public health governance. But the rapid identification of the novel virus was in many ways a remarkable success for viral discovery. Or at least, a partial success—an incomplete victory so tragic that it brings to mind Cassandra's curse.

Building the global virome: 2011–20

In 2011, I met Dennis Carroll, director of USAID's PREDICT program and one of the authors of the *Lancet* paper, at the World Health Organization's China office just outside the East Gate of old Beijing. Carroll is a tall man with silver hair in a shoulder-length, impeccably neat cut, and presents himself as a sophisticated and cultured version of the scientist, more visionary than technician. In his talk at the WHO office, which sprinkled references to Gabriel García Márquez among the epidemic curves ('love in the time of pandemics'), he looked ahead to a future era of viral discovery.

'Think about HIV', he told us. By the time it was detected, HIV was already a global pandemic. Yet what is more terrifying is that 'were another disease like HIV to appear, we have no greater capability than at that time' to detect it. HIV is a disease in which the onset of symptoms is not seen for up to 10 years after infection, so there is a long period of invisible circulation. Though HIV is perhaps an extreme case, traditional public health surveillance is too slow for most emerging diseases—once a cluster is detected in the human population, Carroll argued, it is often too late to contain the outbreak. Global health needed to establish new forms of early warning that focused on surveillance of animals—that is, isolating and analysing the viruses circulating in animals *before* they spill over to humans.

In the following decade, the USAID PREDICT program, the EcoHealth Alliance, Metabiota and other research groups poured funds and resources into sampling viruses from animals, sequencing them, studying them and assessing their potential risk. Carroll himself spearheaded the work of a consortium called the 'Global Virome Project'—an effort to build transnational collaboration across these viral discovery programs in order to construct a map of the *global virome*—a 'global atlas of most of the planet's naturally occurring potentially zoonotic viruses'.[2]

2 Dennis Carroll, 'Building a global atlas of zoonotic viruses', *Bulletin of the World Health Organization* 96, no. 4 (2018): 292–4.

But as Carroll acknowledged in his talk that day in Beijing, such an effort could not realistically sample all viruses on the planet. Rather, it would begin by focusing viral discovery on certain high-risk geographical regions, certain high-risk animal species and certain high-risk virus families.

Where were these high-risk regions imagined to be? Along with tropical regions of South America, Africa and Southeast Asia, China soon came to the centre of global viral discovery programs. And in the aftermath of the SARS outbreak in 2003 and the highly pathogenic avian influenza (HPAI) strains after 1997, viral discovery work in China focused primarily on two types of viruses: influenzas and coronaviruses.

Due to a momentary affinity between global scientific interest in China and China's efforts to develop scientific prowess on the global stage, the results were significant. Between 2011 and 2016, viral discovery programs led by Chinese laboratories, including the Wuhan Institute of Virology, collected at least 15 novel coronaviruses from bats, including 11 isolated from a single bat cave in south China's Yunnan Province.[3] At the Wuhan Institute and its collaborating laboratories (both in China and the United States), these bat coronaviruses were sequenced, analysed and in some cases used in 'gain-of-function' experiments.[4] Based on their research, the scientists put forth something like a prediction: their research had 'revealed', they wrote in 2017:

> that various SARSr-CoVs [SARS-related coronaviruses] capable of using human ACE2 [an angiotensin-converting enzyme] are still circulating among bats in this region. Thus, the risk of spillover into people and emergence of a disease similar to SARS is possible.'[5]

Two years later, in December 2019, when a cluster of atypical pneumonia cases appeared at hospitals in Wuhan, a few samples were sent to the Wuhan Institute of Virology, where scientists rapidly sequenced the virus and conducted the same kind of bioinformatic analysis they showcased in the 2017 paper. Identifying the virus as a SARS-like coronavirus, they also showed that it had a 'probable bat origin', based on its similarity to other viruses in their collection.[6] The prediction that a coronavirus could emerge from bats into humans seemed to have come true, apparently vindicating the program of viral discovery.

3 Ben Hu et al., 'Discovery of a Rich Gene Pool of Bat SARS-Related Coronaviruses Provides New Insights into the Origin of SARS Coronavirus', *PLOS Pathogens* 13, no. 11 (30 November 2017): e1006698, doi.org/10.1371/journal.ppat.1006698.

4 On gain-of-function research, see Andrew Lakoff, 'A Fragile Assemblage: Mutant Bird Flu and the Limits of Risk Assessment', *Social Studies of Science* 47, no. 3 (June 2017): 376–97, doi.org/10.1177/0306312716666420.

5 Hu et al., 'Discovery of a Rich Gene Pool of Bat SARS-Related Coronaviruses Provides New Insights'.

6 Peng Zhou et al., 'A Pneumonia Outbreak Associated with a New Coronavirus of Probable Bat Origin', *Nature* 579, 3 February 2020, doi.org/10.1038/s41586-020-2012-7.

Unfortunately, however, this glimmer of success only highlighted the broader failure—for although they had very accurately predicted the next zoonotic pandemic, doing so did nothing to prevent it. In this paper, I draw on my archival research and fieldwork experiences to explore how this virological paradigm came to dominate pandemic preparedness; interrogate its obsession with discovering points of origin; and, finally, explore why, despite the incredible power of virological tools to reveal the reservoirs where viruses hide, the paradigm of viral discovery remained powerless to prevent the emergence of a pandemic when it arrived.

The origin of pandemic 'origins': The 1957 'Asian' flu

The roots of global programs to identify the origins of pandemics lie in the WHO's effort to build a worldwide surveillance system for pandemic influenza. In the 1940s, the WHO designed the World Influenza Programme, as it was then called,[7] as a worldwide network of laboratories that would be capable of isolating and identifying influenza viruses, and then sending reports and virus samples to the World Influenza Centre (WIC) at the National Institute for Medical Research in London. The idea, according to the Director of the WIC, C. H. Andrewes, was that:

> One might perhaps hope to isolate a strain from the beginning of an epidemic, adapt it to growth in fertile eggs and produce a vaccine in time to be of use before the epidemic is over. In practice, there is not nearly enough time to do this within one country. But if it could be shown that a new—and especially lethal—strain was spreading from country to country, the vaccine might be produced in time to protect countries yet unattacked.[8]

In May 1957, Dr J. H. Hale sent a telegram to the London WIC reporting an 'extensive' outbreak of influenza in colonial Singapore, and followed up by forwarding samples of the 'isolated strains'. Researchers in London identified a 'new variant' of Influenza A in the samples that 'appears to be so different from previous strains that existing vaccines would probably not give protection'.[9] People were unlikely to have any natural immunity to such a new variant, meaning the virus could spread quickly and widely—and would likely cause a global pandemic.

7 Today known as the Global Influenza Surveillance and Response System.

8 World Health Organization, *Minutes of the Fourth Session of the Interim Commission Held in Geneva from 30 August to 13 September 1947* (Official Records of the World Health Organization, 6) (New York and Geneva: World Health Organization, 1948), 194.

9 Memo to All Influenza Centres, 24 May 1957. ARC010-3, Centralized Files, 3rd Generation, Sub-fonds 3, Box I2-418-412. WHO Archives, Geneva. See similar account in World Health Organization, *Weekly Epidemiological Record* 22 (29 May 1957), 277.

Although the WHO did not succeed in developing vaccines quickly enough to prevent disease in any country, the monitoring of the pandemic as it spread across the world did create a clear geographical narrative of where the pandemic began, a narrative that lies behind the pandemic's colloquial name: 'Asian influenza'. Still, the precise origin remained a mystery, though anecdotal reports from Hong Kong suggested that the first cases may have been in southern mainland China, not in Singapore.[10] At China's borders, however, the World Influenza Programme reached the limits of its vision, despite its worldwide pretensions. In 1957, mainland China was not included within the World Influenza Programme, or indeed any WHO program, because the People's Republic of China was not a member of the United Nations. The Republic of China, based in Taiwan, retained the official China seat at the UN until 1972. As a result of Cold War geopolitics, no direct reports about influenza came from Beijing during the course of the 1957 pandemic.

In fact, although WHO officials did not know it for some time, Chinese scientists had discovered the outbreak and identified the novel variant virus around two months earlier. This was largely due to the scientific work of Dr Zhu Jiming (also known as Chu Chi-Ming). Zhu had trained at the University of Cambridge, then worked at the WIC in London from 1948 to 1950 alongside Andrewes. After the Communist revolution, Zhu returned to China, working first in Beijing and then in Changchun at the National Institute of Biological Products. In March 1957 a large outbreak of influenza broke out in Changchun, and Zhu and his laboratory promptly isolated several viruses from patients. They identified the samples as an Influenza A virus, but also found something surprising: in laboratory tests, they 'could not see any relation whatsoever between the new viruses and previous A type viruses'.[11] The 'queer variant', as Zhu called it,[12] was the most significant variation seen in the influenza virus since the 1946 A-Prime pandemic, suggesting a process of antigenic change much greater than could be explained by the annual accumulation of genetic errors through mutation. This was a significant finding: Robert Webster and W. Graeme Laver later remembered that 'the recognition that a major antigenic shift occurs in influenza viruses was first described by Dr. Chu' [Zhu], demonstrating for the first time the now familiar distinction between antigenic 'drift' (mutation) and 'shift' (genome segment reassortment) associated with the evolution of influenza viruses.[13] As A. M. Payne of the WHO acknowledged in a later paper:

10 World Health Organization, *Weekly Epidemiological Record* 19 (10 May 1957), 241; George Dehner, *Influenza: A Century of Science and Public Health Response* (Pittsburgh, PA: University of Pittsburgh Press, 2012), doi.org/10.2307/j.ctt6wrdfm.

11 Jiming Zhu, Xiao Jun, and Zhengzhang Hao (朱既明， 萧俊， 郝成章), 1957 年流行性感冒流行的病毒类型和性状 I . 长春分离的病毒,《科学通报, 30 June 1957, 374.

12 Chi-Ming Chu (Zhu Jiming), 'The Etiology and Epidemiology of Influenza: An Analysis of the 1957 Epidemic', *Journal of Hygiene, Epidemiology, Microbiology, and Immunology* 2, no. 1 (1958): 1–8. A note on the last page indicates the article was received by the journal on 2 December 1957.

13 W. Graeme Laver and Robert G. Webster, 'In Memoriam: Chu Chi Ming (1917–1998)', *Virology* 255, no. 1 (1999): 1, doi.org/10.1006/viro.1998.9551.

> It is clear that [the Chinese researchers] recognized most of the important features of the virus which have since been described elsewhere. It is unfortunate that this information did not reach the rest of the world until the epidemic was already spiraling widely. If it had we would have had two more months to prepare.[14]

But answering the question of the geographical origin of the new influenza virus merely raised new questions. In a paper published in 1958 in a Soviet-bloc Czechoslovakian medical journal, Zhu not only specified that the 'new virus actually originated in China', but also raised a critical question: 'How and why', he asked, did such a variant arise? The answer, Zhu reminded the readers, could not be gradual mutation: the antigenic change in the new variant strain was much more than could possibly occur through mutation. Instead, he suspected an 'unexpected animal reservoir as the origin of this queer variant'.[15] If flu pandemics began when viruses escaped animal reservoirs and into human bodies, then there was something about the *ecology* of human–animal relations that drove pandemic emergence.

The molecularisation of disease ecology

Since the 1960s, research on the origin of influenza pandemics has centred on the hypothesis that new viruses originate from animal reservoirs. Sampling and experimental research efforts have aimed to pinpoint the precise 'zoonotic diagram'[16] of relations between species that results in the periodic emergence of new pandemic strains. Yet precisely as the 'ecology of influenza' became the dominant conceptual framework for understanding the origins of influenza pandemics, scientists experimentally defined this ecology at a molecular scale.

In her recent work on epigenetics, Margaret Lock has described the 'molecularization of the environment'. She writes that epigenetics research enacts something of a paradox: while on the one hand epigenetic findings are demanding that science move away from genetic reductionism towards 'inquiry into the way in which the biology of living entities is continually modified … by environmental stimuli', at the same time this 'environment' is experimentally defined at a miniaturised scale of molecular pathways—effectively bracketing out the role of broader historical, political and sociocultural environments. As she glosses it, '"nurture" is in effect miniaturized and molecularized for the purposes of this research'.[17]

14 A. M. Payne, 'Some Aspects of the Epidemiology of the 1957 Influenza Pandemic', *Proceedings of the Royal Society of Medicine* 51, no. 1009 (1958), doi.org/10.1177/003591575805101205.

15 Chi-Ming Chu (Zhu Jiming), 'The Etiology and Epidemiology of Influenza', 1–8.

16 Christos Lynteris, 'Zoonotic Diagrams: Mastering and Unsettling Human–Animal Relations', *Journal of the Royal Anthropological Institute* 23, no. 3 (2017): 463–85, doi.org/10.1111/1467-9655.12649.

17 Margaret Lock, 'Comprehending the body in the era of the epigenome', *Current Anthropology* 56, no. 2 (April 2015), 151–77, doi.org/10.1086/680350.

What happened in influenza research is similar. Consider the most important researcher on the animal origins of influenza since the 1970s—Robert Webster. His experiments aimed to experimentally demonstrate how antigenic 'shift' could happen—the large antigenic change that Zhu Jiming had discovered, and that Zhu linked to the emergence of flu pandemics. Webster infected one pig with a 'human' influenza virus (Hong Kong virus), another with a 'swine' influenza virus, and then held the infected pigs in a confined area with several uninfected 'contact pigs'. After a week, four 'contact pigs' were not only infected with the two parent viruses, but also infected with *new* antigenic hybrid variants, or mixtures of the two parent viruses—the viruses had **reassorted** inside the pigs.[18] As the scientists concluded:

> these studies provide evidence that recombination between influenza viruses from man, lower animals and birds can occur *in vivo* under conditions of natural transmission and give rise to 'new' influenza viruses[19]

Webster and his colleagues drew particular attention to the role of the pig as a 'mixing vessel' for multiple flu strains.[20]

Once Webster's hypothesis was confirmed in laboratory experiments, others translated the laboratory back onto China's landscapes.[21] In a 1982 paper reporting from a conference on the ecology of influenza, Kennedy Shortridge and C. H. Stuart-Harris cite Webster's findings and point out that 'the densely populated, intensively farmed area of southern China adjacent to Hong Kong is an ideal place for events such as interchange of viruses between host species'. Drawing on anecdotal reports and a couple of cursory visits in rural areas of south China, Shortridge and Stuart-Harris suggested that the experimental conditions created in Webster's laboratory were the actual landscapes of southern China:

> In the villages, it is common to see ducks, geese, and chickens running loose in proximity to pigs and water buffaloes and to see small children playing in this environment. The closeness between man and animals could provide an ecosystem for the interaction of their viruses.

Shortridge and Stuart-Harris proposed that southern China could be the 'epicentre' of influenza pandemics.[22]

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18 For example, R. G. Webster and C. H. Campbell, 'The in vivo production of "new" influenza A viruses: II. In vivo isolation of "new" viruses', *Virology* 48, no. 2 (May 1972): 528–36, doi.org/10.1016/0042-6822(72)90063-3.

19 R. G. Webster, C. H. Campbell and A. Granoff, 'The "in vivo" production of "new" influenza viruses: III. Isolation of recombinant influenza viruses under simulated conditions of natural transmission', *Virology* 51, no. 1 (January 1973):149–62, doi.org/10.1016/0042-6822(73)90375-9.

20 R. G. Webster et al., 'Evolution and ecology of influenza A viruses', *Microbiological Reviews* 56, no. 1 (March 1992): 152–79, doi.org/10.1128/MMBR.56.1.152-179.1992.

21 See Bruno Latour on Louis Pasteur's translations between laboratory and farm settings: Bruno Latour, 'Give Me a Laboratory and I Will Raise the World', in *Science Studies Reader*, ed. Mario Biagioli (London: Routledge, 1999): 258–75; Bruno Latour, *The pasteurization of France* (Cambridge, MA: Harvard University Press, 1988).

22 Kennedy Shortridge and C. H. Stuart-Harris, 'An Influenza Epicentre?', *Lancet* 8302 (9 October 1982): 812–13, doi.org/10.1016/S0140-6736(82)92693-9.

And yet, what is missing from all of this is any inquiry into the actual ecology of host animals, their relationships to each other and to humans, and how these too might be 'new'—changing over time as agricultural technologies advance, as economies develop and as consumption habits change. To adapt a distinction made by the historian Warwick Anderson, research on the zoonotic origins of pandemics adopted ecology as more of a 'metaphor' than an 'analytic';[23] taking the point of view of the virus, it focused attention on parasite–host relations, but ignored the landscape-scale changes that could reshape the range and interactions of the host animals themselves—including, of course, potential human hosts.

The agnotology of virology

As influenza research became the paradigm for the studies of 'emerging diseases' after the 1990s, programs of viral discovery and analysis were extended to a range of new viral threats from Ebola to Henipaviruses to SARS coronaviruses. New tools from molecular biology, genomics and bioinformatics offered ever more precise understandings of the interior structures of these viruses, while infrastructure for sampling and analysing viruses was extended to more and more 'hotspots' of viral emergence, culminating in the development of programs like the Global Virome Project.

With this background in mind, it is not surprising that the molecular-scale approach to disease ecology once again dominated efforts to understand the origins of the pandemic in the response to the outbreak of Covid-19. For example, when the Wuhan Institute of Virology reported the results of their sequencing work on SARS-CoV-2, they argued that the virus had a 'probable bat origin'.[24] Using phylogenetic methods, the Institute compared the sequence of the new virus with an archive of previously sampled and sequenced coronaviruses and showed that the new strain most closely resembled a virus they had isolated from a bat in Yunnan. In other words, the genetic relatedness between viruses was taken as evidence for a zoonotic relation between bats and humans: viral phylogeny standing for interspecies spillover. Others using similar methods have proposed snakes, Malayan pangolins or mink as possible ecological sources of SARS-CoV-2.

An important rationale for the phylogenetic search for origins is the prediction and prevention of the *next* pandemic. As Peter Embarek, who led the WHO's mission to Wuhan in search of the origin of the coronavirus, explained: 'if we understand how this one jumped from bats origin into humans, we can perhaps prevent similar

23 Warwick Anderson, 'Natural Histories of Infectious Disease: Ecological Vision in Twentieth-Century Biomedical Science', *Osiris* 19 (2004): 39–61, doi.org/10.1086/649393.

24 Peng Zhou et al., 'A Pneumonia Outbreak Associated with a New Coronavirus of Probable Bat Origin', *Nature* 579 (3 February 2020), doi.org/10.1038/s41586-020-2012-7.

events in the future. To prevent similar pandemics'.[25] Thus far, however, the impact of the virological search for origins is ambiguous at best. In the infamous posts purporting to depict the consumption of 'bat soup' in China that went viral in early 2020, images of a Chinese woman eating a bat were linked with the research published by the Wuhan Institute claiming the virus had a 'probable bat origin'.[26] In a less Orientalist, but perhaps no less ideological extension of scientific facts beyond what they warrant,[27] 19 Chinese academicians later called for a ban on the consumption of all wild animals, based on evidence that the virus came from a wildlife reservoir. Several months later, China banned all farming of wild animals for food consumption, potentially putting an estimated 14 million people who breed wildlife out of business, including many who previously bred species that are not considered significant zoonotic risks (such as frogs).[28] Yet there remains no evidence that farmed wild animals played any role in the outbreak, nor that consumption of wildlife was responsible for viral spillover. Rather than improving global capacity to prevent the next pandemic, virological claims about bat origins seem to have mostly been adopted in the service of a variety of geographies of blame.[29]

My point is not that virological claims about zoonotic emergence are directly responsible for the spread of false or unproven accusations. Rather, I want to draw attention to what is absent from these scientific studies, or what could be called the agnotology of virology. As defined by Robert Proctor, agnotology is the study of ignorance, based on the premise that ignorance is 'more than a void', and that ignorance is *produced*. In this case, the very power and success of the virus laboratory produced ignorance because of the 'selective' nature of its attention.[30] By defining disease ecology at a molecular scale, enormous advances were made in understanding the structure of viral pathogens and their phylogenetic relationships. Thousands of papers have been published on the SARS-CoV-2 genome, many hoping to infer from this molecular data something about how the virus emerged. Yet even basic

25 World Health Organization, 'Episode #21: Covid-19: Origins of the SARS Cov-2 virus', *Science in 5*, 14 January 2021, www.who.int/emergencies/diseases/novel-coronavirus-2019/media-resources/science-in-5/episode-21---covid-19---origins-of-the-sars-cov-2-virus, accessed 24 September 2021.

26 The full text of the post reads: 'A woman eats a whole bat in disturbing footage which has recently surfaced on the internet, as scientists link the deadly coronavirus to the flying mammals. Bat soup is a popular dish in #Wuhan, #China where the virus originated.' In reality, the image shows travel show host Mengyun Wang eating a local dish served on the island of Palau in the western Pacific.

27 On scientific ideology, see Georges Canguilhem, 'The History of Science', in *A Vital Rationalist: Selected Writings from Georges Canguilhem*, ed. Francois Delaporte (New York: Zone, 1994), 36; and my discussion of Canguilhem in *Virulent Zones*, Chapter 2.

28 However, several animal species were reclassified as 'special-type livestock', rather than wild animals, and permitted for food farming. In addition, farming of wild animals for fur, medicine and other products was still permitted.

29 Paul Farmer, *Aids and Accusation: Haiti and the Geography of Blame* (Berkeley, CA: University of Calfornia Press, 1992).

30 Robert Proctor and Londa L. Schiebinger, *Agnotology: The making and unmaking of ignorance* (Stanford, CA: Stanford University Press, 2008), 3, refer to this as 'ignorance as *lost realm*'.

questions about the migratory ranges of different bat species, or the supply chains and farming system for animal meats and other products in China, are rarely even asked, let alone answered.

In my book *Virulent Zones*, I highlighted the potential offered by 'nonvirological' approaches to the study of pandemics, gathering around terms like 'One Health' and 'planetary health', and involving a broader range of disciplines from veterinarians to ecologists to data scientists.[31] As the ecologically minded team I followed in my fieldwork explained, predictions or forecasts based on viral discovery alone are often poorly equipped to monitor the 'gradual changes in anthropogenic, environmental and wildlife factors' that may drive new viruses to emerge.[32] Viral discovery tells us a lot about how viruses mutate and reassort, but not enough about the 'mutations' and 'reassortments' of livestock economies, rural–urban ecosystems and cultural practices that determine where and when viruses spill over into human populations.[33] Amidst the agnotology of virology, we have achieved greater and greater precision in the understanding of the global virome, but this molecular knowledge failed to prevent a pandemic that viral discovery programs accurately predicted. Unlike Cassandra, the reason virologists have failed to translate prediction into prevention is not because their claims do not command belief. Rather, the vision of viral discovery may be zoomed to the wrong scale.

31 Lyle Fearnley, *Virulent Zones: Animal Disease and Global Health at China's Pandemic Epicenter* (Durham, NC: Duke University Press, 2020), doi.org/10.1515/9781478012580.

32 Marius Gilbert, Xiangming Xiao and Timothy P. Robinson, 'Intensifying Poultry Production Systems and the Emergence of Avian Influenza in China: A "One Health/Ecohealth" Epitome', *Archives of Public Health* 75, no. 1 (27 November 2017): 48, doi.org/10.1186/s13690-017-0218-4.

33 For other anthropological calls along these lines, see Hannah Brown and Ann H. Kelly, 'Material Proximities and Hotspots: Toward an Anthropology of Viral Hemorrhagic Fevers: Material Proximities and Hotspots', *Medical Anthropology Quarterly* 28, no. 2 (June 2014): 280–303, doi.org/10.1111/maq.12092; Frédéric Keck, *Avian Reservoirs: Virus Hunters and Birdwatchers in Chinese Sentinel Posts* (Durham, NC: Duke University Press, 2020), doi.org/10.1215/9781478007555; Celia Lowe, 'Viral Clouds: Becoming H5N1 in Indonesia', *Cultural Anthropology* 25, no. 4 (2010): 625–49, doi.org/10.1111/j.1548-1360.2010.01072.x.

This text is taken from *International Review of Environmental History, Volume 8, Issue 1, 2022*, edited by James Beattie and Brett Bennett, published 2022 by ANU Press, The Australian National University, Canberra, Australia.

doi.org/10.22459/IREH.08.01.2022.08