3. What factors led to the emergence of the

Coronavirus pandemic?

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Introduction

In this essay, I show that several factors led to the emergence of the Coronavirus pandemic including biological, evolutionary, human behavioural and societal factors. The word "factor", which seems ubiquitous, means "anything that contributes to a result or has a causal relationship to a phenomenon" (APA, 2023). Different interpretations of what could be considered a factor in emergence are possible. I begin this essay by examining the notion and models of emergence. Next, I introduce the factor of pandemic potential of the causative agent of the pandemic, *severe acute respiratory syndrome coronavirus 2* (SARS-COV-2). I examine the factors influencing its evolution and localised emergence in the context of human land use, environmental change, and associated biodiversity change. I suggest evidence that SARS-COV-2 may have emerged in humans far earlier and in a different context than has been suggested. I next consider the number of factors leading to the amplification of the virus' spread. Once a pathogen of pandemic potential enters a modern urban population, its global spread is almost predictable through pre-existing capital flows (Wallace, et al., 2015). I suggest that a holistic re-framing of pandemic emergence is necessary to include the structural patterns underlying the global spread of the disease.

Pandemic Emergence: Models and Challenges

The classic definition of a pandemic is "an epidemic occurring worldwide, or over a very wide area, crossing international boundaries and usually affecting a large number of people" (Last, 2001: cited in Kelly, 2011). An epidemic is an unexpected increase in occurrences of a disease in a specific

geographical area. Morse (1995, p. 7) suggested infectious disease emergence is a two-step process. The first is "introduction", defined as the point at which a population first becomes exposed to a pathogen (and vice versa). The second is "adoption", the further dissemination of the pathogen within the population. Expanding upon that, Morse, et al. (2012) suggest stages leading to pandemic emergence. First, "pre-emergence" or the latency phase is defined as local or undetected spread (in humans or animals). This is characterised by primary infection, the first human to be infected with the disease, as well as potential stochastic or invisible spread of the infection (Frutos, et al., 2020). Secondly, "localised emergence" occurs with an initial outbreak (a rise in disease cases) becomes a local epidemic. Between pre-emergence and localised emergence is the "index case", the first person to be to contract the disease caused by the pathogen. The index case occurs later than the primary infection, and the first identified cases are later still. An "amplification cycle" (Frutos, et al., 2020, p. 1), in which individuals who may or may not present disease symptoms disseminate the disease in the local area, typically in densely populated environments. Third, "global emergence" or "pandemic emergence" occurs with further spread until the definition of a pandemic is met.

This question relates to the facts or situations that "led to the emergence of the Coronavirus pandemic". To be clear, I consider that the situations and possible pathways to the evolution and introduction of the pathogen, the adoption of the pathogen, and pathways to local and global emergence are all separate factors that "led to" the emergence of the pandemic. Some might interpret that this should only include factors leading to the pre-emergence stage as "pandemic emergence" might be seen to have already started by the time of the pathogen emergence. However, the simple fact that not all pathogens of pandemic potential become pandemics demonstrates that the biological processes and influences thereupon are fundamental factors leading to the emergence of a pandemic.

All models of emergence present a common temporal sequence of events rather than a specific point in time, which raises the question of when the pandemic can be considered to have emerged. One could align such a threshold with the first or any arbitrary number of SARS-CoV-2 positive individuals who acquired the infection outside of its initial outbreak area, being either Wuhan or China. The first confirmed case beyond China's border was detected in Thailand on the 8th of January 2020, but in retrospect is likely to have been earlier. Roberts et al. (2021) use a method of Optimal Linear Estimation, usually used in conservation modelling and phylogenetics, to assess a dataset of known infection dates and predict the likelihood of them being recorded late. They found the virus had spread globally beyond China by January 2020 and that the index case likely occurred by the 17th of November 2019. This is notably not the same as the primary infection. Ultimately, the most salient reference point is the 11th of March 2020, the date of the World Health Organization (WHO) pandemic declaration.

Besides the ambiguities encountered when attempting to reconcile events from models to their corresponding realities, I suggest further criticism of this notion of pandemic emergence. Pandemics, as Morens et al., (2009) articulate, have mostly been used in the context of studying historical outbreaks. The notion of an "ongoing pandemic" has become something of a construction, intended to raise awareness. Contemporary emergence models are essentially created to inform the prevention and control strategies of predominantly Western governments, with public health systems and the means to instigate surveillance, testing, and seroprevalence to establish the source of the outbreak. While the term pandemic, *pan* meaning all, implies universality, experiences of the pandemic, and levels of preparedness have not been universal. It could be argued that the Coronavirus has not been a pandemic, but a syndemic (Horton, 2020). The global burden of preexisting conditions such as obesity and other diseases, as well as food insecurity and poor healthcare systems which lead to differential disease outcomes, is not shared equally. These factors have shaped the uneven societal impact of the pandemic. In addition, populations directly at greater socio-

ecological vulnerability to infection include those in conditions of poverty and conflict zones (Sparke & Anguelov, 2020) and members of indigenous communities (Curtice & Choo, 2020). Spread in these populations may act as amplification loops for further spread and should be considered factors in the emergence of a pandemic as it is classically defined but perhaps there is a need to go beyond the notion of a universal pandemic.

Pre-Emergence: What factors meant SARS-CoV-2 was a pathogen of 'pandemic potential'?

A possible interpretation of the question at hand might relate to biological "factors": features of the virus, the disease it causes, and selection pressures which enabled it to infect humans and imparted transmissibility upon eventual exposure to the human population. Pandemic potential is something of an elusive concept, as before a disease starts spreading in a population there is no way to predict its impact due to several unknowns: potential epidemiological profile, and immunological and

behavioural responses. Most descriptions of pandemic potential hold that two essential components must be present: firstly, the ability to interact with human cells and secondly, the human-to-human transmissibility of the virus. However, many coronaviruses have demonstrated these characteristics and have not caused pandemics.

Coronaviruses are relatively common. Human coronaviruses (HCoVs), capable of infecting humans, cause an estimated 10-30% of all upper respiratory tract infections in adults, including common colds (Paules, et al., 2020),. There are seven known HCoVs as of writing (Kesheh, et al., 2022). Three of these, SARS-CoV-1, SARS-Cov-2, and Middle East respiratory syndrome coronavirus (MERS-CoV), have been the causative agents of significant outbreaks of severe respiratory illness since the turn of the century but only SARS-CoV-2 has caused a pandemic. As its classification suggests, SARS-CoV-2 is a "sister clade" (Gorbalenya, et al., 2020, p. 536) of SARS-CoV-1, the causative agent of the SARS epidemic of 2002-04. It has 79.6% genomic similarity to SARS-CoV-1 (Zhou, et al., 2020). Both SARS- CoV-1 and SARS-CoV-2, as well as HCoV-NL63, enter human cells through the same host-receptors, known as angiotensin-converting enzyme-2 (ACE2) (Rice, et al., 2022). This is mediated by the spike

(S) protein. The receptor binding domain of the S protein has an affinity to ACE2. Certain features of the SARS-CoV-2 S protein, such as the polybasic furin cleavage site, an amino acid insertion, enable more efficient fusion to the cell surface membrane and increase the replication ability of the virus in human cells (Hossain, et al., 2022). CpG deficiency, a low ratio of cytosine and guanine bases in the 5′-3′ direction which is targeted by the zinc finger antiviral protein also means SARS-CoV-2 can evade innate immune responses of several mammalian hosts (Xia, 2020).

Approximately 75% of all emerging infectious diseases (EIDs) identified in the past 3 decades are zoonotic in origin (Priyadarsini, et al., 2020) so it has become a null hypothesis for pathogenic

emergence. The currently known viral strains most genetically similar to SARS-CoV-2 have been identified in species of the genus *Rhinolophus* (horseshoe bats) in Laos (Temmam, et al., 2022), although none of these are progenitors of SARS-CoV-2. Consensus is currently that the reservoir species of SARS-CoV-2 is an undetermined species of horseshoe bat, though it is "likely to very likely" (WHO, 2021, p. 116) that this occurred through an intermediate host. RNA viruses evolve rapidly. Through recombination events and replication errors due to the error-prone nature of RNA-dependant RNA polymerase used in coronaviruses (Banerjee, et al., 2021), viral "quasispecies" arise, these are characterised by a high degree of genetic diversity and most quasispecies are not viable for further spread. The pandemic potential of SARS-CoV-2 must be considered in the context of human interactions with their environments and other species, as well as the clinical characteristics of the disease it causes, Coronavirus disease 2019 (COVID-19).

The Evolution of SARS-CoV-2: Human, Animal and Environmental Factors

The evolutionary events which lead to a bat-borne virus acquiring the genetic architecture to infect humans are considered highly unpredictable – a "stochastic conjunction of independent low probability events" according to Frutos et al. (2020, p. 1). However, they are likely to have been mediated by changes in human land use, anthropized environments and climate change. Changes in global bat distributions have shifted in recent years due to climate change. Areas of southeast Asia, in particular Laos, Myanmar and Yunnan in China have experienced increases in bat species richness, likely due to raised temperatures which create suitable habitats (Beyer, et al., 2021). This is directly correlated to the presence of coronaviruses. In addition, these changes have material influences on other species which may then also contract the virus. This allows adaptation. This also highlights the importance of biodiversity conservation in pandemic preparedness, anthropic behaviour such as land use change, re-shape the distribution and ecosystems of other species, increasing the likelihood of transmission. Yunnan specifically is also the site of China's largest reserves of aluminium and tin, as well as a large industry of agriculture. Six infections of severe respiratory syndrome causing coronaviruses may have occurred in workers at a Moijang mine in Yunnan in 2012. Investigations of the mine, including members of the Wuhan Institute of Virology, collected bat guano samples in which they found evidence of coronaviruses. One sample included a region of RNA-dependent RNA polymerase from a bat coronavirus designated RaTG13, most similar to SARS-CoV-2 after it was sequenced initially (Zhou, et al., 2020). Some have criticised local health authorities for not launching an epidemiological investigation per the International Health Regulations which had been revised in 2005 post-SARS epidemic. A possible early response could have allowed for the monitoring of viruses of concern and led to a more well-developed surveillance programme in environments such as mine

shafts and farms. Others however have questioned the relevance of RaTG13 in the emergence of SARS-CoV-2, as it is not a progenitor virus due to divergence at the RBD (Frutos, et al., 2022).

In the case that mutations which caused human susceptibility were acquired in an animal host (either the reservoir or an intermediate), the 'fitness landscapes' for the virus in one host would have to "fortuitously align" (Ruiz-Aravena, et al., 2022, p. 308) with that of humans. Fitness in the case of a virus simply means the ability to replicate and infect further cells, not necessarily virulence. The transmission of viruses between species is sometimes known as "viral chatter", this could have been mediated in anthropized environments in which multiple species are kept in close contact with one another, for example. Intense farming has been scaled up in China as part of a poverty alleviation campaign to raise living conditions and tackle food insecurity (Zhang, 2021). This creates the conditions for viral chatter, which can lead to rapid mutations because of diverse selection pressures in different populations, creating a metapopulation of diverse viruses. It is equally possible that an intense farm in a rural environment would house a diverse range of species. If these animals were in poorly managed conditions and housed very close to one another then it is possible that crossspecies transmission just so happened to produce a series of recombination events and mutations required for infection in humans. There is some evidence for this. ACE2 receptors are common in mammals and positive selection on the S protein genes, or antigenic drift, may have occurred in species with very similar ACE2 receptor homologies, Wei, et al. (2021) predict these to include species of civets and pangolins based on genetic similarity. This is also likely to include Nyctereutes procyonoides (raccoon dogs) (Freuling, et al., 2020).

Localised Emergence Before the Wuhan Outbreak?

Another scenario causing the adaptation of SARS-CoV-2 to humans is caused by early, undetected human spread. This aligns with phylogenetic models indicating the first spread of the virus to be far earlier than it was first noticed in Wuhan. Additionally, it aligns with the features of the disease caused by SARS-CoV-2, COVID-19 in humans. While both SARS-CoV-1 and SARS-CoV-2 bind to ACE2 in host cells, and both have similar infectivity in humans, shown by a basic reproduction (R₀) number of between 2-4, SARS-CoV-2 is not as virulent, it does not always cause symptoms of severe respiratory syndrome. When infected with SARS-CoV-2 most people without pre-existing comorbidities experience mild symptoms comparable to the symptoms of the common colds caused by the other HCoVs. SARS-CoV-1 and MERS-CoV cause disease in humans with far greater infection fatality rates (Rice, et al., 2022). Those viruses likely did not cause pandemics due to this characteristic; onward transmission chains could simply not be sustained. In cases of the disease in which even mild symptoms arise, there can be a 4–8-day delay in experiencing them in which transmission may be

possible. Thus, over 50% early of infections are likely to have been asymptomatic or pre-symptomatic transmission (Rice, et al., 2022). This raises the question of whether there had been multiple zoonotic events of ancestor viruses of SARS-CoV-2 in the months before the Wuhan outbreak. There is some evidence of this. Sánchez, et al.'s (2022) model predicts that over 66,000 separate events of zoonotic transmission of bat SARS-related coronaviruses each year in Southeast Asia. These occur across a very large geographical range, inhabited by millions. Bats are also generally immunologically resilient and not materially affected by infections, thus factors such as migratory events may lead to the introduction of these viruses to different communities. Sun, et al. (2020) note the high density of bats inhabiting caves as well as their ability to migrate huge distances of over 1000 kilometres.

Additionally, synanthropic communities, living close to bats, are shown through analysis of seroprevalence – the presence of antibodies – to be immunologically resilient to some SARS-related viruses (Sánchez, et al., 2022). Communities in Southeast Asia may have been living with coronavirus exposure for generations. Encroachment into these areas, through urban development projects and land use change for agriculture, has increased the risk of disease contamination. This, as well as the climatic changes which have shifted biodiversity patterns, can be viewed within a greater context of a globally emerging neoliberal attitude to the environment, the reliance of capitalism on capturing 'ecosystem services' or natural capital establishes illegal wildlife traders and hunters as potential disease vectors (Adla, et al., 2022). Perhaps one or multiple immunologically naïve individuals, possibly tourists or animal traders, became infected in one of the thousands of bat-to-human zoonotic events which occur annually. As above, these events trigger mutations in the virus, quasispecies which most likely are not transmitted but sequential chains of transmission following this may have led to different selection pressures and eventually the traits required for pandemic potential. Considering this, it is possible to offer alternative interpretations of the Wuhan narratives, the apparent site of localised emergence. Given the ability of Coronaviruses to cross species boundaries, through zoonotic transmission into humans, and the reverse such events, anthropozoonosis (El-Sayed, et al., 2021), several transmission chains between humans to animals in markets, farms or even domestic pets is plausible. A scenario in which a small number of infection events occurring in rural areas in southeast Asia is a potential pathway to later localised emergence, possibly thousands of miles away from Wuhan. Crucially, if this theory were true, it would place any human-to-human or further zoonotic transmission in Wuhan as secondary to the primary infection. Conventional narratives all imply that Wuhan should be considered as the location of the primary infection, whereas it is only the location of the first detected case (Frutos, et al., 2022).

Localised Emergence: The Wuhan Outbreak

A key unanswered question relates to where to place transmission in Wuhan on the continuum of pandemic emergence. There are potential environmental similarities between the characteristics of the Wuhan outbreak and other human coronavirus outbreaks. Both SARS-CoV-1 and SARS-CoV-2 first emerged in cold months (November 2002 and December 2019), this may have implications for innate immunity as nasal mucous becomes dry. In terms of human behaviour, this may have brought more people indoors, which facilitated the spread. Winter months could also be seen as opportunities for tourism to warmer parts of the world, tourism can quickly cause transmission chains as a result of chance encounters. Both outbreaks also occurred after draughts, 13000 reservoirs had dried up in the Guangdong province and 286,000 hectares of farmland were short of water. December 2019 in Wuhan also saw the lowest precipitation in 40 years (Sun, et al., 2020). Water scarcity on farms or in animal markets may have played a role in cross-species contamination due to unsanitary conditions. However, given the volume of spillovers distributed widely across Asia, an urban environment such as Wuhan may be a key site of a latency phase or amplification cycle in infections. In this case, probability might suggest a more likely emergence pathway for SARS-CoV-2 is from a different part of Asia such as the theory outlined above. This is simply due to the scale of distribution of potential zoonotic events. Potential tourism from Wuhan to areas of Southeast Asia, perhaps Laos, Myanmar and Yunnan as places where bat species richness has been demonstrated to have increased recently, should be considered as potential factors. An alternative interpretation might be that bats themselves simply migrated to Wuhan, possibly even attracted by seemingly innocuous features of the city such as the red and green lights on the Yangtze River Bridge which are turned on in the evenings (Sun, et al., 2020).

Amplification Factors in Wuhan

The evidence of zoonotic transmission of SARS-CoV-2 at the Huanan Seafood Wholesale Market has accumulated over time. Among the first efforts to determine the cause of the initial outbreak was a preliminary epidemiological investigation by the Wuhan Centre of Disease Control and Prevention. It suggested overlaps between cases of the illness and those who had been working in or visited the Huanan Seafood Wholesale Market. This led to its permanent closure on the 1st of January 2020. Retrospect informs that as of the 2nd of January, 41 people had been hospitalised with suspected cases. Of these patients, 27 had previously had "direct exposure" to the market (Worobey, et al., 2022, p. 951). This also means however that some early infections were not acquired there. According to some accounts, including in the WHO-China report, the first case had no connection to the market. Regardless of this, the primary infection of the disease is likely to have been somebody demonstrating mild symptoms as per the nature of COVID-19.

SARS-CoV-1 had also been linked to animal markets in the Guangdong Province, with species of the bat viruses established as the likely natural reservoir (Li, et al., 2005) and Paguma larvata (masked palm civets) hypothesised as an intermediate host as they were found to have harboured the virus (Wang & Eaton, 2007). In the case of SARS-CoV-2, a similar narrative has been promulgated; that the virus emerged through the illegal wildlife trade. Several animals have been proposed as intermediates, most prominently pangolins and raccoon dogs. Though neither bats nor pangolins were present in the market, Peker et al. (2022) demonstrated that 2 lineages, A and B, which diverged by 2 point mutations, had arisen from at least 2 zoonotic transmission events in the market. Given the nature of ACE2 receptors in mammals, this reflects both the ability of the agent to cross species barriers as well as an environment well placed for multi-species contact. Simultaneously, there has also been a suggestion that the food safety crisis in China has driven the increased purchase and consumption of wild animal products, with consumers potentially wearier of processed meat (Zhang, 2021), meaning animal markets are simply more frequently visited, again this allows for several transmission possibilities. More recent evidence, swab samples collected from the Huanan Seafood Market before closure, suggest that raccoon dogs were indeed carrying SARS-CoV-2 (Mallapaty, 2023). It should be noted that infections in Wuhan fell after a ban on wild animal trade was instigated, however, this decline may be more a factor of other 'lockdown' measures also in place. Ultimately, it seems likely that multiple zoonotic events were occurring in the market, and this environment was a significant amplification factor in wider emergence.

Christopher Wray, Director of the Federal Bureau of Investigation (FBI) of the United States, recently attested before the US Senate Select Committee on Intelligence that the agency believes a laboratory incident was the most likely emergence pathway of the pathogen (US Senate, 2023). An unclassified document prepared in 2021 by the National Intelligence Council of the US suggests that this is an evaluation made with "moderate confidence" (p. 1). However, it also reports that, at the time, all members of the Council considered two possible emergence pathways, a "natural exposure" and a "laboratory-associated incident", as plausible. The only evidence put forward by proponents of the laboratory narrative has been highly circumstantial, misinterpretations of truth or possibly politically motivated attempts to resonate with pockets of anti-Chinese and anti-Asian sentiment in the West (Jaworsky & Qiaoan, 2021). Researchers at the Wuhan Institute of Virology have studied coronaviruses in the past, such as RaTG13 mentioned earlier. A narrative based upon the modification of RaTG13 itself cannot be correct, firstly it is not genetically similar enough to SARS-CoV-2 at the level of the RBD and secondly, RaTG13 has never been isolated, only its RNA dependant RNA polymerase has. It had been reported that some members of this facility became unwell in the Autumn of 2019, but the reported symptoms have been deemed not diagnostic of COVID-19 cases.

A meta-analysis by Coccia (2022) suggested that, in principle, a laboratory leak is more likely than fortuitous evolution causing a novel HCoV to emerge. I would argue that this model misses a few key factors however: first, the presence of unknown or undetected spillover events beyond the 7 HCoV species, second, it places weight on the fact that most Coronavirus research was happening in China, but not that Wuhan as a city also has the facilities to detect such events whereas rural or peri-urban communities do not. Therefore, I argue that it should not come as a surprise that the first infections were only noted in a city with both modern public health infrastructure and local expertise to call upon. The Wuhan lab narrative reflects that outbreak narratives are reflections of "Situated Knowledges" (Haraway, 1988). These are partial perspectives; their limiting factors not only include the available evidence but also the limitations of those interpreting the evidence. Haraway's account that "politics and ethics ground *struggles for and contests over* what may count as rational knowledge" and ultimately "over knowledge projects in the exact, natural, social, and human sciences" (p. 587, emphasis added) reflects the dialogue which has unfolded between those of competing views on the pandemic's emergence.

Global Emergence: Interconnectedness, Political, Health, and Vulnerability Factors

Both the conditions which allowed a virus of likely animal origin to cross species barriers and the subsequent spread and transformation of an animal virus into a pandemic can be seen as a product of human activity in the context of development and urbanisation programmes. The global stage of the emergence of this disease was reflective of social realities and inequalities, as much as physical and biological ones as is revealed by the overlapping of geographies of vulnerability and infection (Sparke & Anguelov, 2020). Wuhan, a large urban settlement which enabled the local amplification of infections, has also become an increasingly interconnected city in recent years. Travel times between Wuhan and the so-called global cities of Beijing and Guangzhou have been reduced from 12 to 4 hours with the development of transportation routes (Zhang, 2021). Should a highly transmissible virus be introduced to such an environment, of course, there is a large potential for spread within its immediate area and to rapid escape into other parts of the world. Increased mobility through tourism and trade could be seen as evidence of the effect of what geographer David Harvey (1989) calls "time-space compression" on disease transmission networks. With ongoing processes of ruralto-urban migration and the creation of faster transportation routes, physical distances between individuals have become significantly less costly as a function of the time and energy taken to traverse them. In EU countries, trade exports and motorway density have been strongly positively correlated with the spread of the disease (Kapitsinis, 2020). In terms of human power relations, only those who are economically able to travel can spread the disease through these networks, causing amplification, exposing those who are potentially more vulnerable. Transnational corporations also

likely aided in facilitating spread through trade and travel routes before mitigations were instigated. In addition, environmental changes in urban environments caused by patterns of industrial activity, such as poor air pollution may have increased the likelihood of spread (Kapitsinis, 2020). Activities such as food and pharmaceutical products were among the few economic activities not to be shut down in response to the virus, although some evidence suggested air pollutant levels fell, there is

still a positive correlation between air pollution and COVID-19 death rate in EU countries (Kapitsinis, 2020).

There is also the factor of what many see as a belated political response to the disease's emergence in Wuhan, some have criticised the WHO for not declaring a public health emergency of international concern sooner. The Chinese Government has faced criticisms, that information was far too released slowly or that initial alerts and subsequent investigations were directly suppressed, contributing to misinformation. Governments in other countries, such as the UK failed to adopt control measures in the early stages, opting for approaches which would not involve shutting down the economy. A lack of response translated into amplification loops as behavioural changes were not implemented. In England, if the eventual measures taken 2 weeks earlier than they were would have reduced case numbers by 93% and deaths by 26-43,000 (Arnold, et al., 2022). Albeit this is a rather crude estimate as assumes that the reduction in transmission would have been the same in either case. A delayed response also reiterates the need for a One Health approach, a recognition of the close ties between human behaviour, environmental changes, animal health, and human health. The 'public health' approach which has been taken in much of the West constitutes a more reactionary set of responses to a threat, for example, the culling of 17 million minks in Denmark during the Covid-19 pandemic when evidence emerged of possible mink to human transmission of SARS-CoV-2.

Under the paradigm of Structural One Health (Wallace, et al., 2015), the emergence of zoonotic infectious diseases can also be understood as the flow of capital and of pre-existing demographic and political structures. Pre-existing socio-ecological and structural vulnerabilities should be included in a holistic model of this pandemic's emergence. The socio-ecological niche which humans have constructed in part for their global expansion, activities such as intense mining, farming, the domestication of animals, and the construction of international transportation routes for tourism and trade have facilitated the wider spread of the SARS-CoV-2, through global networks and flows of capital. The syndemic nature of the disease in those with co-morbidities is telling. These are structurally determined by nutritional, demographic, political and social transitions. Pathogens take hold differently in differentially vulnerable communities, ranging from indigenous communities that have a greater burden of potential comorbidities and often less space to physically distance (Curtice & Choo, 2020), to those in remote areas like the Arctic, where native and institutional health systems

have collaborated in a space which affords relatively low risk of transmitting the disease (Petrov, et al., 2021). The global emergence phase of a pandemic might best be considered as a patchwork of different local 'emergences'.

Conclusion

The emergence of the Coronavirus pandemic is often characterised by 'unfortunate accidents': the process of evolution that gave SARS-CoV-2 the ability to infect humans is seen as unpredictable. However, human behaviour and interaction with their environments created the conditions for the evolution of the virus. SARS-CoV-2 evolved under the influence of several potential selection pressures, but also against a backdrop of neoliberal attitudes to nature and the increased use of natural resources. Under a holistic lens, the apparent dualisms, of the pandemic as a product of 'anthropic influence' or as a product of 'nature' (Hassanin, et al., 2020), evaporate. Human activity in their environments and structural vulnerabilities have all contributed to the convergence of factors that led to the Coronavirus pandemic.

Bibliography

Adla, K., Dejan, K., Neira, D. & Dragana, Š., 2022. Chapter 9 - Degradation of Ecosystems and Loss of Ecosystem Services. In: *One Health*. s.l.:Elsevier Inc, pp. 281-327.

APA, 2023. Factor. [Online]

Available at: https://dictionary.apa.org/factor

[Accessed 2 June 2023].

Arnold, K. et al., 2022. Estimating the effects of lockdown timing on COVID-19 cases and deaths in England: A counterfactual modelling study. *PLOS One*, 17(4).

Banerjee, A., Mossman, K. & Grandvaux, N., 2021. Molecular Determinants of SARS-CoV-2 Variants. *Trends in microbiology (Regular ed.)*, 29(10), pp. 871-873.

Beyer, R. M., Manica, A. & Mora, C., 2021. Shifts in global bat diversity suggest a possible role of climate change in the emergence of SARS-CoV-1 and SARS-CoV-2. *The Science of the total environment*, Volume 767.

Bloom, J. D. et al., 2021. Investigate the origins of COVID-19. *Science (American Association for the Advancement of Science)*, 372(6543), p. 694.

Coccia, M., 2022. Meta-analysis to explain unknown causes of the origins of SARS-COV-2. *Environmental research*, p. 211.

Curtice, K. & Choo, E., 2020. Indigenous populations: left behind in the COVID-19 response. *The Lancet*, 395(10239), p. 1753.

Director of National Intelligence (.gov), 2021. *Unclassified Summary of Assessment on COVID-19 Origins*. [Online]

Available at: https://www.dni.gov/files/ODNI/documents/assessments/Unclassified-Summary-of-Assessment-on-COVID-19-Origins.pdf

[Accessed 28 May 2023].

Domingo, J. L., 2022. An updated review of the scientific literature on the origin of SARS-CoV-2. *Environmental research*, 215(pt. 1).

El-Sayed, A., Abdel-Daim, M. M. & Kamel, M., 2021. Zoonotic and anthropozoonotic potential of COVID-19 and its implications for public health. *Environmental Science and Pollution Research*

International, 28(38), p. 52599-52609.

Freuling, C. M. et al., 2020. Susceptibility of Raccoon Dogs for Experimental SARS-CoV-2 Infection. *Emerging infectious diseases*, 26(12), pp. 2982-2985.

Frutos, R., Lopez Roig, M., Serra-Cobo, J. & Devaux, C. A., 2020. COVID-19: The Conjunction of Events Leading to the Coronavirus Pandemic and Lessons to Learn for Future Threats. *Frontiers in Medicine*, Volume 7, pp. 1-5.

Frutos, R., Pliez, O., Gavotte, L. & Devaux, C., 2022. There is no "origin" to SARS-CoV-2. *Environmental Research*, Volume 207.

Gorbalenya, A. et al., 2020. The species Severe acute respiratory syndrome-related coronavirus: classifying 2019-nCoV and naming it SARS-CoV-2. *Nature Microbiology*, 5(4), pp. 536-544.

Haraway, D., 1988. Situated Knowledges: The Science Question in Feminism and the Privilege of Partial Perspective. *Feminist studies*, 14(3), pp. 575-599.

Harvey, D., 1989. The condition of postmodernity. Oxford: Blackwell.

Hassanin, A., Grandcolas, P. & Veron, G., 2020. Covid-19: natural or anthropic origin?. *Mammalia*, 85(1), pp. 1-7.

Horton, R., 2020. Ofline: COVID-19 is not a pandemic. The Lancet, 396(10255), p. 874.

Hossain, M. G., Tang, Y., Akter, S. & Zheng, C., 2022. Roles of the polybasic furin cleavage site of spike protein in SARS-CoV-2 replication, pathogenesis, and host immune responses and vaccination. *Journal of medical virology,* pp. 1815-1820.

Jaworsky, B. N. & Qiaoan, R., 2021. The Politics of Blaming: the Narrative Battle between China and the US over COVID-19. *Chinese journal of political science*, 26(2), pp. 295-315.

Kapitsinis, N., 2020. The Underlying Factors of the COVID-19 Spatially Uneven Spread. Initial Evidence from Regions in Nine EU Countries. *Regional Science Policy & Practice*, 12(6), pp. 1027-1045.

Kelly, H., 2011. The classical definition of a pandemic is not elusive. *Bulletin of the World Health Organization*, 89(7), pp. 540-541.

Kesheh, M. M., Hosseini, P., Soltani, S. & Zandi, M., 2022. An overview on the seven pathogenic human coronaviruses. *Reviews in medical virology*, 32(2), p. e2282.

Li, W. et al., 2005. Bats Are Natural Reservoirs of SARS-Like Coronaviruses. *Science (American Association for the Advancement of Science)*, 310(5748), pp. 676-679.

Luisetti, F., 2022. The Neoliberal Virus. In: V. Lemm & M. Vatter, eds. *The Viral Politics of Covid-19*. Singapore: Palgrave Macmillan, pp. 181-200.

Mallapaty, S., 2023. COVID-origins study links raccoon dogs to Wuhan market: what scientists think. *Nature (London)*, 615(7854), pp. 771-772.

Morens, D. M., Folkers, G. K. & Fauci, A. S., 2009. What Is a Pandemic?. *The Journal of infectious diseases*, 200(7), pp. 1018-1021.

Morse, S. S., 1995. Factors in the emergence of infectious diseases. *Emerging infectious diseases*, 1(1), pp. 7-15.

Morse, S. S. et al., 2012. Prediction and prevention of the next pandemic zoonosis;. *The Lancet (British Edition)*, 380(9587), pp. 1956-1965.

Paules, C. I., Marston, H. D. & Fauci, A. S., 2020. Coronavirus Infections—More Than Just the Common Cold. *Journal of the American Medical Association*, 323(8), pp. 707-708.

Pekar, J. E. et al., 2022. The molecular epidemiology of multiple zoonotic origins of SARS-CoV-2. *Science*, 377(6609), pp. 960-966.

Petrov, A. N. et al., 2021. Lessons on COVID-19 from Indigenous and remote communities of the Arctic. *Nature Medicine*, 27(9), pp. 1491-1492.

Priyadarsini, S. L., Suresh, M. & Huisingh, D., 2020. What can we learn from previous pandemics to reduce the frequency of emerging infectious diseases like COVID-19?. *Global transitions*, Volume 2, pp. 202-220.

Rice, B. L., Lessler, J., McKee, C. & Metcalf, C. J. E., 2022. Why do some coronaviruses become pandemic threats when others do not?. *PLoS biology*, 20(5).

Roberts, D. L., Rossman, J. S., Jaric, I. & Lee, B., 2021. Dating first cases of COVID-19. *Dating first cases of COVID-19.*, 17(6).

Ruiz-Aravena, M. et al., 2022. Ecology, evolution and spillover of coronaviruses from bats. *Nature reviews*. *Microbiology*, 20(5), pp. 299-314.

Sánchez, C. A. et al., 2022. A strategy to assess spillover risk of bat SARS-related coronaviruses in Southeast Asia. *Nature communications*, Volume 13.

Sparke, M. & Anguelov, D., 2020. Contextualising coronavirus geographically. *Transactions - Institute of British Geographers* (1965), 45(3), pp. 598-508.

Sun, Z. et al., 2020. Potential Factors Influencing Repeated SARS Outbreaks in China. *International journal of environmental research and public health*, 17(5).

Temmam, S. et al., 2022. Bat coronaviruses related to SARS-CoV-2 and infectious for human cells. *Nature (London)*, 604(7905), pp. 330-336.

US Senate, 2023. *US Senate Select Committee on Intelligence*. [Online] Available at: https://www.intelligence.senate.gov/hearings/open-hearing-worldwide-threats-3 [Accessed 27 May 2023].

Wallace, R. G. et al., 2015. The dawn of Structural One Health: A new science tracking disease emergence along circuits of capital. *Social science & medicine*, Volume 129, pp. 68-77.

Wang, L. & Eaton, B., 2007. Bats, Civets and the Emergence of SARS. In: J. Childs, J. Mackenzie & J. Richt, eds. *Wildlife and Emerging Zoonotic Diseases: The Biology, Circumstances and Consequences of Cross-Species Transmission*. Berlin, Heidelberg: Springer, pp. 325-344.

Wei, Y., Aris, P., Farookhi, H. & Xia, X., 2021. Predicting mammalian species at risk of being infected by SARS-CoV-2 from an ACE2 perspective. *Scientific reports*, 11(1).

World Health Organization, 2021. WHO-convened global study of origins of SARS-CoV-2: China Part, Geneva: Joint WHO—China Study.

Worobey, M. et al., 2022. The Huanan Seafood Wholesale Market in Wuhan was the early epicenter of the COVID-19 pandemic. *Science (American Association for the Advancement of Science)*, 377(6609), pp. 951-959.

Xia, X., 2020. Extreme Genomic CpG Deficiency in SARS-CoV-2 and Evasion of Host Antiviral Defense. *Molecular biology and evolution,* 37(9), pp. 2699-2705.

Zhang, L., 2021. *The Origins of COVID-19: China and Global Capitalism.* Stanford, California: Stamford University Press.

Zhou, P. et al., 2020. A pneumonia outbreak associated with a new coronavirus of probable bat origin. *Nature*, 579(7798), pp. 270-273.