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# AN INTERPRETIVE SURVEY AT THE TIME OF COVID-19

By

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# The Great Influenza Pandemic of 1918–20: An interpretative survey in the time of COVID-19

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**Abstract:** The Great Influenza Pandemic of 1918-20—commonly known as the Spanish flu—infected over a quarter of the world's population and killed over 50 million people. It is by far the greatest humanitarian disaster caused by infectious disease in modern history. Epidemiologists and health scientists often draw on this experience to set the plausible upper bound (the 'worst case scenario') on future pandemic mortality. The purpose of this study is to piece together and analyse the scattered multi-disciplinary literature on the pandemic in order to place debates on the evolving course of the current COVID-19 crisis in historical perspective. The analysis focuses on the changing characteristics of pathogens and disease over time, the institutional factors that shaped the global spread, and the demographic and socio-economic consequences

**Key words:** Spanish flu, COVID-19, pandemic, infectious

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# The Great Influenza Pandemic of 1918–20: An interpretative survey in the time of COVID-19*

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## 1 Introduction

As of this writing, the world is in the grip of a virus outbreak, from which no country will emerge unscathed. Since a cluster of infections was reported in the Hunan Seafood Wholesale Market in China on 30 December 2019, coronavirus (COVID-19) has spread at an alarming speed in almost every continent except Antarctica. COVID-19 is a virus that has the inherent feature of a high mutation rate with the possibility of efficient transmission from person to person and is becoming more virulent (Wang et al. 2020). Thanks to marshalling of scientific resources, large-scale testing, and social distancing, some countries have been able to contain the spread of the virus, but even in those countries there is a lingering fear of a second wave, which in some cases has already eventuated. Spread of the virus is accelerating in developing countries, where both pharmaceutical and non-pharmaceutical defences are far less effective, and poverty and comorbidity (prevalence of other infectious diseases) provide ideal conditions for rapid spread. Because the world economy has become irrevocably interdependent, no country can win the fight against the virus on its own. Baring the possible yet unpredictable situation of the virus running its own course, the pandemic will end only when the infection rate is brought down to a manageable level, an effective vaccination is found, and most of the world population is vaccinated.

The purpose of this paper is to piece together and analyse the scattered multi-disciplinary literature on the Great Influenza Pandemic of 1918–20 (commonly known as the Spanish Flu) to place in historical perspective the current debates on the evolving course of the COVID-19 pandemic and its socio-economic implications. The 1918–20 pandemic, the deadliest humanitarian disaster in modern history with a death toll of at least 50 million worldwide, is considered by many epidemiologists and public health authorities as the 'worst-case scenario' in developing pandemic preparedness plans.[1] Pandemics are defining events in human history with lasting effects on the economy and society. History may not repeat itself, but historical information offers valuable insights that are highly relevant to today's concerns. It is important to set pandemics against a historical background and discuss the implications in terms of continuing changes within the context of a shifting global landscape. Reasoning by historical analogy does not, of course, provide definitive lessons but helps to identify areas where more thinking and research are required for designing evidence-based public policy intervention.

For over seven decades, the 1918–20 pandemic remained a 'forgotten human catastrophe' in social sciences and public policy discourse, presumably because of two related reasons: overshadowing by World War I and the Eurocentric nature of scholarship during the colonial era. The first two waves of the pandemic

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[1] The 1918–20 pandemic was caused by a member of the H1N1 family of influenza viruses. All influenza pandemics since that time, and almost all cases of influenza worldwide (except human infections from avian viruses such as H5N1 and H7N7), have been caused by decedents of the 1918 virus, making the 1918 virus the 'mother of all pandemics' (Taubenberger and Morens 2006: 70).

occurred in the final year of World War I.[2] In the warring Western nations, the death rate of the pandemic was much smaller than the war death rate. Therefore, the trauma of the war overwhelmed the memories of the pandemic in people's minds. At the same time, socio-economic implications of the pandemic for present-day developing countries (most of which were colonies of the Western nations) that bore the brunt of the death toll remained virtually beyond the focus of public policy debate and scholarship. Consequently, study of the pandemic remained largely a by-product of works by epidemiologists and virologists, whose prime goal was to discover why it had been so lethal and to find ways to prevent the recurrence of a pandemic of similar proportions, and the ancillary writings of a few medical historians (Phillips 2004; Burnet 1979).

Even in medical research, interest in the pandemic waned from about the late 1940s because of the growing complacency rooted in medical advances that allayed fears about a comparable future lethal pandemic.[3] For instance, in the early 1950s, Sir MacFarlane Burnet, the Australian pioneer in modern influenza research and Nobel Laureate, wrote in the second edition of his magnum opus of infectious disease, 'In many ways one can think of the middle of the twentieth century as the end of one of the most important social revolutions in history, the virtual elimination of infectious diseases as a significant factor in social life' (Burnet 1953: 3). Twenty years later, the fourth and the last edition of the book concluded, 'The most likely forecast about the future of infectious disease is that it will be very dull' (Burnet and White 1972: 263).

There has been a revival of multi-disciplinary interests in infectious diseases and learning from the 1918–20 pandemic over the past three decades. This was propelled by a significant increase in morbidity and mortality during the normal flu season in countries in the northern hemisphere and, more importantly, the outbreak of several influenza epidemics, which had the potential to gain pandemic proportions around the world at a remarkably shorter frequency.[4] Fear of a coming influenza pandemic has motivated epidemiologists and health economists to draw on the 1918–20 pandemic to set a plausible upper bound.[5] Health administrators in many countries draw on this information when developing pandemic preparedness plans (Chandra and Christensen 2017; Gulland 2016; Richard et al. 2009; Nickol and Kindrachuk 2019; Moxnes and Christophersen 2008). The evolving body of knowledge on the propagation of the COVID-19 pandemic and the concern about a possible second wave are heavily based on the experience of the Great Influenza Pandemic (Ferguson et al. 2020; Jones 2020; World Economic Forum 2020; Wang et al. 2020).

This paper begins with a stage-setting discussion on the origin and global propagation of the 1918–20 pandemic. The next section surveys intercountry differences in mortality and morbidity rates using a synthesis of data compiled from a comprehensive survey of the literature. Policy responses to the pandemic and socio-economic implications of the pandemic are discussed in the two subsequent sections. The paper

[2] World War I started on 28 July 1914 and ended in November 1918.

[3] The identification of a virus responsible for influenza in 1933 (by three British medical researchers, Wilson Smith, Christopher Andrews, and Patrick Laidlaw) paved the way to develop treatments to combat the illness. Discovery of penicillin, sulphonamides, and other potential antibiotics helped treat bacterial infection. The death toll of the 1957 Asian flu was no higher than those of any 'ordinary' influenza year, even though there was no reason for believing that the virus was of lesser virulence than the 1918–20 virus, presumably because of advances in medicine in the intervening 40 years (Burnet and White 1972).

[4] These included outbreak of Hong Kong flu (1997), the severe acute respiratory syndrome (SARS) (2002–03), the Middle East respiratory syndrome (MERS) (2012), and swine flu (2008–10) (da Costa et al. 2020; Ewald 2011; Garrett 2005).

[5] For instance, Murray et al. (2006), by relating mortality records of 27 countries during the 1918–20 pandemic to their population in 2004, predicted a death toll of 62 million deaths of a future pandemic of similar magnitude. An update of this estimate to the 2017 world population using data for 47 countries by Barro et al. (2020) suggests a death total of 150 million. A comprehensive survey of influenza epidemics by two prominent epidemiologists infers that 'Even with modern antiviral and antibacterial drugs, vaccines and preventive knowledge, the returning of a pandemic virus equivalent in pathogenicity to the virus of 1918 would kill over 100 million people worldwide' (Taubenberger and Morens 2006).

ends with some concluding remarks on lessons from the pandemic that can be applied when navigating the current COVID-19 pandemic.

## 2 Origin and global spread

The first recorded case of fatality in the 1918–20 pandemic was at the Funston Army Camp in eastern Kansas, USA, which fed a constant stream of men to other military bases in America and Europe during World War I. However, there is no absolute certainty as to where the virus originated. Burnet (1953) concluded that the evidence was 'strongly suggestive' of the pandemic originating in the United States. Taking cue from Burnet, medical historian John M. Barry (2004a, 2004b) traced its origin to Haskell County in northwest Kansas where 'farmers lived in close proximity to pigs and fowl, with cattle, pigs, and poultry everywhere'. Recruits from Haskell County arrived in Funston between 28 February and 2 March 1918. Two suggested alternative sources of origin are the Shanxi province in China (from where Chinese labourers carried the virus to France) (Langford 2005; Humphries 2014) and a British army camp at Étaples in France (Honigsbaum 2020). However, these claims have faded from attention as support for the United States as the ultimate origin of the virus has become the generally accepted view of medical historians.

The pandemic spread in distinct waves, with the number of waves and the timing of each varying among countries and regions. Countries in the northern hemisphere and Asian countries who sent troops to the war experienced three waves: March–August 1918, late August–December 1918, and early 1919 until about May 1919. In the first wave, the virus rapidly spread from Funston to other military bases in the United States during March 1918. The first outbreak in Europe occurred in Brest, France, in early April, where US troops embarked. From the European war front the virus quickly spread from France to Britain, Italy, Spain, Germany, Russia, and other countries in Europe over the following two months. There were recorded cases of infection in India, China, Singapore, and Indonesia by late May through troop movements.

News of the flu and the ravages caused in the trenches of the Western Front and war camps in the United States remained censored in the United States and the warring nations to avoid damaging soldiers' morale. The media obtained news of the flu from war-neutral Spain where there was free media, even though the virus only reached there in May. Hence, the moniker 'Spanish influenza'[6] has stuck to the 1918–20 pandemic to this day. The news from Spain added attraction in the media because Spanish King Alfonso XIII was infected with the virus, along with his Prime Minister and some members of the cabinet (Spinney 2017).

The infection was mild in the first wave, and the death rate was similar to that of normal seasonal flu. However, by late August 1918, the virus had mutated to a more deadly and contagious form, instigating the deadly second wave. The three port cities—Freetown, Sierra Leon; Brest, France; and Boston, Massachusetts—were the initial hotspots. During the next three months, the disease swept not only North America and Europe but also the entire world as far as the Alaskan wilderness and the most remote islands of the Pacific (Burnet and Clark 1942). October 1918 was the month with the highest fatality rate of the whole pandemic. The disease was of sufficient virulence to cause death within a few days of the development of symptoms (*The Economist* 2018; Wever and van Bergen 2014). However, many victims of

[6] In 2015, the World Health Organization (WHO), in consultation and collaboration with the World Organisation for Animal Health (OIE) and the Food and Agriculture Organization of the United Nations (FAO), issued guidelines on *Best Practices for the Naming of New Human Infectious Diseases* with the aim to minimize unnecessary negative impact of disease names on trade, travel, tourism, or animal welfare and to avoid causing offence to any cultural, social, national, regional, professional, or ethnic groups. These guidelines, which we follow in this paper, stipulated that disease names should not refer to specific places, people, animals, or food. https://www.who.int/topics/infectious_diseases/naming-new-diseases/en/

the first wave had become immune to the virus and showed significant resistance to the second wave, providing strong evidence that the deadly virus was a variant of the first one (Gladwell 1997; Taubenberger 2003).

By December 1918, North America and most parts of Europe were free of flu. But a third wave struck in January 1919 when the world was still recovering from the second wave. The virus had mutated again and was less virulent than that encountered in the second wave but much more severe than that of the first wave. The third wave peaked in the United States and Europe in January and February when the Paris peace negotiations were underway. Some analysts treat the third wave as 'a normal series of trailer outcomes' (Patterson and Pyle 1991: 4). However, it is almost certain that it was clearly a continuation of the pandemic: 'the abnormally high proportion of deaths among young adults, the unique characteristics of the second wave (to be discussed below), continued right through the third wave' (Crosby 2003: 203). According to most historical records, the pandemic was over in the northern hemisphere by May 1919.

The timing of onset and duration of the pandemic varied significantly in the rest of the world. In Africa, only north Africa, and belatedly a corner of southeast Africa, experienced the first wave (Phillips 2017). However, between mid-August and late September 1918, the virulent second wave invaded the African continent through the war-swollen seaports (Free Town, Cape Town, and Mombasa) and via rivers, colonial roads, and rail networks (Phillips 2017; Patterson and Pyle 1983; Patterson 1983; Ranger 1992; Ohadike 1991; Pankhurst 1977). In India, the pandemic lasted in most provinces well into 1919 and gave high mortality in that year in Bengal and the United Provinces; local outbreaks continued throughout the country during the next two years (Sen 1967)[7]. In Indonesia, the first case of infection was reported in July 1918, but the real onset of the pandemic was in September 1918. The impact was most intense during eight weeks from late-October until early December 1918, and it took until September 1919 before the mortality rate returned to the levels of the 1912–17 average (van der Eng 2020).

The virus did not reach Japan or countries and territories under Japanese colonial rule (Korea, Taiwan, Kuang-Tun Leased Territory, Sakhalin Island, and South Sea Islands) until October 1918. The first deadly wave there was from November 1918 to January 1919, and the second wave started in December 1919 and lasted in some parts of the country as long as June 2020 (Hayami 2015). In some countries (e.g., Scandinavia, some South Atlantic islands, and some Latin America countries) the pandemic persisted into 1920. In the Peruvian capital Lima, the pandemic peaked in early 1920 and lasted through 1921 (Chowell et al. 2014). Thanks to the remote location, the news of the pandemic reached Australia, New Zealand, and the Pacific Islands in late August 1918. Australia managed to avoid an outbreak until early 1919 (when the third wave set in) through stringent marine quarantine (see below). There is evidence that countries that were exposed to the first mild wave gained considerable immunity in the second wave (Hayami 2015; Rice 1988).

The 1918–20 pandemic was the first historical illustration of 'the unification of the globe by disease' in human history (Ladurie 1981). At the height of the second wave in October 1918, the disease had spread to all human-inherited parts of the world up to the Alaskan wilderness, other than New Guinea and a few other isolated places. The two other mega pandemics of human history are the Plague of Justinian (around 540–541 AD) and the Black Death of 1347–59. Though as deadly as they were, these pandemics were largely confined to geographically contiguous countries and countries linked by main land trade routes (Alfani and Murphy 2017; Scheidel 2017). Thus, even at a time when naval transport was the sole conduit of human interaction across seas, the 1918–19 pandemic vividly illustrated that the 'whole civilised world can be regarded as a single epidemiological unit as far as influenza is concerned' (Burnet 1953: 285).

Another important trait of the 1918–20 pandemic, which has remained a puzzle to both medical researchers and historians, is its brevity (Ranger 2003). As noted, the pandemic dissipated within a period less than one year in Europe and North America. Even in the global periphery, it did not last more than one and a half years after allowing for the time lag involved in spread.

[7] Based on data from Government of India (1924).

What explains this sudden disappearance of the virus? The often-held media view is that people would have gotten accustomed to living with it (Kolata 2020). This is, however, not consistent with the available vital statistics. In all countries for which data are available, the death rate had returned to the average pre-pandemic levels after the 'recorded' ending dates. Because the pathogen causing the disease was not even known at the time, it is certain that the pandemic did not end as a result of medical intervention. It seems that the spread of the virus ran its *natural* course within that short period because of its unique genetic characteristics that still remain a puzzle to epidemiologists (Taubenberger 2003). A survey of historical records dating back to the early middle ages also suggests that epidemics and pandemics were 'not spontaneously persistent' and most of them were short-lived (Ladurie 1981: 37).

# 3 Counting the disaster

There are three commonly used measures for assessing the humanitarian effect of an infectious disease: morbidity—individuals in the population who are infected with the virus (the 'attack rate'); mortality—the number of deaths among the infected; and case mortality—deaths (fatality) among positive cases. Our knowledge of the 1918–20 pandemic is largely confined to data on mortality rates. Early estimates of deaths were based on administrative records and media reports with 'informed' adjustment for underreporting. Most of the recent estimates are 'excess mortality' calculations based on comparison of recorded mortality during the pandemic years with those for a selected number of pre- and post-pandemic years (the number of people who died over and above what might have been expected in a 'normal' [non-pandemic] year). A number of countries in Eastern Europe, Arab Middle East, and Africa are not covered in these estimates because of unavailability of data. Data on morbidity and case mortality are sparse and even less reliable.

Pandemic mortality data, even in countries with vital statistics recording systems, are not accurate, and the degree of accuracy varies among countries. There are identification issues particularly at the initial stage that result in contamination of influenza death with other courses of death because diagnostic criteria for influenza and pneumonia were vague. The magnitude of the pandemic in itself could have distracted accurate recording because physicians and nurses had much more compelling demands than to keep accurate records. Defining the pandemic's duration (when it exactly started and/or stopped) was also arbitrary. In most countries the available data from administrative records are concentrated heavily in the last third of 1918 and the first half of 1919. For these reasons, the best way to get a more accurate picture is to look at excess mortality (Aron and Muelbauer 2020). Accuracy of excess mortality estimates are subject to the quality and coverage of the vital statistic collection systems in any given country. For instance, in 1918, the area from which the US Census Bureau received transcriptions of all death certificates contained only 77.8 per cent of the total estimated population of the nation (Barry 2004a). Also, the extent to which the estimated 'excess' is contaminated with other deaths could vary from country to country depending in particular on comorbidity (concurrent susceptibility to other diseases). In this section, we treat the available estimates at face value to understand the order of magnitude of the death toll and intercountry differences.

The first estimate of the global death toll of the 1918–20 pandemic was in a study sponsored by the American Medical Association (Jordan 1927). This study estimated the global death toll at 27.6 million (Table 1). This estimate was based on data from North America, Europe, and a few large British colonies for which some administrative records were available. In the first major review of the literature on influenza epidemics, Brunet and Clark (1942) stated that the figure could be anywhere between 25 million and 50 million. Patterson and Pyle (1991: 15) came up with an estimate of 24.7 million to 39.3 million while suggesting a 'conservative total of roughly 30 million victims'. Johnson and Muller (2002) updated Patterson and Pyle's figures to 32.4 million to 41.3 million. This was based on a comprehensive synthesis of the literature up to about 1998. Johnson and Muller present these figures with the caveat that 'even this vast figure may be substantially lower than the real total, perhaps as much as 100 per cent understated' (p 105).

Table 1: Global deaths of the 1918–20 influenza pandemic

| Study | Number of countries covered | Deaths, millions | Death rate (%)[1] |
|---|---|---|---|
| Jordan (1927) | ? | 21.6 | 1.2 |
| Burnet and White (1972) | ? | 25.0–50.0 | 1.4–2.7 |
| Patterson and Pyle (1991)[2] | 44 | 24.7–39.3 | 1.3–2.2 |
| Johnson and Mueller (2002)[3] | 57 | 32.7–42.6 | 1.8–2.3 |
| Barro et al. (2020)[4] | 48 | 40 | 2.2 |
| This study (Appendix Table A-1)[5] | 72 | 34.5–43.9 | 1.9–2.4 |

Note: [1] the world population (2017) used in calculating the death rate is 1,832 million (from the UN population database, https://www.un.org/en/development/desa/population/publications/database/index.asp). [2] Based on these figures, the authors suggest 'a conservative world total of roughly 30 million victims' (p 15). [3] Based on these estimates, the authors suggest: 'It [total deaths] was of the order of 50 million. However, ... even this vast figure may be substantially lower than the real toll, perhaps as much as 100 percent understated' (p 105). [4] Total estimated deaths for 48 countries (which accounts for 80 per cent of world population) extrapolated to the total world population. [5] Johnson and Muller (2002) data updated based on studies published during 1988–2020. The total for 72 countries (accounting for 94 per cent of world population) is extrapolated to the world population.

Source: as noted on the table.

In this study we have updated Johnson and Muller (2002) estimates based on a comprehensive survey of the studies published during 1998–2020 combined with data for some countries from hitherto unpublished official sources. Estimated total deaths in the 72 countries we have covered is between 32.5 million and 41.3 million. When extrapolated pro-rata to the total world population, the total global death toll is between 34.7 million and 44.0 million (Table 1). These numbers can possibly understate the true figure to the extent that the average death rate of countries not covered (in particular, countries in the Arab Middle East and some countries in Africa and Eastern Europe) exceeded the average global death rate used in the extrapolation[8]. However, after allowing for such underestimation we believe that 100 million deaths, the figure suggested by Johnson and Muller (2002) as a possible upper bound and widely cited in the recent literature, is well off the mark.

If we take 50 million as a reasonable number, the number of total deaths of the 1918–20 pandemic is comparable to or higher than the other two mega pandemics in recorded human history, the Plague of Justinian and the Black Death (Alfani and Murphy 2017), that occurred during the pre-modern era. The mortality rates of the latter two pandemics would have been far greater given the low world population at those times.

A hallmark of the 1918–20 pandemic compared to other influenza pandemics before and after it is the high mortality rate. The seasonal flu is 'generally a mild, almost pleasurable experience, an opportunity for an unexpected fortnight's holiday from work' (Burnet 1953: 276), with a mortality rate of only about 0.01 per cent (Taubenberger 2003). By contrast the median global mortality rate of the 1918–20 pandemic was about 2.3 per cent (Table 1). Why was the mortality rate so high?

The most ubiquitous explanation relates to 'war conditions' encountered during the time of the pandemic. The crowding of troops in war camps and ships, medical camps, and hospitals and the upheaval of normal life during war time provided the best possible opportunity for the spread of airborne pathogens (Wever and van Bergen 2014; Morens and Fauci 2007; Honigsbaum 2020). Close quarters far from help, such as

[8] There is anecdotal evidence that some countries in the Middle East (e.g., Lebanon, Libya, and Iraq), which are not covered in our estimate, suffered significant influenza mortality through troop movement during the final year of World War I (Gassem 2020; Steinberg 2002). Also, data are not available from Lusophone countries (Portuguese-speaking countries) and many francophone countries in Africa, which were presumably affected by the Africa-wide spread of the pandemic (Phillips 2017).

ships on the high seas and war camps, were an ideal setting for the propagation of infectious disease. Massive troop movement across the seas was the main conduct of the global spread of the virus.

Some researchers in the field of evolutionary theory of virus postulate that the mutation of the 1918 virus into the more virulent form that caused the deadly second wave was a direct response to conditions on the Western Front, where the pool of hosts was not mobile and limited as they were packed into war camps and trenches (Ewald 1994, 2011; Roes 2018; Woolhouse et al. 2002). These conditions allowed individuals immobilized by illness to be transported repeatedly from one cluster of susceptible hosts to another, in trenches, tents, hospitals, and trains. Because of this, there was less evolutionary pressure on the virus to moderate its virulence through *natural selection*[9]. Random genetic mutation could, in principle, produce a more lethal virus, but pathogens that are too lethal might not survive long enough to effectively transmit to different populations if the host is not mobile. The mutated (second wave) virus had the capacity to penetrate through the entire respiratory tract of the infected person and to trigger a cytokine storm, which ravages the immune system (Viboud et al. 2013; Tsoucalas et al. 2016).

The 'war conditions' provided the setting for the propagation of the disease and perhaps the mutation of the virus into a deadly form. However, the 1918–20 pandemic was not merely a 'war pandemic' (Jefferson and Ferroni, 2009: 1). Although the initial epicentre of the pandemic was the warring nations in western Europe and North America, the pandemic gained its own momentum in the 'global periphery', the present-day developing countries[10], most of which at the time were colonies of Western powers. As we will see in the next section, the pandemic mortality rate was almost four times higher in these countries, which accounted for over 90 per cent of the total estimated deaths. Malnutrition and comorbidity (concurrent prevalence of other diseases such as malaria, cholera, and tuberculosis [TB]) added to people's susceptibility to the virus. Near famine conditions and food shortage made matters worse in India, Iran, and some other countries (Afkhami 2003; Arnold 2019; Liew 2007; Mills 1986).

The overall high global death rate undoubtedly mirrored the state of clinical drug development at the time. There were no antidotes to influenza or secondary infection with bacteria. While the virus initiated the illness in every case, it was almost always a superimposed bacterial infection that was finally responsible for fatality. Given the unavailability of treatment for influenza or bacterial infection, the best the medical profession could do was prescribe palliatives or provide patients with supportive care to facilitate natural recovery.

While the poor state of clinical drug development was obviously responsible for high death rates in all countries, there was a vast gap in the provision of institutionalized healthcare between Western countries and developing countries. As has been well documented, imperial powers paid little attention to public health in their colonies (Patterson and Pyle 1983; Balfour and Scott 1924; Arnold 2019; Killingray 2003; Tomkins 1994; Wibowo et al. 2009). Whatever healthcare facilities were available were confined mostly to large cities. In rural areas, where the majority of people lived, infected people and families had to fend for themselves. Most people turned to traditional medicine or folk remedies and practices. In the absence of public healthcare and a social safety net, the rate of mortality was increased by 'innate susceptibility' and a lack of care when all members of a family were incapacitated.

In African countries, the response of the colonial rule to the influenza crisis was 'at worst murderous and at best irrelevant' (Ranger 1992). African people even shunned available facilities and palliatives because of the anticolonial mentality and their belief in the failure of Western medicine. The European origin of the disease and the failure of European medicine to effectively treat the virus gave rise to an influential anticolonial campaign in most African countries that had a significant lingering impact on public health in these countries well beyond the pandemic years (Arnold 1988; Ohadike 1991; Phillips 2017).

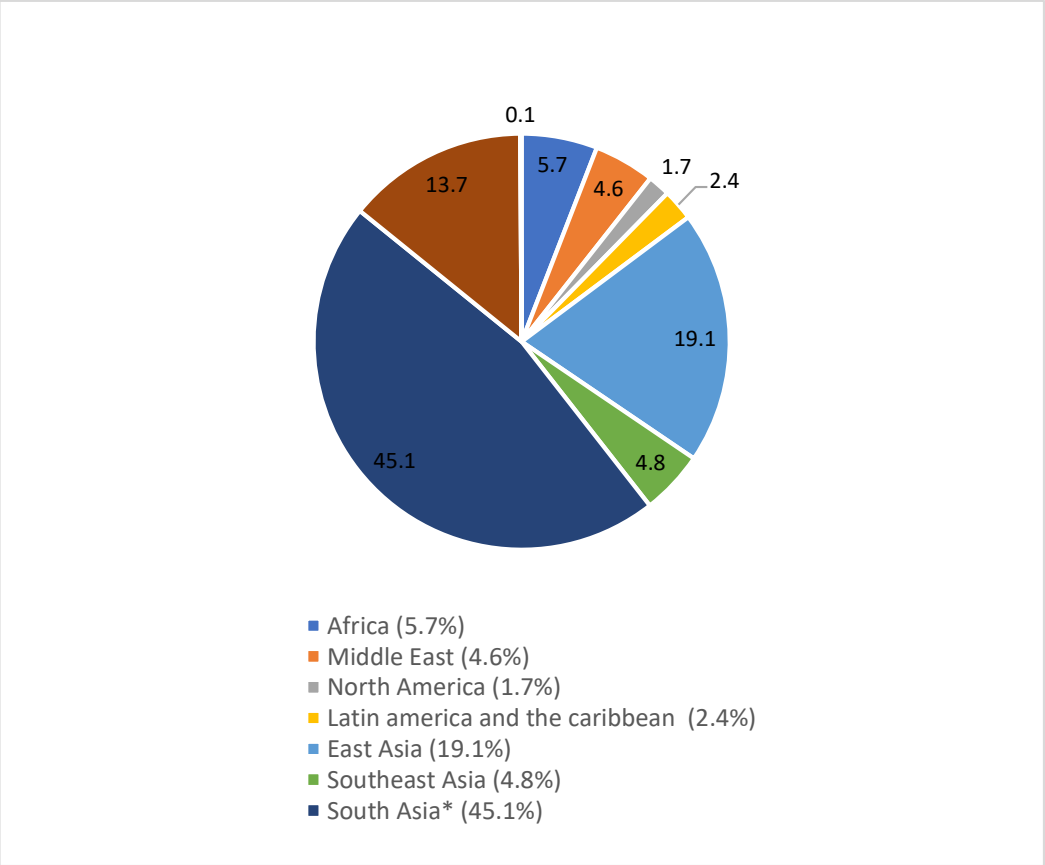
[9] Natural selection of a virus is its mechanism of evolution—the change in the heritable traits that shape its survival and spread.

[10] In the rest of the paper these countries are referred to as 'developing countries' (and the rest as 'developed countries') for brevity.

## 3.1 Geographical patterns

Notwithstanding the 'European' origin of the 1918–20 influenza pandemic, developing countries accounted for 86 per cent of total estimated pandemic deaths of about 42 million[11] (Appendix Table A-1, Figures 1 and 2). The median death rate of developing countries was 3.1 per cent compared to a global figure of 2.3 per cent and the developed-country average of about 1 per cent. There was huge variation in the mortality rate among countries and geographical regions. Countries in Asia and Africa suffered the highest mortality rates—3.0 per cent and 3.5 per cent, respectively. Among all countries, the highest mortality rate was recorded in Western Samoa, where nearly one-quarter of the native population died, followed by Iran (14.5 per cent). In the Western countries, the death rate varied from 0.2 per cent to 1.5 per cent, with some European countries directly involved in the war recording rates at the upper end.

Figure 1: Mortality of the 1918–20 influenza pandemic by major world geographical regions (%)

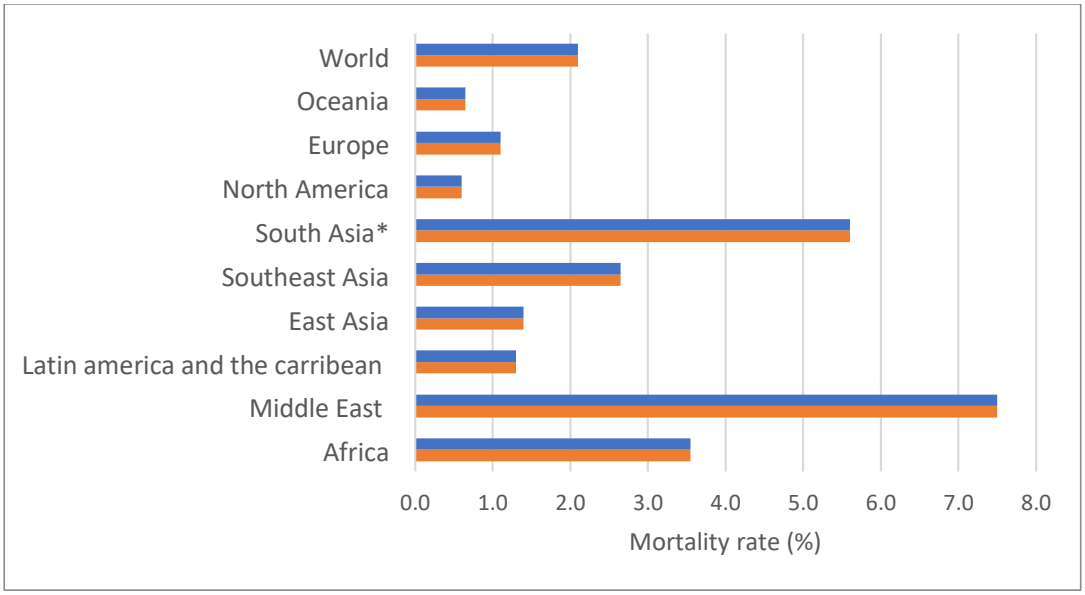



Note: * British India (includes present-day Myanmar, Pakistan, and Bangladesh) and Ceylon (Sri Lanka) (0.2%).

Source: author's illustration based on data from appendix Table A-1.

[11] Median values of the reported lower- and upper-bound figures are used in the discussion in this section.

Figure 2: Mortality rates of the 1918–20 influenza pandemic by world geographical regions (%)




Note: * British India (includes present-day Myanmar, Pakistan, and Bangladesh) and Ceylon (Sri Lanka).

Source: author's illustration based on data from appendix Table A-1.

There was a heavy concentration of global pandemic deaths in British India, encompassing the present-day India, Pakistan, Bangladesh, and Myanmar (Burma)[12]. British India accounted for about 45 per cent of total estimated deaths. The official estimate of total deaths (Government of India 1938) that covered only the British-controlled provinces (which accounted for about 75 per cent of the population of British India at the time) was from 12 million to 14 million. Davis (1951) estimated deaths using the excess death method for the entire British India at 18.5 million (a death rate of 6.2 per cent) with the qualification that it could be as high as 20 million. After adjusting Davis's estimate for possible overestimation bias, Mills (1986) came up with an estimate of 17 million to 18 million. The latest estimate for British India by Barro et al. (2020) is 16.7 million. When estimated at the mid-point of Barro et al.'s and Davis's estimates (reported in the table), British India accounted for 45 per cent of total global pandemic deaths[13]. There were notable spatial differences in deaths in the subcontinent, with western, northwestern, and central regions recording much higher mortality rates (Reyes et al. 2018; Chandra and Kassens-Noor 2014; Mills 1986; Gill 1928). The relative importance of differences in diurnal temperature, comorbidity, food scarcity, and other related factors that determined people's susceptibility to the virus remains an unresolved issue. Unlike in Western countries, in India mortality rates in rural areas far exceeded those of the cities (Davis 1951; Wakimura 1966).

There was no centralized collection of vital statistics in China during this period. The estimates of 5 million to 9 million given in Table 2 (from Patterson and Pyle 1991) is a guess based on the available data on pandemic death rates in neighbouring countries. These figures give a median death rate of 1.4 per cent, which is much lower than that of India (5.3 per cent) and even the global average (2.3 per cent). Iijima (2003), by applying mortality rates estimated from records of Chinese Maritime Customs in Chinese treaty ports, Hong Kong, Taiwan, and Kwangtung Leased Territory, came up with an even lower estimate of between 1 million to 1.28 million. Based on reports of foreign missionaries and information from *China Medical Journal* (a journal edited by foreign experts), Jordan (1927) also observed that the influenza outbreaks in China 'were mild and did not spread widely'. Information put together from contemporary newspapers and administrative records of some large cities by Cheng and Leung (2007) and Langford (2005) suggests

[12] Burma was governed by the British as part of India until 1937. Total population in Burma at the time was about nine million (three per cent of the total population of British India).

[13] We have not considered estimates less than the official figure, which we believe provides a realistic lower bound.

that the disease was widespread in China but was relatively mild and less lethargic than elsewhere in the world. These studies provide three possible reasons for China's presumably relatively low incidence of pandemic death: effectiveness of traditional medicine, immunity gained by many people in China from previous influenza outbreaks (which had been a recurrent occurrence in the county), and limited mobility of people within inland China because of a poorly developed transport system.

In Indonesia, vital statistics of Dutch administration relate almost exclusively to Java Island. Even for Java, data are difficult to analyse not only because of substantial underreporting of the number of deaths but also because of considerable confusion about the accuracy of the population base. Colonial records are virtually complete for the European population and probably nearly complete for the group classified as 'other foreigners' (almost entirely ethnic Chinese) but are deficient in coverage for the remainder of the population (Nitisastro 1970; Gardiner and Oey 1987). Brown (1987) estimates the death toll at 1.5 million. This was an informed guess based on administrative records and case studies (mostly qualitative) of some parts of the other islands. Based on a careful study of vital statistics from the village registers of the Dutch administration and with adjustments for underestimation of population numbers using data from the 1930 population census, van der Eng (2020) estimated pandemic-related excess mortality in Java as 1.13 million and extrapolated it to 1.63 million for the whole country on a pro rata basis. The estimate gives a death rate of 3.2 per cent, which is the second-highest in Asia after India[14].

The coverage of African countries in Table A-1 is incomplete. The country coverage is dominated by those under the British colonial administration. No data are available for all Lusophone Africa (i.e. Angola, Mozambique, Guinea Bissau, Cape Verde, Sao Tome, and Principe and Equatorial Guinea) and most Francophone African countries. The available circumstantial evidence suggests the worst-hit countries were the ones with port cities: Sierra Leone, South Africa, Kenya, Cameroon, Gold Coast, Gambia, Tanganyika, and Nyasaland. Land-locked countries that were not linked with port cities through rivers and railroads (mostly the countries in northern and central Africa) were less affected (Philipps 2017; Gewald 2007).

## 3.2 Morbidity and case mortality

The available estimates of morbidity and case mortality rates of the 1918–20 pandemic are summarized in Table 2. Almost one-third of the world population was infected with the virus, and of them about 2.5 per cent succumbed to death. There are notable differences between developed countries and developing countries listed in the table both in terms of morbidity and case mortality. However, the difference is much larger relating to the case mortality rate. A much larger share of those infected died in developing countries. This is consistent with the view that pandemic death is a lot more than just a 'one germ – one disease affair' (Jefferson and Ferroni 2009). Factors, other than the virulence of the virus, mediated through poverty and deprivation seem to have played a vital part in determining survival of the infected. The median case mortality rate in India was as high as 10 per cent compared to the global average of 2.5 per cent. Interestingly, the case mortality rate among the jail population in India during the pandemic was much lower (4.4 per cent)[15], most likely because people in jail were less likely to die from the disease because of the availability of medical care (and also perhaps because they were better fed) (Mills 1986).

[14] The extrapolation of the Java figure to the entire country was justified based on qualitative information from a major study undertaken in 1920 by a committee appointed by the public health administration of colonial govern to study the pandemic that the impact of the pandemic was roughly the same in the outer islands.
[15] Estimated from data reported in Table 1 in Mills (1986).

Table 2: Morbidity and case mortality during the 1918–20 influenza pandemic (%)

| World/country | Source | Morbidity | Case mortality |
|---|---|---|---|
| World | Burnet and Clark (1942) | 32.0 | 2.5 |
| Australia | National Museum of Austrasia (undated) | 40.0 | 0.7 |
| USA | Frost (1920)[1] | 28.0 | 1.6 |
| India | Sen (1923) | 50.0–80.0 | 7.4–11.8 |
| Japan | Hayami (2015) | 38.2 | 1.2 |
| Korea | Lim (2011) | 44.0 | 2.6 |
| Nigeria | Ohadike (1991) | 50.0–80.0 | 3.5–5.6 |
| New Zealand | *Medical Journal of Australia* (1919)[2] | 33.0 | 1.2 |
| The Philippines | Gealogo (2009) | 40.0 | 6.3 |
| Thailand | Royal Thai Government *(1919)* | 27.8 | 3.3 |

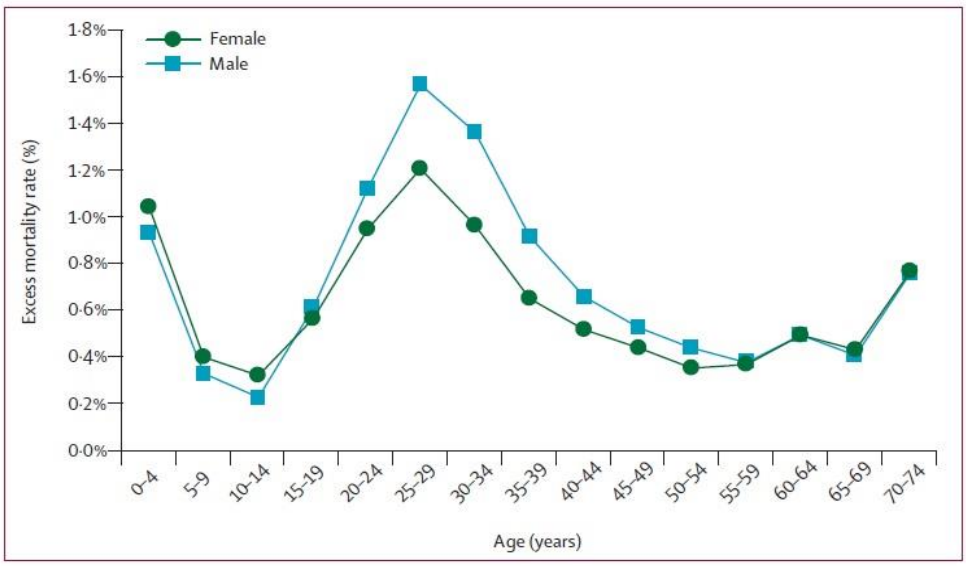
Note: [1] based on a survey of 130,000 people in 11 cities. [2] The data cover the second wave.

Source: as noted on the table.

### 3.3 Patterns of fatality

The age profile of usual (seasonal) influenza mortality is such that victims, as a rule, are very young and very old, with a higher survival rate for those in between, showing a U-shape pattern. This was also the pattern in the first wave of the 1918–20 pandemic. In the second and third waves, however, the pandemic resulted in a higher than expected mortality rate amongst young adults (Burnet and White 1972; Gagnon et al. 2013; Viboud et al. 2013). Both infants and elderly did die in large numbers, but the great spike came in the middle: the death-age graph looked like a 'W' with the middle spike taller than the two sides. Two-thirds of the victims were adults aged between 20 and 40 years. This pattern was similar cross the world. Also, the overall pattern was similar for both male and female victims (Figure 3).

Figure 3: Median mortality by age and sex for the 1918–20 influenza pandemic[1]




Note: [1] based on data from 13 countries for which age-specific mortality data are available.

Source: Murray et al. (2006), Figure 1. Reproduced with permission.

Brunet and Clark (1942: 90–9) came up with the postulate that the unusual 'W' shape age profile of second- and third-wave mortality can be explained in terms of the nature of the mutated virus and the way the body's defence mechanism changes with age. The mutated virus was of a very virulent strain that had the

capacity to penetrate through the entire respiratory tract of all ages, and the young adults' bodies reacted so vigorously to the deadly virus that the reaction drowned them. A young adult has a peculiar ability to produce intense localized inflammatory response (not generalized) similar to the kind of reaction needed to deal with a localized injury such as broken bones, torn ligaments, and wounds. When the stimulus is generalized in 20–40 year olds, as in the case of infection with the virulent flu virus, the intense inflammation in the lungs causes a springtide of fluids to overwhelm the lungs. After about 40 years of age, this ability to produce extreme inflammation declines, and the ability to survive generalized infection rises as the ability to survive localized injuries declines.

In Western countries the death rate amongst females was noticeably lower than that of males (Figure 3). However, the data for India revealed the reverse pattern in all age brackets: female mortality was higher (Chandra et al. 2012; Mills 1986). It is postulated that this pattern is attributed to two innate societal factors: because of sex bias in family care, women were relatively more malnourished compared to men and hence less resistant to the flu virus[16], and women had to bear the burden of taking care of the sick in the family. Interestingly, in Japan, the Indian pattern of higher female mortality was also observed but, in this case, only for the age brackets of 20 to 34 (Hayami 2015). This presumably reflects greater involvement of women in this age group in household chores and greater susceptibility of pregnant women to virus infection (see below).

According to data from the United States, the most vulnerable among the young adult women were pregnant women. The death rate amongst hospitalized pregnant women varied between 23 per cent and 71 per cent, and foetal death was encountered in over one-quarter (26 per cent) of pregnant women who survived (Barry 2004a: 239). At the height of the second wave (September and October 1918) there was a 50 per cent increase in still births in the United States (Jordan 1927: 24). In India, the disease was particularly virulent for women of reproductive age (Mills 1986; Chandra et al. 2012).

Throughout the world, a higher incidence of mortality was experienced by those classes and communities that normally had the weakest grip on life (Mills 1986; Pool 1973; Kraut 2010; Mamelund 2006; Sydenstricker 1931). In the United States, newly arrived migrants suffered more (Kraut 2010). Also, death rates were much higher among native Indians (Crosby 2003). A survey conducted by the US Public Health Service in nine urban localities in the United States showed that there were marked and consistent differences in the incidence of morbidity and mortality among persons of different economic statuses. The lower the economic level, the higher the attack rate. This pattern was found to persist even after allowances had been made for factors of colour, sex, age, and certain other conditions (Sydenstricker 1931). A study of social status and pandemic deaths in Norway found that household wealth and social status of place of residence had a significant and independent effect on mortality after controlling for age, sex, and marital status. In Paris, domestic servants figured prominently among the dead compared to affluent citizens (McBride 1976). In New Zealand, the death toll was much higher among the Maori population compared to the non-Maori majority population[17] (Pool 1973; Rice 1988: Chapter 6). In Ceylon (Sri Lanka), indentured labourers and their families in the plantation sector suffered more than the other communities in the country (Chandra and Sarathchandra 2014; Langford and Storey 1992).

## 4 Public policy response

The available evidence on government policy to contain the spread of the virus ('non-pharmaceutical interventions [NPIs]') comes mostly from studies conducted in the United States. Crosby (2003) provides a comprehensive state-by-state study of social distancing measures in the United States. According to his findings, New York was less affected than other East Coast metropolises because of a solid foundation in

[16] This explanation is consistent with the phenomenon of 'missing women' (Sen 1992).
[17] The official Maori death rate (2.26 per cent) was five times the European rate (Rice 1988: 102).

public health and administrative experience gained from its 30-year war on TB. Crosby (2003) provides comprehensive state-by-state coverage of social distancing measures put in place in the United States: closure of schools, theatres, and places of worship, restrictions on mass gatherings, quarantines at port and railway stations, and public information campaigns. Philadelphia (East Coast) had the most severe experience of any major American city whilst San Francisco suffered the most amongst West Coast cities. The measures introduced early and kept in place after the danger had passed played an import role. Based on an interstate comparative analysis of the nature and effectiveness of NPIs, Crosby came up with the general inference that the demands of national security, a thriving economy, and public health are rarely aligned, and elected representatives defending the first two undermine the third.

According to the findings of Bootsma and Ferguson (2007), time-limited interventions of social distancing reduced total mortality only moderately (perhaps 10–30 per cent), and the impact was often limited because interventions were introduced too late and/or lifted too early. Even in the absence of government intervention, individuals spontaneously reduced their contact rates in response to high levels of mortality during the pandemic. If interventions were very effective at containing the virus at an early stage, the likelihood of a second peak in mortality at a subsequent wave was higher because there was a larger number of susceptible people in the population who had not been previously exposed to the virus.

Markel et al. (2007) estimated the impact of school closures, prohibition of public gatherings, and quarantine isolation in 43 US cities during the second wave of the pandemic (September 1918–February 1919). These NPIs helped flatten the curve in the sense of reducing the peak deaths to average deaths, but the effect on overall deaths was small and statistically insignificant because the interventions had an average duration of only around one month. Barro (2020) re-examined the possible endogeneity bias involved in the results of Markel et al. (2007) using distance from Boston to each city as a measure of how early flu evidence tended to become relevant for cities. The impact on the relative peak death rate is negative and statistically significant; the impact on the cumulative death rate is negative but not significant.

The experiences of countries in Oceania, in particular the difference between Australia and New Zealand, figure prominently in discussions on the effectiveness of quarantine measures during the pandemic (Burnet and Cark 1942; Johnson 2006). Both Australia and New Zealand had the advantage of being 'island' nations situated far away from the epicentre of the pandemic. However, the death rate of New Zealand turned out to be much higher than that of Australia. The virus reached both countries in August 1918 at the end of the first wave. Australia immediately responded with strict maritime quarantine in all ports. The one-week quarantine requirement was applicable to both incoming and outgoing vessels. Thanks to these measures, Australia was not affected by the deadly second wave of the pandemic, and the country celebrated the armistice in November having nothing to fear of the virus. *The Medical Journal of Australia* (1919) reported that 'the Federal Quarantine Service will be in the proud position of having achieved the greatest triumph of its kind in the history of epidemiology'. However, Australia was affected by the third wave after the quarantine requirements were relaxed in early January. New Zealand, where there was no systematic quarantine (and/or social distancing requirements), was affected by both the second and third waves (Rice 1988, 2003). The overall pandemic death rate in New Zealand was 0.65 per cent compared to 0.3 per cent in Australia. Australia's death rate was one of the lowest recorded of any country during the pandemic (Table 2).

Regular shipping service from Auckland (*Talune*) infected Western Samoa, Fiji, Tonga, and Nauru. The death rates in these island nations were much higher compared to other islands—the Gilbert and Ellice groups, the New Herbrides, and Norfolk and Solomon Islands—exclusively served by Australian ships (Burnet and Clark 1942: 14). American Samoa escaped infection because of strict quarantine imposed by the US naval administration (Tomkins 1992).

Given the state of medical research and absence of a known medical antidote to influenza or its complications at the time, pharmaceutical intervention to the pandemic was limited only to general healthcare and nursing with some untested palliatives. As already noted, even general healthcare was largely available only in Western countries. The only recorded evidence of the use of vaccination during the

pandemic comes from Japan and Australia. In Japan the government launched a nationwide vaccination campaign. At a time when the influenza virus was unknown and the medical profession's knowledge was confined only to associate bacteria, most of the vaccines used there were arbitrary mixtures of attenuated pneumococci, streptococci, and Pfeiffer's bacilli (Hayami 2015). It is not known whether these had any effect on preventing secondary pneumonic infections. In Australia, Commonwealth Serum Laboratories (CSL) (set up during World War I) produced an experimental vaccine to address secondary bacterial infections. Between 15 October 1918 and 15 March 1919, CSL issued three million doses for free distribution. Later evaluation found that the vaccine was effective in preventing death in inoculated individuals, presumably by preventing secondary bacterial infection (National Museum of Australia, undated).

# 5 Socio-economic impact

The 1918–20 pandemic had a significant impact on medical research and on public healthcare reforms in most countries during the ensuing years. It stimulated advances in medical research, leading to development of medicine and preventative vaccines for influenza and other respiratory diseases. In the sphere of healthcare, the 1920s saw many governments in Western countries embracing the concept of socialized healthcare and improvement in health data reporting systems with emphasis on epidemic preparedness. These important developments are beyond the scope of this paper[18]. In this section our sole focus is on the socio-economic effects of the pandemic.

## 5.1 Population dynamics and human capital development

The pandemic, given its unprecedented morbidity and mortality rates with devastating impact on most fertile and productive population cohorts, must have had significant demographic consequences that extended well beyond the pandemic years. However, in the Western countries that were directly involved in the war it is extremely difficult to separate out the demographic effects of the pandemic from those of the Great War (Johnson 2006). Therefore, the available evidence on demographic effects of the pandemic comes largely from other countries that were not directly involved in the war.

Chandra and Yu (2015a, 2005b) examine pandemic-associated mortality and subsequent demographic dynamics in Taiwan and Japan. In both countries, there was a significant reduction in births after the pandemic mortality peaked in 1919, primarily through the mechanism of reduced conception and embryonic losses during the first month of pregnancy. In India the age and sex selection effect of the pandemic, a combination of concentration of fatality in prime ages and the greater fatality rate among women, resulted in a decline in the birth rate in 1919 of around 30 per cent (Mills 1986). However, there was a population spurt during the ensuing years of the interwar period, presumably because the spike of mortality during the pandemic left a diminished but healthier population (Klein 1988). Norway experienced a significant reduction in the fertility rate during the pandemic, but the fertility rate recovered within about two years, reaching higher levels compared to the pre-pandemic years (Mamelund 2004). It seems that, as in India, the pandemic created a healthier population that was able to reproduce at a higher rate by pruning the less fit.

What are the long-term implications of the pandemic for the quality of the labour force over and above the direct impact on population dynamics? Almond (2006) and Almond and Mazumder (2005) have addressed this issue by testing the 'fetal origin' hypothesis, which postulates that certain chronic health conditions of

[18] These developments have been well documented (see Burnet 1979; Spinney 2017: Chapter 19; Crosby 2003: Chapter 13).

people in adult life can be traced to the course of their intrauterine development (Barker 1992). The test involved comparing human-capital traits and labour market performance of a cohort in utero during the height of the pandemic and a cohort in utero prior to the pandemic. The test was conducted using a unique data set compiled by combining the US 1960–80 census microdata and data on maternal and health conditions provided by US Vital Statistics. The findings suggest that the former cohort was characterized by lower educational attainment, increased rates of physical disability, accelerated adult mortality, lower income, greater dependence on higher transfer payments, and lower socio-economic status compared with those in the latter cohort. In addition, the results indicate that persons born in states with more severe exposure to the pandemic experienced worse outcomes than those born in states with less severe pandemic exposures. Beach et al. (2018) revisited Almond's finding (2006) by using a new data set that permitted testing whether the cohort that was in utero during the pandemic could have shown worse outcomes in adulthood because they came from families with low socio-economic status and not because of their exposure to flu while in utero. Their findings confirmed that the inferences hold after controlling for the socio-economic status of the parents.

Subsequent studies conducted in Brazil (Nelson 2010), Switzerland (Neelsen and Stratmann 2012), and Taiwan (Lin and Liu 2014) have found evidence of similar long-term damage from prenatal exposure to the 1918–20 pandemic influenza[19]. However, a recent study of Sweden has come up with mixed findings (Helgertz and Bengtsson 2019). Both men and women with fetall exposure in the pandemic experienced higher morbidity in ages 54–87. For males, exposure during the second trimester also affected mortality caused by cancer and heart disease. However, the study failed to provide consistent evidence supporting any long-term consequences of fetal exposure relating to adulthood income, social status, and occupational attainment.

## 5.2 Economic growth

Barro et al. (2020) undertake a multicounty (42 countries) panel data analysis of the impact of pandemic deaths on economic growth rates of per capita gross domestic product (GDP) and private consumption (after controlling for World War I deaths). The results suggest that the pandemic reduced real per capita GDP by six per cent in the typical country. The results for the consumption effect are broadly similar to those of the growth effect. Their results also suggest a mild long-term impact of pandemic death returns on financial assets. The contraction in economic growth and consumption was accompanied by a substantial short-term decline on real return financial assets (measured by returns on stocks and government bonds). Long-term return on these assets also would have been reduced by about 26 percentage points, but this impact was not statistically significant.

Jorda et al. (2020) examine medium- to long-term impacts of return on assets of the 1918–20 influenza pandemic and 18 other disasters (both wars and pandemics, each of which accounted for at least 100,000 deaths) using a data set dating back to the 14th century and covering six countries (France, UK, Germany, Italy, Netherlands, and Spain). Real return on assets is measured in terms of real rates of interest on long-term debt. The results indicate that pandemics significantly depressed real asset return, and this impact lingers on for about four decades. Labour scarcity resulting from pandemic mortality and morbidity that drive up real wages relative to the cost of capital and reshuffling of household asset portfolios towards greater precautionary savings are identified as underlying causes. Interestingly, the comparative analysis of the study reveals a contrasting *positive* effect of wars on asset returns, presumably because capital is destroyed in wars (resulting in an increase in the relative cost of capital in post-war recovery) but not during a pandemic.

[19] See Almond and Currie (2011) for a survey of these and other economic literature on testing the fetal origin hypothesis.

The relative wage effect underlying the Jorda et al. (2020) interpretation of the negative effect of pandemics on asset returns is consistent with the findings of Garrett (2009). His comparative study of the impact of mortalities of the 1918–20 pandemic and World War I on wage growth in the US manufacturing sector indicates that the states and cities that experienced greater influenza mortalities experienced higher wage growth relative to the states and cities that experienced greater war casualties.

Brainerd and Siegler (2003) undertook an econometric analysis of post-pandemic growth in the United States using state-level data. The study found that the growth rate is *positively* associated with the death rate, possibly reflecting the capacity of a society to bounce back after a violent shock and/or weaker people could have been purged by the flu. However, this study has failed to separate the impact of the pandemic from effects of World War I. Peace dividends of ending the war—reallocation of resources locked in the war effort and demobilization of the armed forces—could have significantly contributed to stronger growth (Asquith 2020).

Correia et al. (2020) examine the impact of geographic variation in mortality during the 1918 flu pandemic in the United States and its impact on economic performance, with emphasis on the possible mitigating effects of NPIs. The results suggest that more exposed areas experienced a sharp and persistent decline in economic activity. In particular, the pandemic reduced manufacturing output on average by 18 per cent. Cities that implemented early and extensive NPIs suffered no additional adverse economic effects from implementing those measures when compared with cities that implemented measures later or not at all. Overall, NPIs not only lower mortality but also mitigate the adverse long-term economic consequences of a pandemic. Cities that intervened earlier and more aggressively grew faster after the pandemic was over.

At the time of the 1918–20 pandemic, the present-day developing countries were predominantly agrarian economies. An interesting issue relating to the impact of the pandemic on these countries is therefore how labour shortages impacted agricultural production. Schultz (1964) probed this issue using data for Indian agriculture before and after the pandemic. This study was essentially a test of the theory of surplus labour of Arthur Lewis (1954)[20], which was at the heart of development economics in the immediate post-war decades. Schultz (1964) considered Indian agriculture during the pandemic as an ideal case study of the surplus labour theory because the influenza pandemic did not affect animals and other factors of production except the number of workers and the mortality rate in rural areas during the pandemic far exceeded that of the urban areas.

The test involved a comparison of deaths attributable to the pandemic and change in acreage sown to crops in 10 provinces between 1916–17 and 1919–20. The results suggest that the reduction in the agricultural labour force by about eight per cent as a consequence of the pandemic was associated with a sharp reduction in acreage allocated to crops from 265 million in 1916–17 to 228 million in 1918–19 (pp 66–7). He therefore concluded, 'It would be hard to find any support in these data for the doctoring that a part of the labour force in agriculture in India at the time of the epidemic had a marginal productivity to zero' (p 67).

In a critique of Schultz (1964), Sen (1967) argues that Schultz's test is inclusive for two reasons. First, the concept of surplus labour assumes a pattern of family-wise labour withdrawal in response to some economic incentives. For example, migration to wage employment outside that withdrawal of labour force from the rural economy will keep the total output unchanged, whereas the influenza pandemic was not only unevenly distributed over families within a given region, it was unevenly distributed between different regions in the given province. Second, he cast doubt on the relevance of Schultz's finding for understanding the impact of the pandemic on the agricultural economy of India. The post-pandemic observations of Schultz's analysis were in 1919–20, which was the year immediately following the havoc, so there was little time allowed for the market to achieve the necessary allocation of land or labour, even if such a market

[20] The theory postulated that the agrarian economy of the typical developing country is characterized by a massive pool of surplus labour, and hence, marginal productivity of labour is zero. Therefore, this part of the labour force is wholly redundant and is available for industrialization and other activities in the modern sector at no opportunity costs except the cost of transfer.

worked well. Quoting data from the Census of India in 1921, Sen noted that the pandemic was not even over in the year of the observation.

Interestingly, Brown (1987) has reported evidence, which is in sharp contrast to that of Schultz, relating to the impact of pandemic mortality on agricultural production in Java and Madura in Indonesia. The area under paddy and other smallholder crops in 1919 was generally higher than in 1917, thereby continuing the upward trend established in the years immediately preceding the pandemic. How come the labour shortages did not result in a contraction in the area under cultivation as Schultz observed in India? Brown alludes to two possible reasons: the agricultural sector had surplus labour (characterized by zero marginal productivity of labour) in the agricultural sector and/or the diminished rural labour force might have been supplemented by drawing in workers from other sectors of the economy.

# 6 Concluding remarks

The Great Influenza Pandemic of 1918–20 is by far the most devastating of all pandemics in modern history. Even at a time when shipping was the sole mode of international travel, the 1918–20 pandemic was able to affect the whole civilized world. A high fatality rate, which was 5–20 times higher than expected in a normal influenza season, the high incidence of death among people of prime age, and a short and abrupt ending were its hallmarks that have not yet been fully understood by epidemiologists. The last year of World War I coincided with the first year of the pandemic and facilitated the initial spread of the virus and perhaps mutation of the virus into a more virulent form, but the pandemic was not simply a war epidemic. The brunt of the cost of the pandemic was borne by the countries in the world periphery (the present-day developing countries). Even though the pandemic abruptly ended in less than one year in the Western world, it lingered on later into 1920 in some parts of the developing world.

We live in a time when the world is far more susceptible to 'unification by disease' than it was in the early 20th century. The speed and extent of the global spread of the COVID-19 virus has therefore been unparalleled in world history. However, contrary to some alarming predictions, the mortality rate of the COVID-19 pandemic is unlikely to be as high as in the 1918–20 pandemic. Modern medicine is now much better prepared to deal with a pandemic. The socio-economic cost is likely to be determined by morbidity rather than mortality and economic disruption associated with social distancing and shutting down economic activity. The impact of morbidity on the demographic dynamic and human capital development is likely to dominate the post-pandemic research agenda of many disciplines.

Perhaps the most important lesson arising out of the 1918–20 pandemic for the COVID-19 policy debate relates to unequal distribution of burden between developed and developing countries. At a time when existing therapeutic intervention made little difference, intercountry differences in the death toll of the global influenza pandemic of 1918–20 was strongly mediated by the state of economic advancement, with poverty and deprivation and associated comorbidity playing a vital role over and above the virulence of the virus. Whether advances in medical intervention will assist in reducing the unequal distribution of the overall impact of the pandemic and mortality outcomes remains debatable and to be seen.

Prospects for development of a vaccine for COVID-19 seem promising. A pivotal policy issue is how new medicines and vaccines against COVID-19 are going to be distributed. Issues of patenting and the monopolistic practices of pharmaceutical markets that are dominated by multinational enterprises are directly relevant here (Stiglitz et al. 2020). For instance, relating to the HIV/AIDS pandemic, quickly identifying the virus and developing antiretroviral therapy (ART) was widely cited as an example of the power of modern medical research (what can happen in an outbreak of infectious disease) (Deaton 2013). However, it took many years for the benefits to trickle down to the developing world. An estimated 37.9 million people are still living with HIV/AIDS (Fauci and Lane 2020), and 570,000 to 1.1 million people are still dying from the disease annually, most of them in developing countries, with African counties alone

accounting for 61 per cent of deaths[21]. Under the normal market mechanisms, large stocks of antibiotics or antivirals are unlikely to be available in most low-income countries.

The remarkable propensity of viruses to mutate and unpredictable dissipation of a pandemic as seen in the course of the 1918–20 pandemic have implications for public policy relating to vaccine development, production, and equitable worldwide distribution. A vast body of knowledge on the structure and habits of the flu virus has evolved over the past hundred years, but the virus can frustrate our best efforts by changing its salient features faster than we can recognize the changes, essentially meaning that 'the virus will be one step ahead of the vaccine manufacturers' (Burnet and White 1972: 212). Moreover, the possibility of abrupt dissipation and uncertainty of duration of the pandemic makes massive investment in vaccine development risky from a private enterprise perspective[22]. The upshot is that governments and international developmental organizations have to play an important role to alleviate the commercial risk involved in the development and production of vaccines that the pharmaceutical companies are naturally hesitant to bear on their own.

In a context where the whole world is in the midst of a pandemic, responsible national and international developmental organizations have to take risks on behalf of the public. It is better to err on the side of overreaction than underreaction. Even if the wealthy countries emerge victorious from the COVID-19 war, the victory would be short-lived in this era of economic and social globalization if the pandemic continues to cause havoc elsewhere.

[21] https://www.who.int/gho/hiv/epidemic_status/deaths_text/en

[22] Following the outbreak of swine flu in 1976, pharmaceutical companies spent millions of dollars in production and distribution of a new flu vaccine, but the flu did not trigger a pandemic as predicted (Crosby 2003: xii; Sencer and Millar 2006).

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## Appendix

Table A-1: Mortality of the 1918–20 influenza pandemic[1]

| | Death (thousands)[1] | Death rate (%) |
|---|---|---|
| ***Africa*** | 2,207–2,268 | 3.5–3.6 |
| Belgian Congo | 300.0 | 5.0 |
| Botswana | 7 | 4.0 |
| Cameroon | 25 | 4.5 |
| Gambia | 8 | 3.7 |
| Gold Coast (Ghana) | 89–100 | 3.9–4.4 |
| Kenya | 150 | 5.8 |
| Madagascar | 119 | 3.5 |
| Mauritius | 12 | 3.2 |
| Nigeria | 455 | 2.4 |
| Senegal | 38 | 3.0 |
| Sierra Leone | 46 | 3.0 |
| South Africa | 300 | 4.4 |
| Southern Rhodesia | 38 | 4.4 |
| ***Middle East*** | 1,800 | 7.5 |
| Egypt | 139 | 1.1 |
| Iran | 1,662 | 14.8 |
| ***Latin America*** | 900–1,053 | 1.1–1.5 |
| Argentina | 10.2–46 | 0.1–0.5 |
| British Caribbean | 30 | 1.5 |
| Caribbean other | 70 | 1.4 |
| Brazil | 180.0 | 0.7 |
| Chile | 20–35 | 0.5–0.9 |
| Guatemala | 49 | 3.9 |
| Columbia | 27 | 0.5 |
| Mexico | 300–500 | 2.1–3.4 |
| Peru[2] | 4 | 1.6 |
| Uruguay | 2.1–2.4 | 0.1–0.3 |
| Venezuela | 12 | 0.4 |
| ***Asia*** | 24,232–32,498 | 2.5–3.5 |
| *East Asia* | 4,664–10,305 | 0.9–1.9 |
| Japan | 453–517 | 0.8–0.9 |
| China | 4,000–9,500 | 0.8–2.0 |
| Korea | 185–235 | 1.1–1.4 |
| Taiwan | 26–53 | 0.7–1.4 |
| *Southeast Asia* | 1,719–2,049 | 2.4–2.9 |
| Indonesia | 150–163 | 3.0–3.3 |
| Malaysia | 40–43 | 1.2–1.3 |
| Philippines | 94–288 | 0.9–2.8 |
| Singapore | 5–7 | 1.3–1.8 |

| | | |
|---|---|---|
| Thailand | 80–82 | 0.9–1.0 |
| *South Asia* | 17,097–18,890 | 5.4–6.0 |
| Afghanistan | 320 | 5.5 |
| British India[3] | 16,700–18,500 | 5.5–6.1 |
| Ceylon (Sri Lanka) | 77–80 | 1.7–18 |
| | | |
| *North America* | 588–726 | 0.5–0.7 |
| Canada | 51 | 0.6 |
| USA | 537–675 | 0.5–0.7 |
| *Europe* | 4,997–5,694 | 0.5–0.7 |
| Austria | 21–98 | 0.3–1.6 |
| Belgium | 64 | 0.8 |
| Croatia | 109 | 3.6 |
| Denmark | 6 | 0.2 |
| Eire (Ireland) | 18 | 0.4 |
| England and Wales | 156–200 | 0.5–0.6 |
| Finland | 18–26 | 0.6–0.8 |
| France | 240 | 0.7 |
| Germany | 225–444 | 0.4–0.8 |
| Greece | 25 | 0.5 |
| Prussia | 237 | 0.5 |
| Hungary | 100 | 1.3 |
| Island | 1 | 0.6 |
| Italy | 390–501 | 1.1–1.4 |
| Netherlands | 48 | 0.7 |
| Norway | 15 | 0.6 |
| Portugal | 59–158 | 1.0–2.6 |
| Russia (USSR) | 2,760 | 1.5[4] |
| Scotland | 28–34 | 0.6–0.7 |
| Spain | 257–311 | 1.2–1.5 |
| Sweden | 34–28 | 0.6–0.7 |
| Switzerland | 23–26 | 0.6–0.7 |
| Turkey | 162–221 | 1.1–1.5 |
| *Oceania* | 42–45 | 0.6–0.7 |
| Australia | 15 | 0.3 |
| New Zealand | 44 | 0.6–0.7 |
| Fiji | 9 | 5.5 |
| Tonga | 1 | 3.9–7.8 |
| Western Samoa | 9 | 23.6 |
| Country total | 34,666–43,583 | 2.0–2.5 |
| World total[5] | 36,909–46,404 | 2.0–2.5 |
| | | |
| *Memorandum items*[6] | | |
| Developing countries | 29,745–37,864 | 2.7–3.4 |
| Developed countries | 5,020–5,719 | 1.0–1.1 |

Note: [1] regional totals include estimates by Johnson and Muller (2002) for other countries in the region (after deducting deaths of countries newly added). [2] Covers three main cities (Lima, Liquito, and Ica) only. [3] Includes Burma (present-day Myanmar). [4] Based on Russian-language sources summarized in Slomczynski (2012). [5] Country total extrapolated by the population share of the countries covered (94 per cent). [6] Countries classified based on the UN Standard Country Classification. https://www.un.org/en/development/desa/policy/wesp/wesp_current/2014wesp_country_classification.pdf

Source: Johnson and Muller (2002) (based on a survey of literature on the subject published during 1920 to 1998); Afkhami (2003) Iran; Andayi et al. (2019) Kenya; Ansart et al. (2009) Europe; Alexander (2019) Mexico; Barro et al. (2020) (43 countries); Chowell et al. (2011) Peru; Chowell et al. (2014) Chile; Feldman (2014) Argentina; Gealogo (2009) Philippines; Hayami (2015) Japan; Karsson et al. (2014) Sweden; Killingray (1994) Caribbean Islands; Kim (2011) Korea; Lee at al. (2007) Singapore; Liew (2007) Malaysia; Murray et al. (2006) (27 countries); National Museum of Australia (undated) and Curson and McCracken (2006) Australia; Royal Thai Government (1919) and Thongcharoen (2017) Thailand; Slomczynski (2012) Russia.