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The Spanish Flu and the Great War

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ABSTRACT: World War 1 exacerbated the cost of the influenza epidemic of 1918-19 in two ways. First, it facilitated the spread the flu virus through the movement of clusters of infected soldiers and sailors. Second, it constrained public health measures that would have reduced mortality (as during the Covid-19 epidemic). While there is no obvious way of estimating any resulting mortality, attributing even a modest share of the deaths caused by the flu to the war would significantly increase the civilian death toll.

Keywords: pandemic, influenza, World War I

JEL codes: N30, I18

> War is in a sense simply an incident which man foolishly permits to enter into that greater struggle with germ life.
>
> David John Davis, 'Bacteriology and the War' [1917]

The epigraph above comes from an article listing several infectious diseases that threatened the lives of troops during WW1. It appeared too soon to mention the deadliest of them all, the H1N1 virus responsible for the influenza pandemic of 1918-1919. Like Covi9 19 the 1918 flu virus was a zoonosis, i.e. an infection transmitted from animals (or in this case birds) to humans. Such viruses do not strike humans very often; when they do, it is a matter of luck how rapidly they spread and how severe they are. In this respect, the H1N1 influenza is the worst on record. About half of the U.S. military dead of WW1—43,000—were felled by it, and it also killed 30,000 French troops. Smaller death tolls of 20,000-25,000 and 8,000 have been cited for their fellow combatants from Germany and the United Kingdom.[1] Those numbers, of course, are only a tiny fraction of all deaths due to the pandemic. To what extent were the tens of millions of civilian victims of the flu war casualties too?[2]

In 1989 the great influenza pandemic of 1918-1919 was described by one of its most eminent historians as 'a forgotten pandemic' but, in truth, it had been subject to ebbs and flows of academic interest in the interim; much discussion and analysis in its wake followed by a virtual silence until the 1950s, spasmodic interest in the late 1950s and 1960s, and a reawakened interest in the 1970s, which has not ceased to grow since.[3] And rightly so, because the 1918-19 flu was more global in its reach than even WW1 and killed four or five times as many people, overwhelmingly civilians, as the fighting did soldiers. Although rurality and remoteness offered some protection from it, few places were safe.[4]

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[1] Darmon 2011: 166; Lahaie 2011; Michels 2010: 17; Wever and van Bergen 2014; https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2338238/pdf/brmedjo6781-0023c.pdf..

[2] This chapter was originally intended for inclusion in Ó Gráda 2024. For reasons explained there, it was decided in the end to publish it separately in this form.

[3] Crosby 1989; Phillips 2004; 2014b.

[4] McSweeney *et al*. 2007; Nishiura and Chowell 2008. See also Richard Gray, 'The places the escaped the Spanish flu', *BBC Future*, 24 October 2018.

A significant proportion of the world's population was infected and a higher proportion died in 1918-19 than during any other pandemic since the Black Death. Since the 1970s the pandemic has generated an enormous literature, spurred on in part by the impact of later pandemics but, perhaps, more so by the work of environmental historian Alfred Crosby. In recent years there have been several remarkable global histories of the flu, several excellent country studies, and a huge number of contributions to professional journals in disciplines ranging from epidemiology to economic history, and from geography to literary criticism.

Shortly before succumbing to influenza on 9 November 1918, the French poet Guillaume Apollinaire joked about the king of Spain: "Alfonso XIII has the Spanish flu. The news only half surprises us: a good king should have in mind to use only domestic products." The global character of the pandemic is reflected in its nicknames: the Spanish flu or the Spanish lady in Europe and America, 'the Naples Soldier' and the 'French flu' in Spain, 'the Bolshevik disease' in Poland, '*naya sardee ka bukhaar* (new winter fever)', 'Bombay fever', and 'war fever' in India, 'the German flu' and 'la dansarina' in Brazil, 'the German disease' in Italy, 'Brazilian influenza' in Senegal, 'blitz katarrh' and 'the Russian plague' in Germany, '*die kakie pes* (the khaki plague)' and '*die spaanse griep*' in South Africa, 'the Chinese sickness' in Russia, and 'the American disease' in Japan. And, like all great pandemics, that of 1918-19 spawned its share of conspiracy theories.

In what follows, our main interest will be in the link between the flu pandemic and WW1. But in order to understand that link, first we describe some features of the pandemic such as its symptoms, its waves, its incidence, its lethality, and its demography. Then we review the literature on its death toll. Next, we discuss the evidence on links between the spread of the pandemic and WW1. We also consider whether the war impacted on public health and the use of non-pharmaceutical interventions (NPIs).

A Brief Overview:

The flu struck in waves—two in some places, three in most, four in a few—and, like Covid-19, mutated between waves. The three waves struck in the spring of 1918, in September-November 1918, and in the spring of 1919. The first was mild and killed relatively few, though it confined many to bed for a few days. The second was the most lethal, while the third also exacted a significant toll in deaths. The virus had a mean incubation period of about five days. Most of those stricken by it survived, but for the minority who succumbed, death came quickly. It was not an easy death. The symptoms included shivering, severe headaches, pain in the legs and kidneys, followed by high fever accompanied by a hacking cough. A surgeon at Camp Devens in Massachusetts described how the virus did its deadly work among his patients:[5]

> These men start with what appears to be an ordinary attack of LaGrippe or Influenza, and when brought to the Hosp. they very rapidly develop the most vicious type of Pneumonia that has ever been seen. Two hours after admission they have the Mahogany spots over the cheek bones, and a few hours later you can begin to see the Cyanosis [bluish discoloration of the skin] extending from their ears and spreading all over the face.... It is only a matter of a few hours then until death comes...It is horrible... We have been averaging about 100 deaths per day... For several days there were no coffins and the bodies piled up something fierce...

On Armistice Day (11 November 1918) a nursing sister caring for British troops in France wrote: "We moved up to St André after the army went into Lille, and almost immediately we started taking in wounded and many ... who had Spanish influenza as well ... The boys were coming in with colds and a headache and they were dead within two or three days. Great big handsome fellows, healthy men, just came in and died. There was no rejoicing in Lille the night of the Armistice".[6] Those 'quick' deaths exclude those who succumbed to pneumonia, having apparently recovered from the flu.

[5] John M. Barry, 'How the Horrific 1918 Flu Spread Across America', *Smithsonian Magazine*, November 2017.
[6] As cited in Wever and van Bergen: 2014: 540.

The disease became notorious for striking down those in the prime of life. The usual explanation for this is that the more active immune systems of young adults made them more vulnerable. They fought the virus with cytokines and other toxins, but ended up fatally damaging their own lung tissues. Normally the cells involved in the immune response would release a set of proteins called cytokines to marshal the body's defence against infection. The problem is that cytokines also produce symptoms such as fever and inflammation which can have a detrimental effect in severe cases, and damage the lungs and kidneys. Whence the claim that young people with robust immunity are more vulnerable to the positive feedback loop known as a 'cytokine storm'.[7]

This feature was a key aspect of the epidemic's demography everywhere. Figure 1, based on data for the second and third waves in England and Wales, describes a familiar pattern whereby the standard U-shaped pattern of death rates by wage was converted into a W-shaped pattern, due to the greater vulnerability of young adults to the virus. What of gender? In a 2010 survey of the link between gender and influenza in general the World Health Organisation reported that death rates from H1N1 after puberty (15-44 years) were higher among males in thirteen relatively affluent countries for which age-adjusted mortality data were available.[8] This pattern is also reflected in Figure 1, which also shows that the female advantage extended into old age. Whether this was a universal pattern remains moot. In the city of Bombay, for example, mortality was higher among women, and this was "in agreement with the figures for the whole of India", whereas in New Zealand flu

[7] Short *et al*. (2018) sound an agnostic note about this traditional explanation, noting that the experimental studies which inform it used 'immunologically-naïve animals', very different from a large proportion of those humans facing the 1918 pandemic, who would have been previously exposed to other influenza viruses. More specifically, Short *et al*. maintain that those born before the so-called Russian influenza pandemic of 1889 would have had acquired antibodies against later influenzas, whereas those born thereafter (i.e. aged 30 or under in 1918) would have not. They also suggest that immune suppression due to recent measles infections might have weakened the resistance of young adults against H1N1 in 1918—but not older adults whose immunity would have protected them.

[8] WHO 2010: 9. In the US the rates were 672 and 498 per 100,000 for males and females respectively.

mortality was higher among white males, but there was no appreciable gender gap among the Maori.[9]

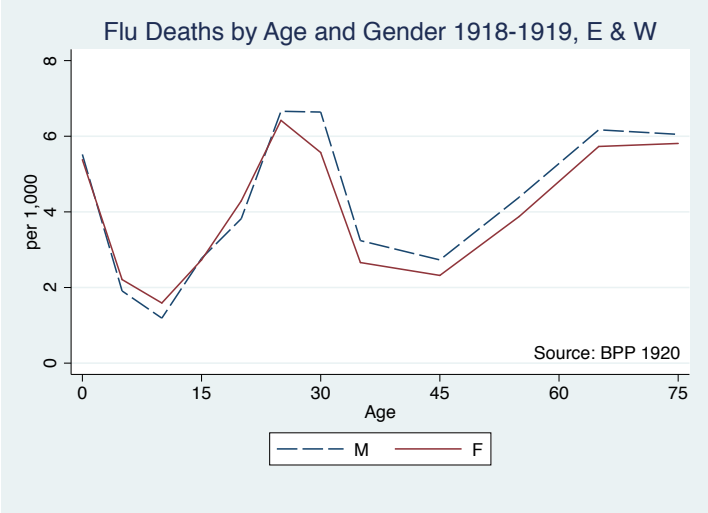

Figure 1. Deaths per 1,000 by Age and Gender in England and Wales
Note: Using 1921 population as denominator

In the wake of the pandemic a surgeon working for the US Public Health Service surveyed large numbers of people in Baltimore and San Francisco twice, in November-December 1918 and January 1919, in order to ascertain whether contracting the flu during the first wave conferred immunity in the second. He found that the small proportion of repeat cases was consistent with immunity being acquired by contracting the virus.[10] A series of studies in the late 2000s revisited this important issue, which gained renewed resonance during the Covid-19 epidemic of 2020-2022. In research on necessarily small numbers of elderly 1918 flu survivors Yu *et al.* and Ikonen *et al.*[11] claimed that antibody responses acquired in 1918 lasted a lifetime and protected very elderly people against infection during the pandemic of 2009. For Dr. Anthony Fauci, director of the US National Institute of Allergy and Infectious Diseases at the time, "This [was] the mother of all immunological memory".[12] Barry *et al.* provided

[9] Compare Phipson 1923: 517; Bengtsson *et al.* 2018: 2568; *British Medical Journal*, 7 August 1920, p. 211; Fornasin *et al.* 2018: 102; Summers *et al.* 2018: 60.
[10] Frost 1919: 318; Frost and Sydenstricker. 1919.
[11] Yu *et al.* 2008; Ikonen *et al.* 2010.
[12] Fauci is cited here: https://www.cidrap.umn.edu/news-perspective/2008/08/researchers-find-long-lived-immunity-1918-pandemic-virus.

evidence that contracting the disease during the first wave conferred a considerable degree of immunity against the more lethal second wave. A study of the Canadian military found that new recruits were more vulnerable to influenza, both in terms of infection and death, than seasoned soldiers. The author attributes this to the likelihood that earlier enlistment entailed immunity conferred by exposure to the first wave of the epidemic. In South Africa too, it is claimed that exposure to the first wave, which hit Durban in early September and spread from there to Natal, the northern Orange Free State, and the southern Transvaal, immunized people living in those areas to some extent against the second most lethal wave which entered South Africa from the south west via Capetown later in September. This could help explain the marked differential in the death rate from flu in the two areas (6-9 per cent against 1-2 per cent).[13] Such findings could mean that public health interventions aimed at curtailing the first wave in 1918 were counterproductive, in that they increased the lethality of the more dangerous second wave.[14] However, such interventions were very limited during the first phase of H1N1.

It is sometimes claimed that the 1918-19 flu virus discriminated between races and ethnicities. In Alaska the native population was much more severely hit than non-native; and in New Zealand the indigenous Maori were much more at risk than others. But it was easy to mistake economic disadvantage for race. In South Africa the flu-related mortality was highest in predominantly black districts, which convinced some contemporaries that blacks were "more susceptible to the disease than Europeans"[15]. But that ignores the greater poverty of the black population and its lack of access to medical services (for what they were worth). In US army camps, for example, black soldiers were more likely to die of the flu than white; but poorer living conditions, less nutritious food, and inferior medical facilities are more likely reasons for this outcome than racial susceptibility.[16]

Although the pandemic was global in its reach, it was quite uneven in its incidence. People living in poorer regions of the globe such as sub-Saharan Africa,

[13] Barry *et al*. 2008; Bogaert 2015; Phillips 2020.
[14] Rios-Doria and Chowell 2009.
[15] Phillips 1984: 155.
[16] Byerly 2010: 88.

India, Central America, the small islands of the south Pacific tended to be worst hit: Europe and North America least so. But there were contrasts within regions and countries too. The epidemic also had a significant, if not straightforward, socio-economic dimension. In Ireland, on the one hand, annualised mortality rates in the last quarter of 1918 did not seem to vary much across broad occupational groups: 31.9 per thousand among the families of professional and business classes, 26.7 per thousand in the middle classes, 29.7 per thousand in artisanal and petty entrepreneur households, and 38.5 per thousand in working class households. However, these broad categories conceal interesting intra-group variations. In the top group, the annualised death rate in "the clerical, legal, military officers and other professions" was 52.4 per thousand, whereas in the households of the mercantile and manufacturing elite, it was only 5.9 per thousand. What Ida Milne dubs "the real disaster" was in the subdivision for army, police, postal deliverers prison services, where the annualised death rate was 120.2 per thousand population. Moreover, county-level scatter plots suggest that there were strong links between poor housing conditions, population density, and the prevalence of manufacturing, on the one hand, and pandemic mortality on the other.[17] In Dublin in the last quarter of 1918 the death rate from influenza and tuberculosis ranged from 9 per thousand in relatively affluent suburban Blackrock Urban District and 9.5 per 1,000 in suburban Glasnevin-Finglas to 21.1 per 1,000 in the inner city North City West and 27.8 per 1,000 in North City East.[18]

In the UK there were proportionately more influenza deaths where the pre-pandemic death rate from all causes was higher, indicating that social disadvantage and influenza mortality were linked.[19] In the Norwegian capital of Kristiania (today's Oslo) apartment size and the social status of location had a significant impact on mortality, after controlling for several other factors.[20] In the case of Sweden, where the data are rich, there were clear cross-class differences, which were greater for men

[17] Milne 2018: 71-72.
[18] 'Influenza epidemic in Ireland: heavy death roll; efforts to combat the disease', *Irish Times*, 2 November 1918; The recent influenza epidemic: Dublin's death rate in the December quarter', *Irish Times*, 6 February 1919.
[19] Pearce *et al.* 2010: 93.
[20] Mamelund 2006.

than for women. Among males, farmers were markedly less at risk; low-skilled workers suffered more than either unskilled or skilled workers and white-collar workers, but the differences were not striking. Socio-economic status mattered, but it was not the full answer.[21]

In Bombay/Mumbai the death rates across different groups mirrored mortality rates in normal times: communities "whose collective hold on life [was] slight" suffered most. The following data refer to deaths per thousand:[22]

Europeans	8.3	Indian Christians	18.4
Parsees	9.0	Caste Hindus	18.9
Eurasians	11.9	Muslims	19.2
Jews	14.8	Low caste Hindus	61.6

In New Zealand, the socioeconomic divide is reflected in the contrasting mortality rates—seven to one—of Maori and Pakeha (white), but there is somewhat more to it than that: "Maori tended to live in isolated, rural areas, and so weren't exposed to the flu's first, milder wave earlier in the year, and so probably missed out on some immunity. Also, they had lost their land and had very poor nutrition and living conditions, tuberculosis was rife, and Maori had high rates of smoking tobacco. All of that likely contributed."[23] In Sweden the socioeconomic gradient was complicated by the nature of the workplace; the bigger the factory one worked in the more likely one was to succumb from the flu.[24] Factors such as geography, institutions, and culture also surely mattered. In Ireland, for example, Dublin's chief medical officer warned against the common practice of waking the dead as 'an objectionable practice… nothing short of criminal'.[25]

[21] Bengtsson *et al.* 2018.
[22] Phipson 1923: 517-9.
[23] Kirsty Johnston, 'Coronavirus: Lessons learned from the great influenza pandemic of 1918', *New Zealand Herald*, 18 Mar 2020.
[24] Tommy Bengtsson, Björn Eriksson, and Martin Dribe, 'Exposure at Workplace and Excess Mortality in Sweden during the 1918 Influenza Pandemic', paper presented at the 4th Conference of the European Society of Historical Demography, Madrid, 3 March 2022.
[25] *Irish Times*, 'Influenza epidemic: warning against wakes', 9 November 1918); also *ibid*. 'Wakes and infection', 4 November 1918.

Studies of the impact of the flu using individual data are uncommon. Grantz *et al.*[26] use individual-level data on influenza deaths [n=7,971] in Chicago in order to investigate the role of neighbourhood-level socio-economic factors at the household level. They find that illiteracy, a good proxy for income, was strongly linked to deaths from influenza and pneumonia: a ten per cent increase in illiteracy increased the death rate by nearly one-third. Unemployment and population density were also linked to higher death rates. Air quality may have mattered too. In the U.S. air pollution levels help explain the sharp variations across urban areas in mortality from influenza. Reducing air pollution in above median-pollution cities to the median would have reduced deaths by 16,000-24,000. Proximity to army bases, as well as the baseline disease environment, municipal spending on public health, and measures of poverty and illiteracy, also increased deaths.[27]

The Death Toll:

There have been several attempts at estimating the pandemic's global cost in lives. This is not an easy task, given the uncertainties about reported causes of death and registration systems sometimes immobilised by war. Thus in Belgium, while contemporary estimates proposed a figure of 20,000, current estimates of best- and worst-case scenarios are 30,000 and 100,000 deaths, respectively.[28] An early attempt by the US public health scientist Edwin Jordan (1927) put the toll there at 21.6 million. David Patterson and Gerald Kyle (1991) proposed a range of between 24.7 million and 39.3 million. Niall Johnson and Jürgen Mueller (2002) increased this to a 'best guess' of about 50 million or 3-4 per cent of the world's population, while acknowledging that the true figure may have been far higher. That could have been either because registration was incomplete or because the cause of death was misattributed. It could also be the case that the virus, by weakening the immune system, increased deaths from other causes; such deaths might arguably be added to those attributed to

[26] Grantz *et al.* 2016.
[27] Clay *et al*. 2019; Fuhrman 2010.
[28] Devos *et al*. 2019: 254.

influenza. Thus, the 23,000 proposed by Milne for Ireland allows for associated deaths from pneumonia.[29]

Johnson and Mueller's proposed range of 50 million to 100 million deaths worldwide has achieved wide currency since.[30] Their estimates for individual European countries, where civil registration data were probably not too far from the truth and often factor in deaths from other respiratory diseases, are broadly consistent with those of Séverine Ansart and her co-authors (2009), who use the deviation from the norm in deaths from all causes in 1918-19 as a measure of influenza deaths. Only in cases when Ansart *et al.*'s estimates are inflated by excess deaths from hunger, disease, or civil war as in the cases of Germany, Italy, and Finland, are they in conflict.

Table 1 incorporates some more recent estimates for individual countries as well as those from Patterson and Kyle and from Johnson and Mueller for others. The first estimate of pandemic mortality in India, based on faulty civil registration data, placed it at 7 million. That estimate assumed that reported deaths from influenza accounted for all pandemic deaths, which was clearly not the case: in Bombay, for example, the rise in deaths from 'respiratory diseases' closely mirrored that for influenza.[31] Subsequent scholarship more than doubled that figure, and Johnson and Mueller increased it further to 18.5 million, or more than one-third of their 'best guess' total for global deaths. In *Age of Pandemics* Chinmay Tumbe reaches even higher, to 20 million, but other recent estimates suggest an in-between figure of 12 million or about 40 per 1,000 population.[32]

Johnson and Mueller rely on Colin Brown's estimate of at least 1.5 million pandemic deaths in Indonesia, but the underlying data are by no means comprehensive, and more recent estimate by Siddarth Chandra based on population counts revises the figure upward to a staggering minimum of 4 million, which would

[29] Milne 2018: Ch. 3.
[30] Recently Spreeuwenberg *et al.* (2018) have proposed a much lower figure of 17.4 million, but this has been contested and is based on far flimsier data than Johnson and Mueller's.
[31] Phipson 1923: Chart 4.
[32] Phipson 1923; Murray *et al*. 2006; Tumbe 2020; Chandra *et al*. 2012; Dyson 2018: 149.

imply a death rate of over 8 per cent of the population, or double the rate found anywhere else.[33] Here I use the more conservative total of 2 million.

Data sources for most of China are sparse. They are more plentiful for its port cities, which imply death rates from influenza of the order of 0.2 to 0.3 per cent in 1918; applying that range to the country as a whole produces an aggregate toll of 1 million to 1.5 million. That is implausibly low; the latest discussions by Peckham and by Federico and Tena-Junguito opt for Johnson and Mueller's range for China -- 4 to 9.5 million, which is reflected in Table 1.[34] In Japan the Central Sanitary Bureau attributed 257,363 deaths to influenza (or 4.25 per thousand) between September 1918 and July 1919.[35] Some of the deaths recorded as due to bronchitis and pneumonia should probably be added to that total. If the flu's duration is extended into 1920, then the officially registered number of flu deaths rises to 385,000, a figure revised upwards to 453,000 by Hayami.[36] But it could be argued that the epidemic of September 1919-February 1920 should not be included, since the virus would have been subject to 'antigenic drift' by then and, thus, constituted a new epidemic. Hayami's revision implies 8 deaths per 1,000 inhabitants. Nishimura and Ohkusa attribute what they see as Japan's low death rate to its high literacy rate, social cohesiveness, and the effective implementation of measures such as the mandatory wearing of facemasks and the closure of public spaces and schools. Did Japan's non-participation in WW1 play a part? Did its non-participation help China to escape relatively lightly relative to say, India? Cheng and Leung propose a role for traditional Chinese medicine, but candidly dub this a hypothesis to be investigated.[37] Being removed from the war is a more likely reason.

Table 1 includes new estimates of flu mortality for Italy, Spain, and several other European countries. It sticks with Patterson and Pyle's estimate of deaths in Russia, which Johnson and Mueller also use,[38], although at 450,000 or 2.5 per

[33] Brown 1987: 235; Chandra 2013.
[34] Peckham 2022; Federico and Tena-Junguito 2023; Johnson and Mueller 2002: 112. Compare Iijima 2003.
[35] Rice and Palmer 1997: 397.
[36] Both estimates from Nishimura and Ohkusa 2016.
[37] Cheng and Leung 2007.
[38] Patterson and Pyle 1991.

thousand, it seems far too low, even if the earlier 'Russian' flu of 1889-1890 conferred some immunity on older Russians. Estimates for Greece are lacking but the toll seems to have been modest by European standards and, indeed, the authorities were more worried at the time about the risks posed by malaria than the pandemic.[39]

Turning to Latin America, the impact of the flu on mortality in Argentina and Uruguay was also relatively light. A study of the pandemic in the latter concludes that "The social alarm caused by influenza epidemic was probably more related to its morbidity than to the impact of its lethality"[40]. In Chile, however, although most of the population also lived in Latin America's temperate zone, mortality was very high. The first wave hit Chile in July 1918, implying rapid transcontinental diffusion of the virus via boats and railways"[41]. The second wave came later than in the northern hemisphere, between July 1919 and February 1920, and there was a further wave in 1920-21[42]. Estimating influenza deaths as excess deaths relative to a baseline, Chowell *et al.*[43] found 34,978 excess deaths extending over four waves, the final wave in 1921 being particularly severe in Santiago. The total implies a cumulative national death rate of 9.4 per 1,000 per thousand inhabitants. The rate ranged from 2.5 deaths per 1,000 in Tarapacá in the extreme north to 20.6 per 1,000 in the capital region on Santiago in central Chile, with the gradient of excess mortality rising from north to south. Geography was by no means all. In Santiago the crisis prompted the passage of Chile's first sanitary code—like locking the gates after the enemy had entered—and a campaign to improve hygiene and sanitation, but when the authorities sought to isolate suspected cases and to disinfect their homes, many people reacted by not reporting cases for fear of being hospitalized.[44]

Hard evidence on the death toll in Brazil is lacking. In São Paulo, 5,331 died during the second wave of up to 350,000 who were infected, and about 15,000 died in Rio de Janeiro out of the 600,000 infected there, two-thirds of the population. A 1993

[39] Bournova and Dimitropoulou 2015.
[40] Cristina *et al*. 2019: 224.
[41] Chowell *et al*. 2014.
[42] Compare Cristina *et al*. 2019.
[43] Chowell *et al.* 2014.
[44] López and Beltrán 2013.

Brazilian study focus on the region straddling Rio and São Paulo, partly due the paucity of source material, but also "because it was in these more populous urban centres that its consequences were more tragic". Johnson and Mueller cite a figure of 180,000 but we opt for a lower figure of about 60,000 here.[45] The data on Mexico are also sparse and of dubious quality. Lourdes Márquez Morfín and América Molina del Villar compare press reports of 150-200 deaths per day in Mexico City to the 7,375 deaths recorded in the official archives to argue that the impact of the second wave was modest relative to that of previous epidemics. The latter figure represented less than one per cent of the population of the city and 1.2 per cent of the population of the city proper, whereas Chowell *et al.* propose a figure of 0.7 per cent. However, Chowell *et al*. report a much higher death rate of 1.9 per cent for Toluca, a city of 30,000 people in central Mexico, which they surmise may reflect "poorer socioeconomic conditions, issues with access to health care, or environmental conditions". Opting for a compromise rate of 13 per thousand would imply an aggregate total of 0.2 million.[46] A contemporary report in March 1919 claimed that there was a general decline in mortality by the end of January, with some estimates of the death toll running as high as 0.5 million by then. However, a statement published in early January 1919 for 25 states and the capital city reported the highest mortality in the state of Michoacan (48,000 out of a population 991,600), and the lowest in Colima (900 out of a population of 77,000).[47]

The case of Iran highlights how research on the flu in some cases is so problematic when statistics are lacking and other archival data limited. In twelve Iranian cities for which there are estimates of both deaths and either population or the numbers infected, the aggregate death rate was 30.8 per thousand, if the latter are assumed to be half of the population. But those cities represented only about 6 per

[45] Schuck-Paim *et al*. 2012; Guimbeau *et al*. 2020; http://www.invivo.fiocruz.br/cgi/cgilua.exe/sys/start.htm?UserActiveTemplate=espanol&infoid=1111&sid=45; José Cássio de Moraes, as cited by Anamario Nascimento in *Diario da Pernambuco*, 4 April 2020; Luiz Antonio Teixeira, *Medo e morte: Sobre a epidemia de gripe espanhola de 1918*. Rio de Janeiro: Universidade do Estado do Rio de Janeiro/Instituto de Medicina Social, 1993.
[46] Márquez Morfín and Molina del Villar 2010; Chowell *et al.* 2010; Alexander 2019: 446 (who proposes "somewhere between 300,000 and 600,000 people").
[47] *Public Health Reports*, 7 March 1919.

cent of Iran's population. Data for the rest of the country are lacking; guessing 40 per thousand for Iran as a whole – close to the highest rate in Table 1 -- would imply about 0.5 million deaths.[48]

Adding up our country and regional estimates yields a global total of somewhat under thirty million deaths, which is lower than the most commonly cited estimates nowadays.[49] But while "everything seemed to contribute to the underestimate of flu mortality" before the new global estimates of Paterson and Pyle (1991) and Johnson and Mueller (2002), research since suggests that the wisest counsel is to "only accept global estimates with a substantial margin of error".[50]

Table 1. Estimated Deaths from Influenza 1918-1920

Country	Population (1,000s)	Deaths	DR (per 1,000)	Ansart *et al.* [2009]
Austria	6,727	20,458	3.3	
Belgium	7,660	30,000	*3.9*	
Bulgaria	5,072	50,000	*10.8*	51,156
Croatia	3,443 [1921, Wiki]	15,000-20,000	5.1	
Czechoslovakia	12,979 [1920]	66,000-107,000	5.1/8.2	
Denmark	3,165	10,185	3.3	10,650
E & W [1921]	37,887	200,000	5.8	153,152
Finland	3,125	18,000	5.8	[24,771]
France	38,542	240,000	7.3	237,509
Germany	61,757	335,000	5.4	[426,574]
Hungary	7,950	53,000	6.9	
Iceland	0,092	484	*5.3*	
Ireland	4,300	23,000	5.3	
Italy	37,520	410,000-466,000	10.9/12.4	[544,288]
Malta [1921]	212	588	2.8	
Netherlands	6,752	48,042	7.1	
Norway	2,578	14,676	5.7	14,465
Poland	23,968 [1920]	200,000-300,000	10.4	
Portugal	6,013	135,257	9.8	135,600
USSR/Russia	184,000	2.8 million	15	
Scotland [1921]	4,888	27,650-33,771	*4.1*	20,879

[48] Afkhami 2003: 384.
[49] See, however, Federico and Tena-Junguito (2023: 26), whose 'best guess' is 35 million.
[50] Rasmussen 2014: 354.

Spain	20,950	257,082	12.3	252,121
Sweden	5,807	34,374	5.9	38,453
Switzerland	3,880	24,449	6.1	29,995
Australia	5,304	14,528	2.7	
New Zealand	1,158	9,000	7.8	
USA	103,208	675,000	6.5	
Canada	8,148	50,000	6.1	
Argentina	8,517	14,997	1.8	
Uruguay	1,316	2,015	1.5	
Chile	3,634	34,978	9.6	
Brazil	30 million	60,000	2	
Mexico	14,920	200,000	13	
Ceylon	5,109	50,000	11	
India	305 million	12 million	40	
China	462 million	4-10 million	8 to 20	
Japan	54,886	453,000	8.2	
Korea	16.7 million	140,000	8.4	
Afghanistan		320,000		
Indonesia	49 million	2 million	41	
Philippines	10 million	80,000	8	
Iran	12 million	0.5 million	40	
Other Asia	200 million	4 million	20	
South Africa	*6.5 million*	*300,000*	46	
Other Africa		2 million	20	
TOTAL		30-35 million		

Sources: Patterson and Pyle 1991; Johnson and Mueller 2002; Ansart *et al*. 2009; Bandeira 2009 (Portugal); Fornasin *et al*. 2018 (Italy); Ingholt *et al*. 2020: 54 (Denmark); Carbonetti 2010 (Argentina); Trilla *et al*. 2018 (Spain);); Milne 2018 (Ireland, includes deaths from pneumonia); Chowell *et al*. 2014 (Chile); Cristina *et al.* 2019 (Uruguay); Summers *et al*. 2018 (New Zealand); Grabowski *et al*. 2017 (Poland); Szegö 2018 (Hungary); Michels 2010 (Germany)[51]; Wiki citing Hutinec 2006 (Croatia); Langford and Storey 1992 (Ceylon); Nascimento 2020 (Brazil); Iijima 2003, Peckham 2022, Federico and Tena-Junguito 2023 (China); Phillips (South Africa); Johnson and Mueller (Other Africa); Van der Eng 2020 (Indonesia); Athukorala and Athukorala 2020 (Russia/USSR); Gealogo 2009 (Philippines). Population estimates for 1918-1920 from the Maddison Database or Wikipedia, except for Germany (Michels 2010).

[51] On Germany see too Udo Buchholz *et al*. 'Todesfälle durch Influenzapandemien in Deutschland 1918 bis 2009 Schätzwerte auf Basis der Literatur und ergänzende eigene Berechnungen', *Bundesgesundheitsblatt - Gesundheitsforschung – Gesundheitsschutz*, 59[4] (2016), 523–536. Michels (2010) includes an allowance for subsequent deaths from *encephalitis lethargica*, although the link between the flu and this condition is disputed.

Some of the small number of communities that seemed to escape the pandemic have attracted scholarly attention. They included a few remote islands in Alaska's Bering Strait, and villages in north Alaska where local armed vigilantes deterred visitors. However, the coastal town of Nome, also remote, was less lucky. It took a month for the virus to make its way there on a steamship with some infected passengers from south-east Alaska. Despite some precautions being taken, the virus spread with catastrophic consequences, particularly for the local Inuit population. A local schoolteacher wrote:[52]

> As one walked the streets of Nome, it seemed a city of the dead. A panic had struck the Natives, and their feverish conditions suggested the need of colder air … They would leave their beds of sickness and go into the cold air, which, inducing pneumonia, carried them away rapidly… From ten to twenty Natives were dying each day on average in Nome, and the dead wagon was in use constantly… Many were frozen to death during the night, their fires having gone out.

There were nearly one thousand influenza deaths in Alaska in 1918, and a further 151 in 1919. That represented twenty deaths per 1,000 inhabitants, or three times the US average. Nearly two-thirds of the total occurred in the Nome census area, with the indigenous Inuit population most likely to succumb.[53]

In the US the Defence Department's Threat Reduction Agency commissioned a study of seven communities: Fletcher, a rural farming village in Vermont; Gunnison, Colorado; two academic institutions, Bryn Mawr College in Pennsylvania and the military trainee section of Princeton University in New Jersey; and a home for the blind in in Pittsburgh and a tuberculosis sanatorium at Saranac Lake, New York. Elsewhere American Samoa, Tasmania, and New Caledonia escaped lightly and only the Fijian volcanic islands of Lau and Yasawa, Marajó near the mouth of the Amazon

[52] As cited in Tony Hopfinger, 'How the Alaska Eskimo village Wales was never the same after 1918 flu', *Anchorage Daily News*, 27 May 2012 [https://www.adn.com/rural-alaska/article/part-3-how-alaska-eskimo-village-wales-was-never-same-after-1918-flu/2012/05/27/]. See Mamelund *et al.* 2013 ; Alaska Department of Health and Social Services. 2018.
[53] See too deValpine 2015.

in Brazil's northeast, St. Helena in the south Atlantic, and a few small communities in Alaska seem to have escaped entirely.[54]

The War and the Flu:

Just two days after the end of WW1 Great Britain's most senior public health official, Sir Arthur Newsholme, declared that troop movements had been "responsible on a large scale" for the increased virulence of the flu and that wartime priorities had ruled out taking measures that would have curbed its spread.[55] However, most older histories of the war either ignore or dismiss any potential link between it and the pandemic. Echoing that literature, WW1 historian Jay Winter insisted in 1988 that the pandemic "was neither caused by the first World War, nor can its victims be counted among the victims of the conflict", and again in 1997 that "the flu and its huge death toll were a product not of the war, but of the (then as now) unknown processes of viral morphology". And Guy Beiner, an expert on popular memory with an interest in the 1918-19 pandemic, has pointed out that "the annihilation of Eskimos or the death of millions in Asia cannot simply be attributed to the war without charting a comprehensive global map of war-related transport, which may well fall short of proving a direct correlation".[56]

That is now a minority view among historians and epidemiologists. Few scholars, perhaps, would go so far as to assert "no war, no pandemic", or claim that "you cannot understand the history of the Spanish flu without understanding World War I. And you can't really understand what went on in the last year of World War I without understanding the flu".[57] But most would take it for granted that that troop clusters during WW1 acted as breeding grounds for the virus and eased its spread, and

[54] Markel *et al*. 2006; Richard Gray, 'The places that escaped the Spanish flu', *BBC Future*, 24 October 2018 [www.bbc.com/future/article/20181023-the-places-that-escaped-the-spanish-flu]; McLeod *et al*. 2008.
[55] Newsholme 1918-19.
[56] Winter 1988: 23; 1997: 489; Beiner 2006: 502.
[57] Kenneth C. Davis, author of the accessible *More Deadly Than War: The Hidden History of the Spanish Flu and the First World War* (NY: Holt, 2018) on https://www.wbur.org/commonhealth/2020/05/11/historian-draws-parallels-between-the-1918-spanish-flu-and-todays-coronavirus-pandemic.

in that way increased its lethality. Geoffrey Rice, foremost authority on the pandemic in New Zealand, put it thus: "The world was at war, so there were lots of troops moving around the world - including coming back to New Zealand where they then scattered back to their homes… New Zealand had early warnings of the infection from events overseas but they were largely ignored, and the government of the day was slow to react."[58] Others have referred to the pandemic and the war as being 'fused symbiotically' and 'inextricably linked', making the point that, on the one hand, the war helped the spread of the virus, while on the other, the virus influenced the conduct if not the outcome of the war. One of the pandemic's best-known historians goes as far as to argue that "In effect, the H1N1 virus was globalized by World War I which systematically turned a local outbreak in one continent into a world pandemic".[59] And Anne Rasmussen's chapter on the pandemic in *The Cambridge History of the First World War*, edited by Jay Winter, while stressing "the preponderant role played by virological and genetic aspects" of the flu and its easy transmissibility among humans, also highlights how "war provided extremely favourable conditions for the spread of infectious disease", with the "staggering traffic worldwide associated with it "provid[ing] every circumstance favourable to the emergence of a killer epidemic"[60]. From the realm of influential non-specialists, Steve Pinker reasons in his much-debated history of violence that including the victims of the flu pandemic as war deaths could be justified on the grounds that "the flu virus would not have evolved its violence if the war had not packed so many troops into trenches". That way "one could multiply the number of civilian casualties many times over". Anton Erkoreka concludes his search for the origins of the virus in the Archives of the Health Services of the French Army with the remark that "After all, the Spanish influenza could be considered to be a "historic accident", another cruel consequence

[58] Kirsty Johnston, 'Coronavirus: Lessons learned from the great influenza pandemic of 1918', *New Zealand Herald*, 18 Mar 2020.
[59] Phillips 2014. Phillips (2018b) points out that two widely-consulted websites commemorating the centenary of WW1 feature the pandemic prominently, and one of them even includes a computer model of its diffusion. For the websites and the flu see: http://ww1centenary.oucs.ox.ac.uk/body-and-mind/the-spanish-influenza-pandemic-and-its-relation-to-the-first-world-war/; https://encyclopedia.1914-1918-online.net/article/influenza_pandemic.
[60] Rasmussen 2014: 341.

of the terrible First World War".[61] True, there was influenza in places where no soldiers ventured. But sometimes too, the war reached even the remotest places, such as where Eskimos (or Inuit) lived. It is a long way from Boston to Alaska, but it was a naval ship that made the journey via the Panama Canal, reaching the Puget Sound Naval Station on 17 September 1918, that brought the flu to Seattle and the Pacific northwest and, indirectly, from there to Juneau in southeast Alaska by mid-October.

Over the past decade or so historians of the pandemic have been providing some of the raw material for that 'global map of war-related transport' that Beiner demanded (see above), using both narrative and analytical approaches. Several have noted that the first reported cases most often in the armed forces, and that wartime movements of military personnel exacerbated the situation.

North America: The first identified outbreak at Camp Funston, a US Army training camp in Kansas, in early March 1918 hospitalised a thousand troops within a fortnight, of whom 38 died. On 30 March the chief medical officer at Camp Funston wrote to the army surgeon general's office in Washington: "Virulent secondary streptococcic pneumonia following epidemic pseudo influenza present. Many deaths influenza following immediately two extremely severe dust storms" (cited in Byerly 225: 70). There were reports of infection from American Expeditionary Force hospital in Bordeaux (April 15) and in St. Nazaire (May), and in June AEF troops near the Somme were being affected, though where these infections came from remains unclear.

In California, the first wave was linked to the visit of two Japanese training ships carrying one thousand sailors. They docked in San Francisco on 22 March, where they toured and socialised widely, and in Los Angeles on 1 April, where they were also warmly welcomed. These visits were followed by flu outbreaks, and were blamed on "the visit of a Japanese Squadron". Another outbreak, on Mare Island Naval Shipyard near Vallejo, had military links, as did that at Camp Fremont south of San Francisco. The outbreak in San Quentin prison in mid-April did not have military origins, it seems. In general, the first wave of influenza in 1918 did not set off panic

[61] Pinker 2011 (ebook); Erkoreka 2019: 194.

buttons, because its symptoms were relatively mild and it was not considered very lethal. In San Quentin, three quarters of all 1,900 inmates were infected, but only three died.[62]

The most likely source of the second, much deadlier, variant is army training camps in the United States and the movement of the American Expeditionary Force to Europe in the autumn of 1918. The Boston area and, more specifically, the military encampment at Fort Devens west of Lowell, were the first places in the U.S. to be hit by the second wave in September 1918. The virus made its way south and west from Massachusetts "following wartime transportation routes with its human hosts". The first recorded death in New Jersey in the same month happened at Fort Dix in the south of the state on 18 September. Soon there were reports from other army camps Kansas (20 September), California (27 September), and Iowa. Two remote camps in Georgia were not hit until 11 October. Within a month the virus was nationwide: the *New York Times* reported on 16 October that influenza had reached epidemic proportions in "practically every State in the country"[63]. The overcrowded conditions in which troops lived and travelled greatly eased the transmission of the virus. The pattern of diffusion in Europe (from Brest) and the Americas (from Boston) was not dissimilar, with infected soldiers and sailors once again being the principal carriers.[64]

Burial ceremony for AEF troops at Kerfautras graveyard
(Archives de Brest Métropole)

[62] North 2020: 9-10.
[63] Byerly 2005: 75, 79.
[64] Cormier 2020.

Observation room in the American hospital at Pontanézen
(US National Archives and Records Administration)

Mass troop movements ensured that the virus spread rapidly from these camps to many other military bases in North America, to the civilian population and then across the Atlantic to Britain and France, primarily aboard troopships. From this heartland of the world war and world trade, it was then spread to several parts of the northern hemisphere by similar means between May and July 1918. The SS Kroonland arrived in Brest on 12 September 1918, full of US troops for the war in Europe. Two of her crew died of the flu *en route* and she brought 117 cases of influenza and 6 of pneumonia.[65] The huge army camp in nearby Pontanézen facilitated the spread of the virus. A peak in deaths in the Brest region followed on 17-20 September.

The SS Leviathan – at the time one of the biggest and fastest ships in the world-- left New York on 29 September with 9,000 soldiers and 2,000 crewmen. It arguably should not have travelled. Those on board included men who had marched from camp in New Jersey on the night of the 27th. Several men had to drop out, and more collapsed before they embarked. Conditions on board were poor, and within 36 hours seven hundred had caught the virus and one had died. Conditions deteriorated and some troops refused to obey orders. They arrived in Brest on 7 October in awful weather; thirty-one died on that day and 969 were admitted to hospitals. About two

[65] Ford 1927: 462.

hundred Americans were buried in the US cemetery at Lambezellec. In all about 6 per cent of troops died while crossing the Atlantic; counting those who died in military camps in the five days after disembarkation, the navy would have lost four thousand men in the last four months of 1918, twice as many as in fighting the Germans.[66]

The flu's most likely point of entry to Canada was the Maritimes port of Halifax, where the hospital transport ship *Araguaya* arrived on 7 July 1918 with 175 cases on board. Some invalid patients were taken off on the following day; and the quarantine on the *Araguaya* was lifted too soon, on 10 July. Almost simultaneously, two empty military transports arrived in Montreal to ferry Canadian and American troops, respectively, to Europe, but on inspection several of their crews were found to have somehow contracted the virus. Further investigation found the flu to be present on other ships in early July 1918, and not only military ships. The traffic of troops to and from the US on their way to Europe, including large numbers from the US who volunteered to fight for the Polish army in France, but received their military training in Niagara-on-the-Lake across the border in Canada, helped spread the virus. The combination of long-established transport networks and military cooperation between the USA and Canada "disproportionately funnelled [the first wave of] influenza" into the Maritimes, where mortality was significantly higher than in the rest of Canada. The traffic in the virus was thus two-way: it arrived in Canada from overseas ports, but also spread eastwards on troopships.[67]

Over the summer the flu seems to have mutated into the H1N1 subtype that combined infectivity and lethality. The contrast in mortality between the Camp Funson and Camp Devens was stark. By the end of September 14,000 camp inmates in the latter, which was seriously overcrowded, had been infected—over one in four of the total—and 757 of those had succumbed. A physician at Fort Devens hospital noted: "These men start with what appears to be an ordinary attack of La Grippe or Influenza, and when brought to the Hosp. they very rapidly develop the most vicious type of Pneumonia that has ever been seen. Two hours after admission they have the Mahogany spots over the cheek bones, and a few hours later you can begin to see the

[66] Breche 2012: 63.
[67] Bogaert 2015, 2017; Rewegan *et al*. 2015.

Cyanosis"—the term refers to a person turning blue from lack of oxygen—"extending from their ears and spreading all over the face….It is only a matter of a few hours then until death comes…It is horrible….We have been averaging about 100 deaths per day…For several days there were no coffins and the bodies piled up something fierce…"[68]

Morgue in U. S. Army Hospital, Gievre, France
Source: http://resource.nlm.nih.gov/101399168 (public domain)

Great Britain: The 1918-1919 annual report of the medical department of the Local Government Board for England and Wales was quite explicit about the early incidence and spread of the disease. It was first noticed in mid-April on Royal Navy ships stationed at Scapa and Forsyth, peaking there on 10 May with 744 men reported sick with influenza. In all, one in nine of the 90,000 officers and men contracted the virus. The earliest cases detected in the British army were in France in April and May 1918. The epidemic struck the Second Army 'with great violence' at the end of April; soon the First and Third Armies were hit, followed by the Fourth Army in mid-May. The first likely cases noted in the civil population were in a few factories and an industrial home in Glasgow in May, but it was not until June that the first wave struck several parts of England more or less simultaneously, with "in many places the first

[68] Extracts from a letter written by N.R. Grist, a physician stationed at Camp Devens, to a fellow physician, 29 September 1918 [retrived from: https://www.digitalhistory.uh.edu/disp_textbook.cfm?smtID=3&psid=1112; last accessed 14 March 2022].

cases occur[ing] in association with soldiers or sailors convalescent or at home on leave". The second wave struck the naval forces at Portsmouth and on board American transports first, in August and September. The report linked this to the civilian populations in the chief ports to have been the first to catch the second wave.[69]

Ireland: The flu killed about 23,000 people in Ireland.[70] The first cases on record occurred on the USS Dixie as it docked outside Queenstown (Cobh) in May 1918. Within a month there were reports from all over the country. The first wave in May 1918 passed quickly, however, and mortality was light. The second wave, lasting from mid-October to December, was the most virulent of the three; and, as in the first wave, the provinces of Leinster and Ulster were worst affected. The third wave, also severe, lasted from mid-February to mid-April 1919, some counties in the west being worst hit by it. Dubliners were badly hit by all three waves; it has been suggested that returning troops help explain this. In Dublin, there was a close link between housing conditions and mortality, with areas such as Summerhill and Capel Street in the north inner city being particularly affected. There was a strong east-west gradient to the pandemic, with a few counties almost escaping it, notably Clare in the west (with fewer than 0.5 deaths per 1,000 population; see Figure 2). Research on the spread of the pandemic at poor law union level in Ireland suggests that the death rate from flu mirrored the main railway lines; the presence of military barracks may also have been a factor.[71] Reluctant to see a link between influenza and the war, the *Irish Times* was open to a connexion to this extent: "the world's mind has been in a state of abnormal strain for four years, and certain states of the mind may encourage certain forms of disease."[72]

[69] BPP 1919: 7-8.
[70] Milne 2018: Ch. 3.
[71] Thanks to Francis Ludlow, Georgina Laragy, and Ida Milne for allowing me to reproduce their map.
[72] 'Influenza and War', 29 October 1918.

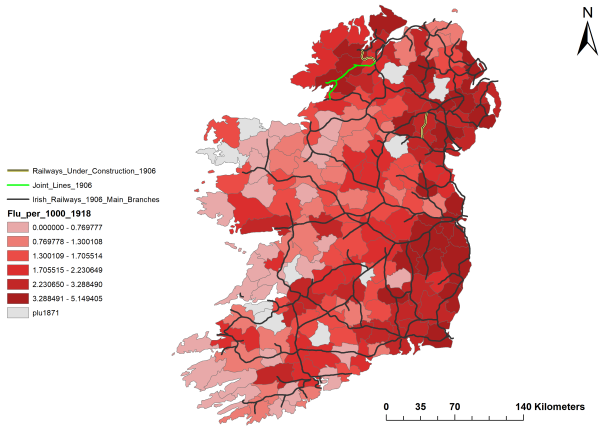

Figure 2. Variation in Pandemic Deaths, Ireland 1918-19

India: The pandemic first struck Bombay in late May or early June 1918. It seemed natural to link this to the war. About 1.3 million Indian soldiers had served in WW1, about one-tenth of them on the western front, but most in the Middle East and in Mesopotamia. Bombay's health officer pointed to the role of a ship transporting troops home from Mesopotamia. However, the Indian colonial government linked the flu to the city's poor sanitation, asserting that the disease was endemic and that its spread could not be prevented. In the event the disease, which would prove more lethal than anywhere else on the subcontinent, was spread by trains packed with refugees fleeing the infection, many of whom were infected themselves.[73] The post office, which had its own rail service, also spread the virus; indeed, contemporaries often linked the spread of the virus to the postal service.[74] In the words of the Indian Sanitary Commissioner's Annual Report for 1918, "The railway played a prominent part, as was inevitable". The report contains the following interesting anecdote from the Punjab:[75]

> A recruit suffering from symptoms of influenza arrived at Narkanda [a town in the Himalayan foothills, near Simla] and stayed a night with the

[73] Ramanna 2003; Chandra and Kassens Noor 2014.
[74] Ojo 2020.
[75] As cited in Shenoy Karun, 'Railways spread the Spanish flu in India in 1918', *Times of India*, 22 March 2020 [https://timesofindia.indiatimes.com/city/kochi/railways-spread-the-spanish-flu-in-india-in-1918/articleshow/74759235.cms?frmapp=yes&from=mdr; last accessed 14 March 2022].

> *khansama* [i.e. butler] of the hotel. Three days later the *khansama* and his wife went down with influenza. Within a few yards of the *khansama*'s house lived a blacksmith and the two used to inter-visit. A few days after the *khansama* was attacked all the members of the blacksmith's house were down with the disease. The postal peons used to frequent the *khansama*'s house, with the result that they all contracted the disease and two of them died of it… In a short time the Hindustan Tibet Road was thoroughly infected… Marwari traders, shopkeepers, and even doctors, in fact, all who travelled by water, road or rail, were potential factors in distributing infection.

Mortality from the pandemic varied considerably from region to region. An official report on the epidemic found that the death rate in was the Central Provinces and in Delhi was twelve times as high as in Bengal.[76] A fiscal squeeze on the economy, exacerbated by a serious harvest failure in 1918/19, led to unemployment and hunger in 1917 and 1918 throughout most of India. The combination probably increased the threat from influenza.

Africa: The second wave of the epidemic struck sub-Saharan Africa, which had been spared the first, hard. The ports of Freetown (Sierra Leone), Mombasa (Tanzania), and Cape Town (South Africa) were key entry points. In Freetown a Royal Navy warship that docked on 14 August 1918 has been identified as the vehicle; although many of its crew were infected, their conditions were mild, and it was not placed under quarantine. The local population was quickly infected at this time with the second wave virus, with a morbidity rate of 70 per cent within a fortnight and the loss of 4 per cent of total population. Cape Town was infected via Freetown, as ships carrying South African troops home had docked there. A lax quarantine was insufficient to prevent the virus from spreading. South Africa's relatively advanced communications system hastened the spread of the disease.[77] "A single ship from India" has been blamed for bringing the virus to Mombasa in late 1918; whether there was a military connection in this case remains unclear. But the soldiers, porters, and labourers who caught the virus helped spread it inland. On whole, the colonial

[76] *Preliminary Report of the Influenza of 1918 in India*, 1919: p. 4, as cited in Ojo 2020: 1027.
[77] De Kadt et al. 2021; Jayes and Fourie 2022.

authorities were sceptical of their ability to prevent the disease from spreading, and focused on coping with its effects as it spread, with limited funds and very limited success.[78]

The second wave of the virus arrived in South Africa in early September via two troopships carrying home two thousand South African Native Labour Corps who had served in Europe. Some of the workers had contracted the virus in Freetown, where the ships had stopped over to re-fuel. The arrivals were quarantined, but for an insufficient period (two days), with the result that the virus spread with them as they made their ways home. Phillips described how the troop-carrying railways spread the pandemic to 'the predominantly black rural areas of the country. "Five trains left Cape Town between 16 and 30 September, carrying soldiers to all parts of the Cape Province. As they disembarked at every railway platform, the virus travelled with them". And, indeed, de Kadt *et al.* find that "proximity to railways does indeed appear to explain mortality – the further a magisterial district is from the railway, the lower the mortality rate, though these coefficients are only statistically significant at the 10% level". Freetown, in turn, was infected by men on board the HMS Mantua, which arrived from Devonport on 15 August. The claim that the virus arrived in New Zealand via RMS Niagara, which docked in Auckland on 12 October, has been discredited; much more likely is that it arrived on troopships whence it spread to military camps and further afield.[79]

Brazil: The military connection is also plain in the case of Brazil. Its first contact with the virus was during the second wave, when eight of its naval vessels were sent to patrol the west coast of Africa. The convoy docked in Dakar on 29 August after spending three weeks in Freetown, Sierra Leone, and within days crews were suffering from the flu. The virus was probably spread by British naval personnel, also docked in Dakar. Over 8 per cent of the Brazilian military (127 of a total of 1,527) died from the flu. Almost simultaneously, a ship from returning from Europe via Dakar seems to have brought the disease back home as crew disembarked in Recife in

[78] Phillips 2014b; Tomkins 1994; Andayi 2019.
[79] Phillips 1984: 152; de Kadt *et al*. 2020: 7.

Brazil's northeast in September 1918, "which everything indicates came from Dakar, brought by Brazilian sailors on military service in the region"[80].

Russia: The pandemic seems to have arrived from the west through contact with Austro-German forces and from the northeast via Allied troops supporting local anti-Bolshevik forces, and 'was almost certainly transported along the rail system by troops and refugees'.[81] Nationwide data are lacking. A recent study of the pandemic in Russia finds little evidence to support the case for high mortality, apart from a few localities "associated with the military-political situation", and suggests that classic diseases such as typhus, cholera, relapsing fever, and smallpox were much more lethal at the time.[82]

Iran: The virus fist arrived in northeast Iran via Bolshevik and British armies competing for control of the country. From there it is said to have followed British supply lines to the city of Mashhad, a Shia holy site, where it killed about 5 per cent of the population, whence it was spread further by a combination of soldiers and pilgrims. The second wave made its way from Bombay and Basra to the port cities of Bushihr and Muhammara.[83]

Australia and New Zealand: Australia had the advantage of location. It monitored all incoming ships and imposed a maritime quarantine on 17 October 1918. Given its remoteness and the five-day incubation period of H1N1, detection on incoming vessels was straightforward. Over the following six months 323 ships were inspected, more than half of which were carrying the virus. The quarantine delayed the spread of the virus until a suspected case was detected in the state of Victoria until 9/10 January 1919. The authorities were slow to confirm the case, which allowed the virus to spread unhindered for a few weeks. When a soldier from Melbourne was diagnosed with the virus on arrival in Sydney nearly three weeks later, the state of New South Wales closed its border with Victoria. Still, the impact of the pandemic in

[80] Anamaria Nascimento, 'As lições aprendidas—e os erros que voltamos a cometer—da gripe espanhola de 1918', *Diario da Pernambuco*, 4 April 2020.
[81] Patterson and Pyle 1991: 10.
[82] Morozova *et al*. 2021. Interestingly, influenza is not among the infectious killer-diseases listed in Wheatcroft (1983).
[83] Afkhami 2003: 373-5, 377, 381.

Australia would turn out to be relatively light. No quarantine was imposed in New Zealand. The precise origins of the virus there are unknown but the second wave was first detected among 'native troops' at Narrow Neck Camp in Auckland in October 1918. Soldiers accounted for about one in nine of the 8,500 deaths in New Zealand, indicating a huge mortality rate (8.8 per cent) from the flu in the army.[84] The epidemic was New Zealand's worst natural disaster on record.

Most accounts trace the origins of H1N1 to US army camps. Another war-related hypothesis blames "conjunction of soldiers, gas, pigs, ducks, geese and horses" in army camps at Étaples in France for creating ideal conditions for the emergence of the flu in the trenches and army camps of the Western Front in 1916. Virologist John Oxford and co-authors drew attention to the outbreaks in camps at Étaples and at Aldershot in England in 1916 and 1917 of "purulent bronchitis", which they identified as phase two influenza, which was apparently contained within the camps. This interpretation also remains controversial, however.[85] Yet another interpretation of the propagation of the virus locates its origin in China, whence 94,000 labourers were shipped to England and France in 1917, as part of a plan to increase military recruitment for action on the western front. Most arrived via the Suez Canal, but about 20,000 took the Pacific route across Canada, where, it is claimed, a significant proportion of them ended up in medical quarantine with flu-like symptoms. This explanation has been greeted with some scepticism.[86]

The War and Public Health:

The war most likely increased the death toll from the flu in another way: by constraining measures that would have curbed the spread of the virus such as quarantining and social distancing on public transport and in the workplace. The dilemmas facing those responsible for public health are clear from the following

[84] Pool 1973: 275.
[85] Oxford *et al.* 2005; Oxford and Gill 2018.
[86] Humphries 2014; Worobey *et al.* 2019; Shanks 2016.

excerpt from remarks made by Sir Arthur Newsholme to a medical meeting two days after the Armistice:[87]

> I did in my official capacity in July prepare a memorandum for public use, but on the balance of considerations, its distribution was not considered expedient at that time. There are national circumstances in which the major duty is to "carry on", even when risk to life and health is involved. This duty has arisen as regards influenza among the belligerent forces, both our own and of the enemy, milder cases being treated in the lines; it has arisen among nutrition workers and other workers engaged in work of urgent national importance; it has arisen on a gigantic scale in connexion with the transport during 1918 of many hundreds of thousands of troops to this country and to France from overseas. In each of the cases cited some lives might have been saved, spread of infection diminished, great suffering avoided, if the known sick could have been isolated from the healthy; if rigid exclusion of known sick and drastic increase of floor space for each person could have been enforced in factories, workplaces, barracks and ships; if overcrowding could have been prohibited. But it was necessary to "carry on", and the relentless needs of warfare justified incurring this risk of spreading infection and the associated creation of a more virulent type of disease or mixed diseases.
>
> The same problem has arisen in connexion with crowded trains, trams, and omnibuses. These, doubtless, are prolific sources of infection but the service cannot be immediately increased, and meanwhile the vast army of workers must not be impeded by regulations as to over-crowding of vehicles in their efforts to go to work and to return home, and I have had no hesitation in recommending the Local Government Board to advise police authorities to this effect.

During the summer of 1918 Newsholme offered reassuring briefings to newspapers run by compliant press barons. That changed as the second wave intensified and the

[87] Newsholme 1918-19: 13. Newsholme's role during the pandemic is controversial. For opposing assessments see Eyler 1997; Honigsbaum 2008. See too Flecknoe, Wakefield and Simmons 2018; Tomkins 1992b.

armistice beckoned and, in the end, the *Times* lost patience with Newsholme's inaction. For the *Dail Mail*, the armistice was a signal to revert to the upbeat rhetoric of early summer: "The fear of influenza has vanished from the public mind. Patients suffering from it are being cured by the best of all remedial agents, cheerfulness".[88]

Army medical officers in the US recommended that war planning should allow for the potential risks of infection. Epidemiologists who inspected Camp Devens at the height of the outbreak there advised a halt to transfers in and out of the camp while the epidemic lasted. Against medical advice, however, infected soldiers were moved from overcrowded camp to overcrowded camp in packed trains and army vehicles. Pleas to delay the transport of troops to Europe were similarly of no avail. In mid-October even President Wilson pleaded for a delay, but gave way to chief of staff General Peyton Marsh's insistence that "the shipment of troops should not be stopped for any cause."[89] There was an explicit trade-off between military and civilian lives, but nobody sought to measure it. When the war was over, a senior medical officer lamented, "How many lives were lost I cannot estimate."[90]

When the pandemic struck in 1918, there were no medical tests or cures for infection; there was no systematic surveillance of its spread, no vaccines, no antiviral drugs, and no mechanical ventilators, available. Even today a recurrence of H1N1 would pose extraordinary challenges to medical resources and public health systems. That is not to deny that, then as now, non-pharmaceutical interventions (on which more below) could play a role.[91] How much public health measures reduced the incidence of the disease is a moot point. The most sophisticated work comes from the USA, where three studies of the pandemic in cities appeared in 2007. The first, in the *Journal of the American Medical Association*, constructed a database of 43 cities which included weekly deaths from the virus and details on three non-pharmaceutical interventions (NPIs): school closings, bans on public gatherings, and isolation and

[88] Citations from Honigsbaum 2013.
[89] Ashley Halsey III, 'A killer flu was raging. But in 1918, U.S. officials ignored the crisis to fight a war', *Washington Post*, 3 Feb 2018.
[90] Byerly 2010; Rasmussen 2014: 354.
[91] Jordan (2019) offers a good non-technical introduction to the discovery of H1N1 virus; Ingholt *et al*. (2020) found no link between access to medical care and mortality in Denmark.

quarantine. Simultaneously two more appeared in the Proceedings of the National Academy of Sciences: Hatchett *et al*. on the impact of a larger range of NPIs in a sample of 17 cities and Bootsma *et al*. in a sample of 23 cities. All found that the impact on peak mortality was greater than that on total or cumulative mortality. Cities where preventive measures saved many lives include St. Louis and San Francisco, while Philadelphia, which was slow to take action, recorded one of the highest death rates in large cities. A much-remarked feature of Philadelphia's attitude to public health was its decisions to allow a huge Liberty Loan parade at the height of the pandemic on 28 September and to keep the shipyards working at full tilt. In the wake of the parade Wilmer Krusen, the city's public health chief, announced: "The epidemic is now present in the civilian population and is assuming the type found in naval stations and cantonments [army camps]." The number of deaths per 100,000 from the flu in St. Louis (358) was less than half that in Philadelphia (748).[92]

Barro[93] reinterprets the data in the first of these as showing that the NPIs flattened the curves, but that their overall effect on deaths was small; in other words, for the most part, interventions delayed rather than prevented deaths. The likely reason for this is that the NPIs did not last long enough. Barro finds that a one standard deviation in his NPI variable reduces peak rate by over a quarter. However, the overall effect on death rates is weaker. All this research highlights the cost of premature ending restrictions. In yet another study of the impact of NPIs on mortality in cities Fuhrman[94], like the others, found that policy mattered. However, he also found that the impact of NPIs was greatest where cities were at risk from the propinquity of army bases. The impact of a city being located near a camp declined markedly with the duration of the intervention.

The range of non-pharmaceutical interventions (NPIs) suggested or imposed by policymakers varied from place to place. In July 1918 Sir Arthur Newsholme had contemplated banning large meetings of people, preventing overcrowding on public

[92] Kenneth C. Davis, 'Philadelphia Threw a WWI Parade That Gave Thousands of Onlookers the Flu', *Smithsonian Magazine*, 21 September 2018 (citing Krusen); Markel *et al*. 2007; Hatchett *et al*. 2007; Bootsma *et al*. 2007; Strochlic and Champine 2020.
[93] Barro 2020.
[94] Fuhrman 2010.

transport, and maintaining social distance in workplaces in the event of a recurrence of the pandemic, but the ongoing war put paid to such plans. Ireland's LGB merely issued generic advice regarding personal hygiene and staying in bed 'till all fever has gone'. Some local authorities issued recommendations of their own. The rather anodyne advice issued by the Public Health Committee of Dublin Corporation is a good example:

1 Keep away from crowded assemblies

2 Do not spit on the floor or tramcar or on the streets. Expectorated matter may be full of objectionable microbes. In sneezing keep a handkerchief on your face. Keep a little pad of cotton containing eucalyptus and smell it often, especially when in contact with other people

3 Allow plenty of air into your dwelling. Avoid crowded rooms

4 Vermin and dirt convey contagion. The strictest cleanliness should be observed.

5 Do not over-exert yourself or give way to a panic.

6 If you feel a pain in the head, of feverish, go to bed, and send for a doctor.

7 If recovering from influenza only see the persons you are obliged to see, so as to avoid infecting others.[95]

Sir Charles Cameron, Dublin's 88-year old chief medical officer, relied mainly on what behaviour economists would today be called nudging. At the height of the second wave, he "was glad to see that that the attendances at places of public amusement were showing a large decrease", he "earnestly entreated" managers of closed schools not to re-open until 4 November, and he recommended that public libraries cease circulating books. Rather than close Dublin's cinemas and theatres, he asked their managers not to admit children under 14 and to close for half an hour at 6 p.m. "for ventilation purposes"; rather than limit the numbers using public transport, he had "ascertained" that the city's trams were "most carefully disinfected" nightly. But in the Curragh and Newbridge area, near a major army camp, an order from the military

[95] *Irish Times*, 'The closing of schools', 28 October 1918; 'The influenza epidemic', 30 October 1918.

authorities for local theatres not to admit military personnel was sufficient to close them all down.[96]

The success of some U.S. cities such as St. Louis in 'flattening the curve' is well-known, whereas others such as Philadelphia fared poorly. The contrast between Western Samoa, where there were 8,500 influenza deaths (22 per cent of the population) and nearby American Samoa, where there were none, has also been well documented. Western Samoa's problems began with the arrival of the island steamer from Auckland on 4 November 1918 and the landing of six passengers suffering from influenza. Within a week the epidemic had spread across the main island Upolu and its neighbour Savai'i, with a morbidity rate of 90 per cent. Western Samoa had been a German colony but was occupied by New Zealand in 1914 and the responsibility for failing to place the steamer under quarantine rested with the New Zealand authorities:[97]

> We are strongly of opinion that Samoa should have been informed by wireless immediately influenza was, by regulation, made a notifiable disease in New Zealand, and that the Public Health Department and [or] the Defence Department failed in its duty in ignoring the fact that New Zealand was, for the time being at least, responsible for the welfare of the inhabitants of these islands.[98]

In American Samoa, by contrast, the US naval commander, acting on press wireless reports of the epidemic's progress, imposed a very strict quarantine, repulsing fugitives from nearby islands. It worked. The epidemic passed American Samoa by. The authorities in Australia also imposed a quarantine, which was not entirely successful, but which restricted the death toll to 15,000 or less than 0.3 per cent of the population. Islands dependent on Australia for outside contacts also fared relatively

[96] *Irish Times*, 'Influenza epidemic', 31 October 1918; 'Influenza in Ireland', 2 November 1918; 'The influenza epidemic', 2 November 1918. For an extensive discussion of these issues see Milne 2018: Ch. 6.
[97] Tomkins 1992a.
[98] Public Records Office. CO209/300/59822, Governor General of New Zealand to Alfred Milner, Colonial Secretary, 21 August 1919, end (as cited in Killingray 2003: 48fn81).

well whereas New Zealand suffered badly and so did its trading satellites such as Western Samoa and Fiji.[99]

The role of crowds in spreading infection has been highlighted during the Covid-19 pandemic. Their role was also recognized a century earlier and while there was rarely any general lockdown, many places where people congregated were closed. In the UK these included theatres and music halls, but most sporting competitions continued, and schools, pubs, and churches remained open, whereas in France theatres, schools, and churches were closed. Whereas in England decision-making was centralised, municipal authorities in the US had considerable discretion over what to do. The Surgeon General might circulate urgent recommendations on "the advisability [of] discontinuing all public meetings, closing all schools and places of public amusement on appearance of local outbreaks" but the decisions were made at state and municipal level. And at local level there were disagreements and conflicts between public health officials, business interests, citizens committees, and parents. In Minneapolis, for example, schools were closed between 12 October and 17 November, but the local school board bristled at the cost of paying teachers for not working and re-opened schools on 21 October, only to have them shut down again on police orders. Theatres and cinemas were temporarily shut but hoteliers resented the prohibition against the use of elevators; sports teams evaded regulations against playing; bars and restaurants broke closing orders. Masks were distributed but their use was not compulsory. There use was strongly urged in St. Paul but the secretary of the State Board of Health, while advocating them, would not wear one himself, explaining that "I personally prefer to take my chances".[100]

Another war-related event, the huge street celebrations that followed the announcement of the armistice on 11 November 1918, coincided with the peak of the second wave of the pandemic.[101] In Dublin "From Sackville St to St Stephen's Green a dense crowd filled the streets and cheered themselves hoarse. The windows of the houses were occupied by people wearing flags; the tops of tramcars were packed with

[99] Tomkins 1992a.
[100] Ott *et al*. 2007.
[101] *Irish Times*, 'Celebration in Dublin'; 'London Correspondence', 12 Nov 1918.

cheering passengers; motor cars were laden with jubilant occupiers; the jarveys had more 'fares' than their cars could carry…" In London as the news spread "it seemed as if half London had rushed into the streets… Lorries laden with soldiers and munition girls, and taxi-cabs 'festooned with Saxons' … passed along the principal streets in informal, but unending procession to a chorus of full-throated cheers… Most of the shops closed because the work-people wanted to join in the rejoicings, and this example was quickly followed by the factories and the Government offices."

But the claim the celebrations increased the death toll there had been no social distancing before them in any case. People had been at work as usual, etc. Indeed, in the wake of the celebrations in Dublin the *Irish Times*, then a Unionist paper, reported that "the electric feeling in the air for past couple of days and nights in Dublin has apparently 'electrocuted' the influenza germs in millions and routed them as completely as the Allies have routed the Germans".[102]

Armistice Day Crowds in London and Paris, 11 November 1918

Links between the flu and the outcome of the war have also been proposed. In his (arguably unreliable) memoirs General Ludendorff blamed the flu in part for the failure of the German spring offensive of 1918. The admittedly patchy data reproduced in Table 2 do, indeed, suggest that the first wave hurt German forces than those of the Allies. But the pandemic came too late to have a major impact on the course of the

[102] *Irish Times*, 'Influenza epidemic', 16 November 1918. This sentiment was

war, even if perhaps it was one factor among several that hastened its end.[103] In addition, two of the best-known historians of the pandemic—Alfred Crosby (1976) and John Barry (2004)—argue the third wave of flu influenced the peace, by incapacitating U.S. President Wilson at a crucial moment during the Versailles Treaty negotiations. They claim that a confused and physically weak Wilson yielded to the hard-line demands of French prime minister Georges Clemenceau. But that claim too remains controversial.

Table 2: Estimates of numbers of reported influenza cases among troops on Western Front, April-October 1918 [percentages in parentheses]

	April	May	June	July	August	September	October
German		77,000	135,002	374,524			420,000
French		24,886	12,304	2,369	3,135	24,282	75,519
American	1,850	1,124	5,700	5,788		37,935	38,655
British		36,473					22,136

Source: Phillips 2014

The Third Wave and After:

Not only was the third wave of the epidemic less lethal than the second: its spread was also slower and its geographical reach more limited. It seems not implausible to link this in part to the absence of the troop movements that were so much a feature of the previous year. The third wave seems to have spread from Australia, which had been hit lightly during the second wave thanks to a maritime quarantine, to Europe and the United States, where it peaked during the spring on 1919. Mexico was hit too.[104] However, the timing and scale of the third wave have not been studied much.

Apart from its direct impact, the 1918-19 pandemic has also been linked to the spread of *Encephalitis lethargica* or 'sleeping sickness', a debilitating neurological condition first diagnosed by Viennese neurologist Constantin von Economo in 1917. The syndrome began its spread across the globe in 1916-17 and disappeared in the early

[103] Bauer and Vögele 2014; Phillips 2014a.
[104] https://earth.org/data_visualization/pandemic-map-the-spanish-flu/.

1930s. The number of victims is unknown but a figure 1 million is often mentioned, many of whom died. Von Economo, like other specialists, was inclined to think that the influenza made people more vulnerable to *Encephalitis lethargica,* but that interpretation has by now been largely discredited.[105]

Our survey has pointed to several ways in which the war impacted on the pandemic. In an early review of the pandemic's historiography, Phillips (2004: 131) lamented the lack of a "systematic investigation" of the link between World War 1 and the pandemic. The gap has not been filled since, although several studies have modelled the spread of the epidemic in particular countries, and most highlight the link to wartime activities. Smallman-Raynor *et al*. model the three waves for Britain and show that whereas the first wave spread from north to south, the second spread from south to north. They identify cities in northern England, in the midlands, and London as being to the fore in the first wave, whereas a hallmark of the second was "the early involvement of southern localities (most notably, the seaports of Portsmouth and Cardiff) [which] may be attributed to the early and heavy seeding of some south coastal settlements by naval personnel. They also offer a detailed account of the spread of the virus in the university city of Cambridge, "especially valuable because the data …[span] the town, the colleges of the university, and the military contingent billeted in the town". The outcome underscored "the pivotal role of highly mobile military populations, both in … the initial introduction of the influenza virus and … the localized 'supercharging' of disease transmission."[106] Another interesting analytical study of the spread of the pandemic models the spatial diffusion of the pandemic in India and shows that it faithfully followed local and national railway networks, with areas without railways and away from the coastline less likely to be infected.[107] Such patterns are consistent with more impressionistic accounts.

A twelve-country study by Yoneyama and Krishnamoothy[108] relies on implausibly high estimates of deaths in China and Russia. The most impressive

[105] Leslie A. Hoffman and Joel A. Vilensky, 'Encephalitis lethargica: 100 years after the epidemic', *Brain: A Journal of Neurology*, 2017: 140; 2246–2251.
[106] Smallman-Raynor *et al*. 2002.
[107] Reyes *et al*. 2018.
[108] Yoneyama and Krishnamoothy 2010.

attempt so far at simulating the spread of the virus and its links to WW1 is the model created in connection with a website marking the war's centenary. The model explicitly addresses the counterfactual of a war-less pandemic, allowing for various historical settings and theories as to where the flu originated, and for the impact of events such as the armistice celebrations and the arrival of US forces in Europe.[109] But we still know too little about how, and how fast the virus was transmitted, and about what life might have been like had there been no war, or had the war ended sooner. The question "How much did H1N1 add to the civilian death toll of WW1?" is currently unanswerable. Suppose, however -- and this pure speculation -- that the war increased deaths from the flu by one-tenth. Then even on the reduced estimate of the pandemic's death toll proposed above, WW1 added 3.5 to 4 million, or at least a quarter, to a new estimate of civilian deaths from other causes.[110]

[109] See: The 'Spanish' Influenza pandemic and its relation to World War I' (http://ww1centenary.oucs.ox.ac.uk/?p=2190) by Kenneth Kahn (http://ww1centenary.oucs.ox.ac.uk/author/kkahn/) licensed as Creative Commons Attribution-Non-Commercial-Share Alike 2.0 UK: England & Wales (http://creativecommons.org/licenses/by-nc-sa/2.0/uk/). Pandemic experts John Oxford and Douglas Gill offered historical guidance for the project.

[110] Ó Gráda 2024.

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