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Aerosol transmission of human pathogens: From miasmata to modern viral pandemics and their preservation potential in the Anthropocene record

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Abstract

Ongoing uncertainty over the relative importance of aerosol transmission of COVID-19 is in part rooted in the history of medical science and our understanding of how epidemic diseases can spread through human populations. Ancient Greek medical theory held that such illnesses are transmitted by airborne pathogenic emanations containing particulate matter (“miasmata”). Notable Roman and medieval scholars such as Varro, ibn al-Khatib and Fracastoro developed these ideas, combining them with early germ theory and the concept of contagion. A widely held but vaguely defined belief in toxic miasmatic mists as a dominant causative agent in disease propagation was overtaken by the science of 19th century microbiology and epidemiology, especially in the study of cholera, which was proven to be mainly transmitted by contaminated water. Airborne disease transmission came to be viewed as burdened by a dubious historical reputation and difficult to demonstrate convincingly. A breakthrough came with the classic mid-20th century work of Wells, Riley and Mills who proved how expiratory aerosols (their “droplet nuclei”) could transport still-infectious tuberculosis bacteria through ventilation systems. The topic of aerosol transmission of pathogenic respiratory diseases assumed a new dimension with the mid-late 20th century “Great Acceleration” of an increasingly hypermobile human population repeatedly infected by different strains of zoonotic viruses, and has taken centre stage this century in response to outbreaks of new respiratory infections that include coronaviruses. From a geoscience perspective, the consequences of pandemic-status diseases such as COVID-19, produced by viral pathogens utilising aerosols to infect a human population currently approaching 8 billion, are far-reaching and unprecedented. The obvious and sudden impacts on for example waste plastic production, water and air quality and atmospheric chemistry are accelerating human awareness of current environmental challenges. As such, the “anthropause” lockdown enforced by COVID-19 may come to be seen as a harbinger of change great enough to be preserved in the Anthropocene stratal record.

1. Introduction

The COVID-19 public health emergency has refocused attention on the relative importance of aerosol transmission in the spread of human epidemic diseases. Initial emphasis by influential health organisations on the role of fomite and near-range droplet transmission of SARS-CoV-2 has triggered a robust counter-response from the aerosol scientist community (e.g. Fennelly, 2020; Jimenez, 2020; Morawska and Cao, 2020; Morawska and Milton, 2020; Prather et al., 2020; Tang et al., 2020, 2021a,b; Bourouiba, 2021; Greenhalgh et al., 2021; Morawska et al., 2021; Randall et al., 2021). This response has increasingly insisted that many COVID-19 infections are caused by the inhalation of airborne virus-bearing particles rather than via fomite or gravity-driven droplet contact, and that future health and urban planning policies need seriously to concentrate on indoor air quality issues.

The unwillingness of some members of the medical community to accept that aerosols commonly act as an important disease vector is nothing new. Forty-one years ago, an infected man travelling home from Pakistan initiated a nosocomial outbreak of smallpox in the German town of Meschede. Once the patient had been correctly diagnosed he was transferred to a smallpox isolation unit, but by then it was too late: some days later, new cases of the viral
disease appeared quite unexpectedly on all three floors of the hospital (Fig. 1a). None of these new smallpox cases had registered direct contact with the index patient, and transmission via fomites was examined in detail but rejected as highly unlikely. The only remaining route of transmission considered reasonable was airborne spread of a virus-containing aerosol, a possibility against which all of the investigators were initially prejudiced (Gelfand and Posch, 1971). Despite their misgivings, the investigators nevertheless finally concluded that their detailed epidemiological studies have clearly indicated that 17 of the cases were infected by virus particles disseminated by air over a considerable distance within a single hospital building (Wehrle et al., 1970). This conclusion has not only stood the test of time (Fenner et al., 1988; Tellier et al., 2019) but one overview study this century has argued that the weight of evidence suggests that fine particle aerosols were the most frequent and effective mode of smallpox transmission (Milton, 2012).

The arguments in favour of aerosol transmission of COVID-19 are primarily based on case studies of outbreak clusters in indoor microenvironments such as restaurants (Kwon et al., 2020; Lu et al., 2020; Li et al., 2021), cruise ships (Almilaji and Thomas, 2020), buses (Luo et al., 2020; Shen et al., 2020), choir practices (Charlotte, 2020; Hamner et al., 2020; Miller et al., 2020), fitness centres (Jang et al., 2020), meat processing plants (Guenther et al., 2020), call centres (Park et al., 2020), department stores (Jiang et al., 2021) and apartment blocks (Huang et al., 2021; Hwang et al., 2021; Fig. 1). In addition, there are supportive data from sampling campaigns, especially in hospitals (e.g. Guo et al., 2020; Lednicky et al., 2020; Liu et al., 2020; Nissen et al., 2020), epidemiological studies, modelling and data reviews (e.g. Adam et al., 2020; Endo et al., 2020; Jayaweera et al., 2020; Morgenstern, 2020; Zhang et al., 2020; Bazant and Bush, 2021; Cao et al., 2021; Dillon and Dillon, 2021; Eichler et al., 2021). There are also publications on aerobiological experiments (e.g. Fears et al., 2020; Nielsen and Liu 2020; Stadnyskij et al., 2020; van Doremalen et al., 2020; Shao et al., 2021) that include successfully spreading the disease to uninfected animals via ventilation systems (Kutter et al., 2021; Sia et al., 2020; Richard et al., 2020). Taken together, the weight of evidence from this extensive body of recently published scientific work, filtered through media reporting, appears to be shifting public opinion towards embracing the importance of aerosol transmission. Thus, a stereotypic public space “superspreading event” propagating COVID-19 has come to be viewed under our current understanding as driven by short and longer range aerosol transmission from infected but symptom-free individuals with high viral loading socializing without facemasks in crowded places, especially in indoor environments with inadequate ventilation.

Fig. 1. Examples of transmission clusters of viral epidemic diseases in buildings. A) Meschede Hospital, Germany 1970 smallpox outbreak, attributed to virus-bearing particles moving on air currents from index patient to infect others on all three hospital floors (Wehrle et al., 1970; Gelfand and Posch, 1971). B) Amoy Gardens residential apartments, Hong Kong 2003 outbreak of SARS-CoV-1 where infection was attributed to virus-bearing particles escaping through floor drains from the sewage-system into bathrooms and then driven as aerosols by ventilation fans into light wells serving the tower block (Yu et al., 2004; Li et al., 2004b; McKinney et al., 2005; Lee 2012; Ng, 2020; photo: WMwiki, CC By Creativecommons.org/licenses/by/3.0&lt;g; via Wikimedia Commons. https://commons.wikimedia.org/wiki/File:Amoy_gardens_2017.jpg). C) Apartment building in Seoul, South Korea in 2020 SARS-CoV-2 outbreak where movement of virus-bearing particles through vertical air ducts serving bathrooms was implicated in COVID-19 transmission (Hwang et al., 2021). D) Apartment building in Bilbao, Spain in 2021 SARS-CoV-2 outbreak where the viral transmission mode remains unclear (Galloway et al., 2021). https://english.elpais.com/society/2021-02-15/architecture-of-an-outbreak-the-spanish-apartment-building-hijacked-by-the-coronavirus.html.
It is notoriously difficult, however, to demonstrate absolute proof of disease transmission via aerosols. In a prescient paper by Pan, Lednicky and Wu, published in the December 2019 issue of the Journal of Applied Microbiology, the same month SARS-CoV-2 was officially recognized in Wuhan, the authors comment: “...accurate determination of the presence of airborne viruses is challenging. This shortcoming limits our ability to evaluate the actual threat arising from inhalation or other relevant contact with aerosolized viruses (Pan et al., 2019).” Even more technically challenging is the demonstration that pathogens detected in airborne particles or ventilation systems are still viable (Brown et al., 2015). Thus many of the studies sampling for SARS-CoV-2 in potential viral “hotspots” have only demonstrated the presence of viral traces (e.g., Ong et al., 2020; Zhou et al., 2020; Hadei et al., 2021; Moreno et al., 2021), with very few using sampling methodologies capable of proving the presence of viable virions (e.g., Lednicky et al., 2020; Santarpia et al., 2020; Lednicky et al., 2021). A continuing problem here is that current air sampling technologies are more aggressive than simple human exhalation and therefore potentially capable of damaging a captured virion sufficiently to prevent it growing in a culture (Tang et al., 2021a). However, following the traditional aphorism “absence of evidence is not evidence of absence”, the lack of proof for viable virions does not prove they are not there (Greenhalgh et al., 2021). The data published so far on COVID-19 patterns of propagation, the precautionary principle demands that we focus our attention on face-masks and ventilation in indoor environments.

As the evidence for aerosol involvement with COVID-19 propagation has grown stronger, so the case for emphasizing fomite and droplet transmission has weakened, despite continued reluctance expressed in some medical circles. By July 2020 the World Health Organisation, faced with the data on COVID-19 clusters in restaurants, choir practices and fitness centres, agreed that short range aerosol transmission...cannot be ruled out but insisted that droplet and fomite transmission could also explain human-to-human transmission within these clusters (WHO, 2020). As more evidence accumulated there has been increasing emphasis on the importance of indoor ventilation and avoidance of breathing stale air (WHO, 2021a), but why such initial reticence? In this paper, we attempt to answer this question by approaching the subject from an historical perspective, tracing how our understanding of epidemic diseases and their causes are to be found in the Hippocratic Corpus, a collection of early Ancient Greek medical writings dating back to the 4th and 5th century BCE. From these Hippocratic doctrines evolved the idea that pestilential diseases, rather than having been sent by the gods using toxic atmospheric emanations as punishment for wrongdoing, are caused by inhaling vaporous “miasma” in the air and lack a supernatural origin (Parke, 1983). The idea evolved into miasma theory, which became widely accepted by medical practitioners throughout much of the Old World. The theory postulated the presence of pathogenic evil smelling mists derived from decaying organic matter and full of tiny particles, the pestilential seeds of the Greco-Roman medical practitioner Galen (Galen, 165–175; Jouanna, 2012). In a famous quote, the Roman scholar Varro thought this bad air had an organic origin because certain tiny animals grow, which the eyes cannot detect and which pass through the air and can enter the body through the mouth and nose to cause serious diseases (Varro, 36BC; Jarcho, 1976).

Medieval medicine continued to champion miasma theory as a key explanation for the spread of epidemics, although tempered by lessons learnt from the reality of successive waves of bubonic plague. It became clear to some of those with direct experience of the plague that the disease was best dealt with less by worrying about bad air and more by avoiding physical contact, imposing quarantines, and using isolation hospitals. Conflict between authorities entrenched in their miasmatic traditions and those emphasizing the dangers of direct contagion is famously demonstrated by the fate of the 14th century Andalusian scholar Ibn al-Khatib who served as vizier to the Nasrid court in Granada. Arguing, along with others, that bubonic plague could be brought into a city by infected persons arriving by boat and spread between people and in clothing, al-Khatib insisted that isolation protected people from contagion (Ober and Aloush, 1982). His demands that orthodox teachings based on beliefs in divine will and miasmic toxic vapours should be modified to encompass the evidence for disease transmission by close contact were perceived to be heretical and resulted in his murder in 1374 (Steams, 2009; Hopley, 2010).

Moving from Medieval to early Modern times, another well-documented contribution to our understanding of epidemic disease was that by Girolamo Fracastoro. Building on the ideas of some of his predecessors Fracastoro in 1546 postulated that such epidemic illnesses spread via different types of seed-like “seminaria” or germs that could be transmitted by direct contact, on fomites (his “fomes”: such as contaminated clothing and surfaces), or through the air (e.g., Garrido, 2016). The clarity of his thinking is demonstrated by his direct relevance to the current fomite-droplet-aerosol debate on the transmission of COVID-19 over 475 years later. However, although certainly influential, the ideas of Fracastoro on contagious diseases (the contagium animatum) did not fundamentally shift the continuing dominance of Greco-Roman miasmatic theory with regard to the explanation of epidemic diseases in European medical science. As William H Welch, one of the founders of the John Hopkins School of Hygiene and Public Health, was to observe in 1925 hypotheses born before their time are often sterile (Welch, 1925; Ackerknecht, 2009). As a geological aside, Fracastoro was similarly ahead of his time in his logical conclusions that fossil shells had once belonged to living organisms and could not be explained by a Great Flood. Charles Lyell later lamented The clear and philosophical views of Fracastoro were disregarded, and the talent and argumentative powers of the learned were doomed for three centuries to be wasted in the discussion of these two simple and preliminary questions: first, whether fossil remains had ever belonged to living creatures; and, secondly, whether, if this be admitted, all the phenomena could be explained by the deluge of Noah (Lyell, 1830).

By the mid-19th century, before the key discoveries of Pasteur, Koch and other microbiologists, the medical world still lacked evidence for a unifying theory of infectious disease. Given the economic opportunities of an increasingly populated and globalised society, politised argument between “contagionists” favouring quarantine restrictions to control epidemics and those free-marketeers who championed freedom of movement had reached a peak. Yellow fever epidemics (caused by a virus later proven to be transmitted via mosquitoes) such as that in Philadelphia in 1793 in particular had convinced a majority that quarantine laws
should be relaxed (Ackerknecht, 2009). Amid this confusion, a key driving force for change and clarity was scientific study of a series of cholera pandemics (Table 1), the first originating in the Indian sub-continent in 1817 and spreading from the Ganges Delta across Asia and as far west as the eastern Mediterranean (Barau, 1992). Subsequent waves of 19th century cholera pandemics included Western Europe in their reach (Fig. 2), and in 1854 medical breakthroughs in the identification of the cholera bacillus Vibrio cholerae, its epidemiological propagation and possible sources were made separately in Italy, Spain, and Great Britain. In Italy Filippo Pacini isolated and described the bacterium in samples taken directly from cholera patients during an outbreak in Florence, publishing his work in December 1854 (Pacini, 1854; Fig. 3). In Spain, where over 236,000 people are reported to have died from the outbreak (Kohn, 2008), Joaquín Balcells also discovered the bacterium. In late August, when the epidemic was at its peak in Barcelona, Balcells described live bacteria in a pure water sample that had been left close to the bed of a cholera patient (Fig. 3). His microscopic observations revealed hundreds of vibrios endowed with an astonishing mobility; their movement angular like lightning (Balcells, 1854). Balcells deduced that his water sample had been contaminated by airborne microorganisms carried in exhalations (exhalaciones mórbidas) from the cholera patient (Corbella i Corbella, 1989).

Meanwhile, London in the same month of August 1854 was experiencing its worst cholera epidemic on record and, as in Florence and Barcelona, miasma theory continued to dominate medical thinking. The legendary epidemiological work by John Snow in systematically demonstrating that cholera infection in London was linked more to contaminated water rather than air had a dramatic impact (Snow, 1855; Parkes, 2013). It helped stimulate renovation linked more to contaminated water rather than air had a dramatic impact (Snow, 1855; Parkes, 2013). It helped stimulate renovation of the entire city sewage system, transforming the capital into one of the healthier modern cities at the time. It also brought into disrepute those in the medical establishment with traditional “miasmatic” explanations who had refused to accept and had even strongly denigrated Snow’s conclusions. Echoes of the shock produced by this change in the scientific medical landscape may be traced even into this century. In 2013, for example, The Lancet published an extraordinary correction after an unduly prolonged period of reflection to their original dismissive obituary of Snow, who died suddenly in 1858, in which no mention of cholera is made (Hempel, 2013). The then editor of The Lancet had indeed been an outspoken critic of Snow but his views were shared by most (Hempel, 2013). He himself implied, tuberculosis was to provide the test case. The Lancet published an extraordinary correction after an unduly prolonged period of reflection to their original dismissive obituary of Snow, who died suddenly in 1858, in which no mention of cholera is made (Hempel, 2013). The then editor of The Lancet had indeed been an outspoken critic of Snow but his views were shared by most (Hempel, 2013). He himself implied, tuberculosis was to provide the test case.

3. Tuberculosis: The proof of aerosol transmission

The path to proving that aerosol transmission of human diseases can be important lay in the study of tuberculosis (TB), an ancient respiratory infection that has accompanied humans since at least Late Pleistocene times (Cambau and Drancourt, 2014; Barbaris et al., 2017). By the 19th century, TB had become one of the primary causes of death by epidemic disease in Europe. After many years of debate, it was finally demonstrated to be capable of transmission between mammalian species (Villemin 1865, 1868) and caused by the bacterium Mycobacterium tuberculosis (Koch, 1882; Sakula, 1982). The disease is now recognized to be propagated typically via exhalatory bioaerosols that are subsequently inhaled into the deep lung where they initiate an infection of alveolar macrophages (Nardell, 2016). This recognition of a primarily airborne pathology is founded upon painstaking mid-20th century experimental work centred on the three outstanding figures of William F. Wells, Richard L. Riley and Cretyl I. Mills (née Crumb) (Fig. 4). By the time these experiments had been performed and their key results written up by Riley (the Principle Investigator for the project), Wells was dying of cancer and Mills had herself contracted TB (Riley et al., 1959, 1962; Riley, 2001).

Wells had received his public health training in the wake of the influential late 19th century work of Carl Flügge on infective exhalatory particles. Potentially infective “Flügge droplets”, demonstrated by Flügge as released by breathing, talking, sneezing, coughing or vomiting, were generally thought capable of transmitting disease only in close proximity to the patient (although not, perhaps, by Flügge himself: see Randall et al., 2021). In his influential book Sources and Modes of Infection (1910), Charles Chapin wrote: Bacteriology teaches that former ideas in regard to the manner in which diseases may be airborne are entirely erroneous; that most diseases are not likely to be dust-borne, and they are spray-borne for only 2 or 3 feet... although he did add Tuberculosis is more likely to be air-borne than is any other disease (Chapin, 1912). In the same chapter Chapin went on to argue that to emphasise airborne infection is most misconceived because it discourages attempts to minimize infection by close contact: It is impossible, as I know from experience, to teach people to avoid contact infection while they are firmly convinced that the air is the chief vehicle of infection. Such remarks express a genuine medical concern that insisting on longer-range aerosol transmission without clear proof makes the challenge of patient care and hygiene much more difficult. The same point is cogently placed into modern context over 100 years later in a commentary written just months before the emergence of SARS-CoV-2: Although short-range large-droplet transmission is possible for most respiratory infectious agents, deciding on whether the same agent is also airborne has a potentially huge impact on the types (and costs) of infection control interventions that are required (Tellier et al., 2019).

Nevertheless, despite the orthodoxy of the day and as Chapin had himself implied, tuberculosis was to provide the test case. Wells, working with his wife Mildred Weekes Wells (e.g. Wells and Wells, 1936a, Wells and Wells, 1936b), knew this and applied himself on a mission to convince unbelievers (Riley, 2001) to demonstrate beyond reasonable doubt that TB could indeed be transmitted via aerosols. His persistent line of enquiry was based on the argument that particles exhaled from infected patients can remain airborne long enough for viable pathogens, in this case Mycobacterium tuberculosis, to be carried considerable distances away from the emission source. He and his colleague Riley envisaged rapid evaporative diminution of exhaled droplets to smaller-sized particles, which they termed “droplet nuclei” (Wells, 1934, 1955; Wells and Riley, 1937; Wells et al., 1948). The idea was that these “droplet nuclei” could spread the disease as aerosols, and it was upon this premise that the classic Baltimore Veterans Hospital experiment was constructed (Riley et al., 1959, 1962; Riley, 1974, 2001).

Having demonstrated that TB can be spread by aerosol inhalation between rabbits (Wells et al., 1948), the primary objective became to investigate if aerosols released by human TB patients could move through a ventilation system into an animal chamber and infect guinea pigs with the same disease. The study took place in a specially prepared hospital annex of six rooms all connected via a ventilation system to a penthouse directly above and containing live animals (Riley, 2001). Initial experiments demonstrated that TB bacilli atomized into the ventilation system could infect rabbits in the penthouse. This was followed by a 2-year study during which infectious human TB patients were transferred to the annex and their exhalations passed through the ventilation shaft to 150 guinea pigs housed in the penthouse. The resulting publication (Riley et al., 1959) demonstrated that on average three guinea...
Table 1
Notable documented outbreaks of human bacterial and viral epidemic diseases, estimated mortality, and growth in World population to the present day level approaching 8 billion. Most of the epidemic diseases listed are zoonotic in origin, especially those newly emerging in recent times. Increasingly rapid rise in human population becomes evident in the 18th century and especially characterises the Anthropocene “Great Acceleration” from the mid-20th century onwards. Aerosol transmission of diseases such as tuberculosis, smallpox, measles, chickenpox, influenza and COVID-19 has been scientifically demonstrated and is likely to be commonplace, highlighting the importance of controls on indoor air quality (see text for details).

| Date          | Diseases | Estimated mortality | Estimated global human population(billion): bold type for years listed in column 1 |
|---------------|----------|---------------------|--------------------------------------------------------------------------------|
| 1332BCE-1888CE | Hittite epidemic 1, 1332BCE | Demonstrably accurate mortality records for most pre-20th century outbreaks unavailable | 0.1 in 1332BCE |
|               | Plague of Athens 2, 429BCE  | Estimates commonly vary depending on sources | 0.16 in 429BCE |
|               | Antonine Plague 3, Roman Empire: 165CE | Epidemics recorded as causing outstanding numbers (millions) of deaths before the 20thC include those involving the pathogenic diseases smallpox, bubonic plague, influenza, measles, tuberculosis, cholera and haemorrhagic fevers of uncertain diagnosis such as “cocoliztli” | 0.21 in 165CE |
|               | Smallpox 4,  has infected humans since prehistoric time; early examples include Japan 735; frequent historic epidemics until eradication in 1979 | | 0.23 in 165CE |
|               | 1st bubonic plague pandemic 5, Eurasia 541 with later outbreaks in 7-8thC | | 0.3 in 165CE |
|               | 2nd bubonic plague pandemic 6, Eurasia 1346 with later major outbreaks in 16-18thC | | 0.39 in 1346 |
|               | 3rd bubonic plague pandemic 7, China 1855 then globalised with outbreaks into 20thC | | 0.4 in 1234 |
|               | Influenza 6, frequent Eurasian epidemics since at least 14thC and pandemics since globalisation | | 0.45 in 1940 |
|               | Columbian Exchange 9, 1492 and onwards as epidemics of smallpox, measles, etc. swept the Americas | | 0.5 in 1345 |
|               | “Cocoliztli” hemorrhagic fever 10, Mexico 1545 with repeated outbreaks until 1815 | | 0.51 in 1545 |
|               | Remote island 19thC epidemics 11, e.g. Faroe Islands 1846, Hawaii 1848, Mauritius 1874, Fiji 1875 | | 0.6 in 1699 |
|               | Typhus 12, epidemics common in warring armies e.g. Europe 1489, 1526, 1632, 1812 | | 0.7 in 1726 |
|               | Measles 13, endemic in Eurasia since at least Middle Ages | | 0.8 in 1747 |
|               | Yellow fever 14 from Africa to Caribbean, American continent and S Europe; outbreaks frequent in 19thC | | 0.9 in 1774 |
| 1889-92       | Influenza A (possibly subtype H3N8) or coronavirus OC43 (“Russian flu” 15): First pandemic spread by highly connected rapid global transportation (rail and shipping) | “1 million” | 1.0 in 1817 |
| 1911          | Measles 16, Remote island “virgin soil” epidemic on Rotuma | Dem demonstrably accurate mortality records for most pre-20th century outbreaks unavailable | 1.1 in 1824 |
| 1916          | Poliomyelitis 17, United States | Estimates vary | 1.2 in 1841 |
| 1918-20       | Influenza A subtype H1N1 pandemic (“Spanish flu” 18): Many fatalities due to secondary bacterial infections causing pneumonia. | | 1.24 in 1846 |
|               | Typhus 19, Russia during and after WW1 & revolution | “2-3 million” | 1.3 in 1855 |
| 1947          | Cholera 20, Egypt | 10,277 | 1.4 in 1871 |
| 1948-52       | Poliomyelitis 21, United States | c. 9000 | 1.5 in 1884 |
| 1957-58       | Influenza A subtype H2N2 pandemic (“Asian flu”) 22 | >1 million | |
| 1960-62       | Yellow Fever 23, Ethiopia | 30,000 | |
| 1968-70       | Influenza A subtype H3N2 pandemic (“Hong Kong flu”) 24 | >750,000 | |
| 1972-73       | Influenza A subtype H3N2 pandemic (“London flu”) 24 | unce | |
| 1977-78       | Influenza A subtype H1N1 pandemic (“Russian flu”) 25 | | |
| 1981-present  | HIV/AIDS pandemic 26 | >35 million | 4.5 in 1981 |
| 1996          | Meningococcal meningitis 27, Nigeria | >11,000 | 5.8 in 1996 |
| 2002-04       | Severe acute respiratory syndrome coronavirus (SARS-CoV-1) 28 | 774 | 6.28 in 2002 |
|               | First pandemic of the 21stC 29 | | 6.44 in 2004 |

(continued on next page)
4. Respiratory bioaerosols and viral pathogens

Much more is known today about human respiratory aerosols than 50 years ago, not least because of the increasing sensitivity of measuring instruments such as optical particle counters and sizers which demonstrate that most such particles range in size from just several hundred nanometres to a few microns (e.g. Fairchild and Stamper, 1987; Papanesi and Rosenthal 1997; Fabian et al., 2008). Thus, in terms of number concentrations exhaled aerosols will be mostly too small to carry many, if any, airborne bacterial pathogens. Mycobacterium tuberculosis for example typically measures 2–4 μm in length and has been observed to be present in exhalatory particles less than 5 μm in size (Fenelly et al., 2012).

Individual viral pathogens, in contrast to bacteria, typically lie in the size range of 30–150 nm, categorizing them in aerosol science as “ultrafine to quasi-ultrafine particles”. They are much smaller than most aerosol sizes emitted by normal tidal breathing (e.g. Morawska et al., 2008; Johnson et al., 2011; Fabian et al., 2008). A key mechanism for their expulsion from the infected host involves particles of respiratory tract lining fluid being generated deep in the lung by the breakup of liquid films during repeated reopening of airway capillaries (Johnson and Morawska, 2008; Haslbeck et al., 2010; Schwarz et al., 2010). Exhalation of such particles will present a threat of airborne disease transmission if they carry virions capable of maintaining viability long enough to infect a new host (e.g. Jones and Brosseau, 2015; Lv et al., 2021). For a discussion on infection mechanisms and controversies over “droplets” and “aerosols” see Randall et al. (2021), but suffice to state here that there is by now a wealth of evidence that infectious exhalatory bioaerosols can be transmitted through the air for considerable distances: they are the “Modern Miasmas” of our time (Mubareka et al., 2019).

Studies on viable virus-bearing aerosols, here shortened to “viraerosols”, known to be capable of spreading epidemic diseases highlight those carrying measles (e.g. Riley et al., 1978), chickenpox (e.g. Gustafson et al., 1982), now-eradicated smallpox (Wehrle et al., 1970), and influenza (e.g. Cowling et al., 2013). These pathogens have produced massive loss of human life but information on their transmission modes can be equivocal. The relative importance of smallpox viraerosols remains unclear because the mode of smallpox transmission was never conclusively established (Milton, 2012), despite this disease having been one of the great epidemic killers. In contrast, other viruses have been more con-

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Table 1 (continued)

| Date          | Diseases                                      | Estimated mortality | Estimated global human population (billion) |
|---------------|-----------------------------------------------|---------------------|-------------------------------------------|
| 2009-10       | Influenza A virus subtype H1N1 pandemic (“swine flu”) 31 | >250,000            | 6.85 in 2009                               |
| 2010-19       | Cholera 32; Haiti                              | >10,000             | 6.93 in 2010                               |
| 2012-present  | Middle East Respiratory Syndrome coronavirus (MERS-CoV) 33 | >900                | 7.10 in 2012                               |
| 2013-16       | Ebola 34; Western Africa                       | >11,000             | 7.18 in 2013                               |
| 2019-2021     | Severe acute respiratory syndrome coronavirus (SARS-CoV-2) 35 | >3.8 million        | 7.67 in 2019                               |

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Fig. 2. Cholera tramples the victor and the vanquished both: Title of a 1831 sketch by the famous illustrator Robert Seymour during the second 19th century cholera pandemic which spread west from Russia during the Polish Russian War 1830–31. Note the depiction of a black miasmatic cloud accompanying the figure of death: prevailing miasma theory did not adequately explain cholera transmission and there was much debate and speculation at the time amongst the medical and scientific community. Image in the collection of The London Library of Medicine, London. (Wikipedia Commons).
vincingly demonstrated to be commonly spread by airborne particles. It has been proposed, for example, that around 50% of influenza A virus transmissions occur via aerosols (Cowling et al., 2013), whereas measles appears to be the most “aerosol-adapted” of them all and is recognized as a classic human-specific airborne disease with an unusually high infection attack rate (e.g. Wallinga et al., 2005). When these pathogenic viruses enter unprotected human societies, such as happened in the 15-16th century American continent (Nunn and Qian, 2010) or in 18th century Australia (Dowling, 1998), the resulting epidemics can decimate entire populations. Even in societies normally more adapted to these pathogens, the appearance of a new viral strain capable of aerosol transmission in dense, mobile human populations can produce dramatic spikes in mortality, as evidenced by the H1N1 influenza A event of 1918–20 (Fig. 5) and the ongoing COVID-19 pandemic.

Fig. 3. Hypotheses born before their time are often sterile. Both Filippo Pacini and Joachín Balcells separately observed the cholera bacillus in 1854 during the third 19th century cholera pandemic but the subject received little attention until the rediscovery of the microorganism by Robert Koch in 1884. Top left: one of a series of publications on cholera by Pacini (Source: The Historical Medical Library of The College of Physicians of Philadelphia. Pacini F. Du Choléra Asiatique. Tr. Janssens E. Bruxelles: Librairie Médicale de H. Manceaux, 1865); lower left: Pacini’s 1854 microscope slide of the cholera bacillus (Wikipedia Commons); top right: the Spanish publication in which Balcells reported his experimental observations; lower right: translation of the front page editorial announcing Balcells’ discoveries.

Fig. 4. From left to right, William Firth Wells (1886–1963), Richard L. Riley (1911–2001), Cretyl Inez Mills (née Crumb) (1919–1990). These three scientists were key to proving that tuberculosis can be transmitted via aerosols (see text for details). Kind permission to reproduce the Wells and Riley photos has been given by Professor Edward Nardell who was given them by the Riley family after 2001. Nardell writes of Cretyl Mills: Riley told me that she was writing the history of their classic aerosol studies when she died but her manuscript’s location is unknown. Riley (also) told me that she deserved a lot of credit for keeping track of a huge amount of data that those studies generated.
5. Human coronaviruses

The first coronavirus to be described was that causing avian infectious bronchitis (AIB) in chickens (Schalk and Hawn, 1931), an acute, highly contagious disease that is commonly transmitted via airborne exhalatory and faecal particles. Airborne transmission is via aerosol and occurs readily between birds kept at a distance of over 1.5 m (Ignjatovic and Sapats, 2000). Although normally associated with relatively close contact transmission, one study implicated prevailing nighttime winds as capable of spreading the disease between farms separated by over 1 km (Cumming, 1969).

The morphological similarity between the AIB virus and previously uncharacterized human “common cold” respiratory viruses was revealed by the electron microscopy work of June Almeida in 1967 (Almeida and Tyrrell, 1967; Fig. 6) and the generic group subsequently named coronavirus (Almeida et al., 1968; Wildy, 1971).

At present 7 coronaviruses are known to infect humans (HCoV-OC229E, HCoV-NL63, HCoV-OC43, HCoV-HKU1, SARS-CoV-1, SARS-CoV-2 and MERS-CoV). The first four of these coronaviruses are well established in the human population, typically causing relatively mild upper respiratory tract infections and only rarely leading to critical illness (Patrick et al., 2006; Dijkman and van der Hoek, 2009). Although now relegated to “common cold” status, regularly infecting and re-infecting humans in seasonal epidemics, their original appearance as a new respiratory coronavirus strain would have likely caused widespread serious illness until brought under control by group immunity. HCoV-NL63 and HCoV-OC229E for example are thought to have emerged from bats to infect humans possibly in the 13-15th and 18th centuries respectively (Huynh et al., 2012; Corman et al., 2015; King, 2020). In this context, HCoV-OC43 has been speculated to be linked to a late 19th century outbreak of bovine respiratory disease and synchronous 1889–1890 “Russian flu” pandemic in humans (Vijgen et al., 2005; Table 1). More recent and therefore much better documented newly emergent human coronavirus respiratory infections (SARS and MERS) have this century joined the list of human diseases with pandemic potential (Table 1).

In November 2002, reports began to emerge of atypical pneumonia cases occurring in Guangdong Province, China. The first well-publicised outbreak of this new disease, termed severe acute respiratory syndrome (SARS) and later traced to a zoonotic coronavirus reservoir of bats in Yunnan Province (Wang et al., 2018), was brought from Guangdong to Hong Kong by a Chinese medical professor in February 2003 (Peiris et al., 2004). In the resulting outbreaks, while most cases were thought to be passed on during close contact with infected individuals, there are clear indications of some contribution by aerosol transmissions. In the largest nosocomial outbreak, for example, which centred on Ward 6A of the Prince of Wales Hospital Hong Kong in March 2003, the ventilation system was identified as likely having helped to spread the disease (Li et al., 2004a, Li et al., 2004b; Yu et al., 2005). Another well-studied Hong Kong disease cluster was that occurring in the Amoy Gardens residential tower block complex where 187 SARS cases were reported (Fig. 1). In this case, the spread of the disease was attributed to viraerosols escaping through floor drains from the sewage-system into bathrooms and then driven by ventilation fans into light wells serving the tower block (Li et al., 2004a, Li et al., 2004b; Yu et al., 2004; McKinney et al., 2006). A third case study from the Hong Kong 2003 epidemic also implicated a contribution from viraerosol transmission in reporting data from a flight to Beijing when a SARS-symptomatic individual spread the disease to an estimated 22 other passengers (Olsen et al., 2003).

A decade after the appearance of SARS-CoV-1, Middle East Respiratory Syndrome (MERS) was recognized as the second 21st century emergent zoonotic coronavirus respiratory disease with pandemic potential (de Groot et al., 2013). The disease in humans probably originated from bats infecting dromedary camels, with subsequent human-camel contagion likely and human–human
transmission confirmed (e.g. Zumla et al., 2015). Discussions as to how the illness is passed to and between humans have centred on the familiar “Fracastoro tripartite” of fomite versus close contact versus longer range aerosol transmission, usually with emphasis on direct contact with camels and nosocomial close contact between people (WHO, 2021b). One modelling study has suggested that longer-range aerosol transmission from superspreader hospital patients best fits the data available from a major nosocomial outbreak in the Republic of Korea (Xiao et al., 2017). So far, however, the disease does not appear to be especially efficient in human-to-human transmission, and it has been eclipsed by the emergence of SARS-CoV-2 in 2019 and the resulting COVID-19 pandemic. Nevertheless, unlike SARS-CoV-1, the MERS coronavirus problem is far from having been eliminated, and it continues to expand its reach across the Middle East and parts of Africa and Asia. With a confirmed case fatality rate of over 30%, much higher than the SARS pathogens, MERS-CoV currently presents an imminent pandemic threat through genetic mutation or recombination with other human coronaviruses (Zhang et al., 2021).

6. Pandemic preservation potential in the geological record

The sudden appearance of a rapidly spreading acute disease has a dramatic effect on the target population and can result in long-term societal and environmental impacts. On a local level, there are many documented cases of devastating epidemics affecting isolated communities when they became exposed to the global pathogen pool. A classic example, referred to as a “benchmark in Pacific history” (Cliff and Haggett, 1985), was the arrival of the measles virus to Fiji in 1875 (Table 1). The resulting epidemic killed over 20% of the population, wiped out the leadership, and encouraged foreign worker immigration that changed the agro-economic and political future of the island (Shanks, 2016). The same pathogenic process, although magnified to continental scale during the Columbian Exchange and involving a diversity of diseases, reduced the late 15th–16th century indigenous population of the Americas by an estimated 80%–95% (Nunn and Qian, 2010). It has been hypothesised that carbon sequestration due to the regeneration of natural vegetation after this catastrophic loss of farming communities was enough to explain a minor (7–10 ppm) transient drop in atmospheric CO₂ levels (e.g. Lewis and Maslin, 2015; Koch et al., 2019). This idea is not proven and alternative explanations exist for the CO₂ decline (Zalasiewicz et al., 2015, 2021; Rubino et al., 2016), but the hypothesis does usefully serve to highlight the possibility of atmospheric chemistry being altered by the consequences of anthropogenic epidemic disease. This in turn leads to the concept of disease-driven environmental change being of such a magnitude that it has a realistic possibility of being preserved in the geological record.

The precipitate loss of tens of millions of people by epidemic disease was to be repeated in the 1918 H1N1 influenza “Spanish flu” pandemic (Table 1), by which time the world human population had grown to >1.5 billion, over three times that when Columbus sailed to the Americas (Table 1). In contrast to the Columbian Exchange epidemics, which involved different diseases spreading through the American continent over the decades after 1492, the 1918 influenza event was caused by just one pathogen and took place during four successive waves in only two years. A warning of such a possibility had been issued nearly 20 years earlier by another flu-like pandemic (the “Russian Flu”: either influenza A or the coronavirus OC43) which is reported to have killed around 1 million people and was the first to spread rapidly around the world via modern transport infrastructure (Table 1). The likely importance of aerosol transmission of the influenza A virus has been indicated by several studies (e.g. see review by Tellier, 2009), and emphasizes the dangers of highly infectious new strains of these respiratory pathogens in crowded indoor environments. Repeated outbreaks of new influenza A strains in the 20th century produced pandemics in 1957–8 (H2N2), 1968–9 (H3N2), and 1977–9 (H1N1) (Michaelis et al., 2009), as world human population ballooned to exceed 6 billion (Table 1). This new disease pattern has accompanied a time of unprecedented human hypermobility, energy use, technological development and environmental impact on the biosphere, a collective phenomenon that has come to be termed the “Great Acceleration” of Anthropocene time (e.g. Waters et al., 2016; Syvitski et al., 2020; Zalasiewicz et al., 2021).

Over the first two decades of this century outbreaks of influenza A have continued and have been joined by the appearance of no less than three new human respiratory zoonotic coronaviruses with pandemic potential (Table 1). Whereas SARS-CoV-1 was brought under control, and MERS-CoV has so far been of limited spread, SARS-CoV-2 rapidly fulfilled its pandemic threat. This latest pandemic is unlike any other in history. The SARS-CoV-2 pathogen entered an unprotected urban-based human population approaching 8 billion individuals (Table 1), many of whom were accustomed to traveling on what by the beginning of 2020 amounted to almost 40 million airline flights annually. Lessons regarding the likelihood of aerosol transmission of “flu-like” respiratory viruses had not been well learnt, and as a result the respiratory disease COVID-19 claimed over 4 million lives worldwide by August 2021 and will kill many more before controlled by mass immunisation.

Among the various immediate environmental impacts of the ongoing COVID-19 pandemic, many are associated with waste generation and disposal. Some of these have been positive, such as the sudden cleansing effect on highly polluted rivers such as the Ganges (Ganga) during the 2020 industrial and touristic shutdown (Shukla et al., 2021). Other impacts in contrast are clearly detrimental, such as the sudden dramatic increase in use of personal protective equipment. It can be calculated for example that over 1.5 trillion facemasks are currently adding to waste plastic worldwide, accompanied by their associated toxic additives such as organophosphate esters and heavy metals (e.g. Prata et al., 2020; Fernández-Arribas et al., 2021; Sullivan et al., 2021). While it is conceivable that an abrupt change in plastic waste output on this scale could be registered in the sedimentary record (Zalasiewicz et al., 2021).
et al., 2016), most immediate effects will be short-lived and probably quickly overtaken by the attempted re-establishment of the economic status-quo. Potentially more promising candidates for fossilising the memory of the COVID-19 crisis in deep time may derive from our societal response to changes in atmospheric chemistry. During the 2020 pandemic lockdown “anthropause” (Rutz et al., 2020) many urban populations experienced noticeable changes in air quality. Levels of NO₂ for example fell by as much as 30%–50% in cities such as Delhi, Madrid and Wuhan, mainly because of road traffic decline (e.g. Querol et al., 2021; Shi et al., 2021). Large areas of the world recorded similarly significant reductions in ambient PM₂.₅ (Bonardi et al., 2021), and CO₂ emissions from fossil fuel combustion fell by an estimate 2.6GtCO₂ in 2020, around 7% below that of 2019 (Friedlingstein et al., 2020; Le Quéré et al., 2021). Although such a drop in anthropogenic greenhouse gas emissions is unprecedented, it is not in itself a signal of significant change. It translates to a slowing of atmospheric CO₂ growth by less than 1 ppm in 2020, and the rebound is already in place (Monroe, 2021). However, there are signs that institutional policies in response to the COVID-19 crisis and a “new normal” environmental awareness in a globally networked population are stimulating an accelerating effort to tackle climate change (e.g. World Economic Forum: Boccaletti, 2020). In this context 2020–2021 may become to be seen as a pivotal moment, galvanised by the experience of COVID-19, that will hold the key to “bending the Keeling Curve” enough to change Anthropocene history.

7. Concluding remarks

The term Anthropocene in geoscience describes an Earth system state in which human activities have become predominant drivers of modifications in the stratigraphic record and has been recommended by a globally networked population for inclusion in the Earth’s stratigraphic record (Barnosky et al., 2011). This reflects the transforming reality of our technologically accelerated, globalized world in which we are responsible for an ongoing Great Extinction event during an experiment on wildlife habitat invasion, zoonotic disease patterns, oceanic ecosystems, atmospheric chemistry and climate. As a species, we are increasingly conscious of this, and thus increasingly capable of taking economic and political decisions that will affect future environmental outcomes more positively than has so far been the case. A recent European Parliament report observes: "The COVID-19 pandemic is an example of the inextricable links between human health and the ecosystem health (EU, 2021). Thus, although the impact of the COVID-19 crisis on the atmospheric CO₂ Keeling Curve for 2020 may appear minor, this does not mean that nothing has changed. The entire human race is suddenly much more aware of the importance of good air quality, whether it be indoors, well ventilated and viraeresol-free, or outdoors and uncontaminated by fossil fuel combustion emissions. Just as there is ever-increasing pressure to act on climate change and urban air pollution, there are already strong calls for a fresh start in the way we think about indoor air, ventilation, and minimising the spread of infectious diseases (e.g. Morawska et al., 2021; WHO, 2021a). Enhanced societal awareness of airborne particle issues has been aroused as never before by our changing "anthropause for thought" experience of COVID-19, and is being aided by a better appreciation of modern bioaerosol science won from a spirited burst of open access publications (e.g. Jimenez, 2020; Bourouiba, 2021; Burridge et al., 2021; Tang et al., 2021b, and many others). This is an opportunity for serious progress in air quality issues. Our reaction to the current pandemic and its airborne transmission is likely to have consequences because of its universal nature: to re-quote the Hippocratic Treatise of Polybus, written around 2,400 years ago, it involves that which we all use the most; and this is what we breathe.

Declaration of Competing Interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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