The brevity of the influenza pandemic of August–September 1918 posed great problems to doctors at the time... It has posed great problems to historians ever since.

Terence Ranger

The “Spanish” influenza virus—or at least its viral offspring—have been circulating between the northern and southern hemispheres for 100 years now, but it is arguably only in the past few years that histories of the pandemic virus have achieved a similar ubiquity in our culture. Indeed, in a period that has seen a steady flow of books and films about World War 1, it should come as no surprise that the 1918–19 influenza pandemic should have become an object of similar popular interest, with the BBC, America’s National Public Radio, and the US Smithsonian Institution all commissioning major documentaries and exhibitions to mark its centenary this year.

This, it could be argued, is no less than victims of influenza deserve. After all, in 11 months between the spring of 1918 and the winter of 1919, influenza killed at least 50 million people worldwide. That is 40 million more than perished on the killing fields of Flanders and northern France and 10 million more than AIDS has killed in nearly 40 years. The result is that a disease event that medical historians once described as “forgotten” is now anything but. On the contrary, the 1918 pandemic has become a subject of respectable academic study and something of a global publishing phenomenon.

At this point, I must plead mea culpa for I was one of those historians who, lamenting the pandemic’s neglect by my colleagues, once promoted the notion that it had somehow been forgotten or excised from public memory. But for an event to have been forgotten it must first have been remembered in a significant way. Yet except for those who nursed the sick or watched loved ones succumb to the deadly pneumonic complications of the disease, there is scant evidence the 1918–19 pandemic left a deep impression on the collectivity of society in the generation that lived through it. Sure, one can find memorials testifying to the courage of doctors and nurses and extraordinary works of art such as Edvard Munch’s Self-Portrait with the Spanish Flu. And searches of digital archives reveal reports by medical officers of health attesting to the measures taken to stem the spreading infection and the conspiracy theories peddled by newspapers at the time (some blamed German U-boats; others, the deployment of mutagenic gases on the Western Front). But with the exception of Katherine Anne Porter’s extraordinary 1939 short story, Pale Horse, Pale Rider, inspired by her near-death experience of influenza while working as a newspaper reporter in Denver, Colorado, USA, in 1918 (her colleagues were so convinced she was a goner they set her obituary in type), the virus inspired few works of fiction or non-fiction, and for those who were there it was the absence of concern that was most striking. As The Times commented at the height of the deadly second wave of the pandemic in December, 1918, “Never since the Black Death has such a plague swept over the face of the world [and] never, perhaps, has a plague been more stoically accepted.”

Why this was the case has puzzled commentators ever since. “There is some psychological interest in the fact... that actually the emotional impression created [by the influenza pandemic] was fainter than that produced by much less grave epidemiological happenings”, observed the epidemiologist Major Greenwood, one of the authors of the official British Government report on the pandemic, in 1935. 40 years later, the American environmental historian Alfred Crosby was similarly perplexed: “One searches for explanations for the odd fact that Americans took little notice of the pandemic, and then quickly forgot whatever they did notice.” The critic H L Mencken thought the answer lay in a basic feature of human psychology, writing that “the human mind always...
tries to expunge the intolerable from memory”. But if so, why is it that the 14th-century Black Death, a similarly intolerable and devastating outbreak, saw an outpouring of plague tracts and memento mori?

These are not easy questions to answer. The historian Terence Ranger suggests that the brevity of the pandemic, with most of the deaths concentrated over 4 weeks in the autumn of 1918, mitigated against a "conventional vertical historical narrative". Others point to the scale of the catastrophe and the impossibility of imagining deaths of the order of tens of millions, as well as the timing of the pandemic and the way, in Europe at least, it was overshadowed by World War 1 and the Armistice celebrations in November, 1918. Finally, there is the fact that, unlike today when 24/7 news channels and social media ensure that reports of new disease outbreaks are broadcast far and wide, in 1918 most of the belligerent countries suppressed news of the influenza for fear of panicking civilians. By contrast, in neutral Spain, foreign correspondents in Madrid freely reported influenza’s depredations, hence the moniker “Spanish” that sticks to the pandemic to this day.

Perhaps the biggest mystery is not the dearth of emotional and cultural traces of the pandemic, but when and where influenza originated and why it proved so much more virulent than any other pandemic influenza virus before or since. Until the late 1990s, historians thought there was little chance of medical researchers ever being able to answer these questions. “It has been the dream of scientists working on influenza for over a half century to somehow obtain specimens of the virus of Spanish influenza, but only something as unlikely as a time capsule could provide them”, ventured Crosby in 1976.

In 1997, however, scientists based at the US Armed Forces Institute for Pathology obtained just such specimens, not from a time capsule but from an overweight Inuit woman, known as “Lucy”, who had been buried in a mass influenza grave overlooking the Seward Peninsula in Alaska. The permafrost had preserved just enough of Lucy’s lung tissue to enable the molecular pathologists Jeffrey Taubenberger, Anne Reid, and their colleagues to retrieve fragments of influenza virus from her cells. Combining the viral RNA with other fragments from preserved lung autopsy material obtained from American soldiers who had died of influenza between September and November, 1918, and using PCR—a technique that had not been available when Crosby wrote his book—to fill in the missing bits of genetic code, by 2005 Taubenberger and his colleagues had succeeded in reconstructing the virus’s entire genome.

Their research findings in *Nature* came as a shock—and not only to historians. Previously, epidemiologists had observed that influenza pandemics were preceded or followed by epizootics of influenza-like illnesses in dogs, cats, and horses. Furthermore, shortly after the 1918 pandemic, pig farmers in the midwestern USA had observed similar respiratory illnesses in swine, and in 1933 a group of British researchers had succeeded in transmitting a descendant of the 1918 pandemic influenza virus to ferrets. But until the announcement in *Nature*, few scientists thought “Spanish” influenza might have originated in wild waterfowl. But as Taubenberger put it, the 1918 pandemic virus was one of the most “avian-like” mammalian influenza viruses he had ever encountered. So much so that he could not discount the possibility that it had jumped directly to human beings from birds without transiting through an intermediary mammalian host first.

Since Taubenberger and colleagues’ groundbreaking paper, these questions have only deepened. Although most people in 1918 suffered nothing more serious than a mild illness, in about 2·5% of cases the virus penetrated deep into the respiratory tract causing extensive inflammation and cyanosis, a ghastly mauvey-blue discoloration of the lips, ears, and cheeks as victims’ lungs filled with choking fluids. This could explain why, in association with secondary pneumonia-causing bacteria, the death toll was so high. However, it does not explain why two-thirds of the victims were adults aged between 20 and 40 years, which is the reverse of most influenza seasons where mortality falls most heavily on people aged 60 years and older.
or children aged 5 years or younger. Nor does it explain why school-age children, who should have been similarly susceptible, fell sick but did not experience the same fatal complications as adults.

Using molecular clock techniques to compare all the influenza viruses that have or are currently circulating in different bird and mammalian hosts over the past century, the evolutionary biologist Michael Worobey reported that seven of the eight genes of the 1918 virus closely resembled influenza genes found in birds in North America. That would seem to support the claim by John Barry, the author of *The Great Influenza*, that the pandemic strain originated in a remote farming community in southwest Kansas from where it spread to Camp Funston, Fort Riley, and other large US Army training camps along the eastern seaboard of the USA, before being introduced to northern France via American transatlantic troop carriers. Certainly, the earliest report of what was retrospectively identified as the spring wave of the 1918 influenza pandemic occurred at Camp Funston when a soldier there fell ill on March 4, 1918. In all, 1100 soldiers were admitted to the camp’s hospital, forcing orderlies to requisition a gymnasium for the overflow. But unlike the later summer and autumn influenza waves, there was no sign of cyanosis and although 38 soldiers died most suffered nothing worse than a mild 3-day illness. Even more problematic for Barry’s theory is evidence of what looks like a herald wave of “Spanish” influenza in New York, USA, in February through April, 1918, and similar outbreaks in military camps in Norway around the same time, followed by a pre-pandemic wave in Copenhagen, Denmark, in the early summer. These findings are hard to reconcile with Barry’s thesis and reopen the possibility that the virus might have been introduced to the USA from Europe, not the other way round.

Yet alternative hypotheses—that the pandemic began at Etaples, a vast military hospital camp near the bird migratory routes at the bay of the Somme, or that the virus was introduced to France by Chinese labourers—are also hard to reconcile with the available virological and epidemiological evidence. For instance, 1 year before the pandemic, two papers in *The Lancet* described an unusual respiratory disease that had erupted at Etaples and Aldershot barracks in the winter of 1917. Labelled “purulent bronchitis” for want of a better term, the disease proved fatal in half the cases and many soldiers also developed cyanosis. 2 years later, British respiratory experts, also writing in *The Lancet*, would decide the disease had been “fundamentally the same condition” as “Spanish” influenza and commissioned an artist from the Royal Academy to capture the characteristic stages of cyanosis. However, there is no record of similar outbreaks in the winter of 1917 among civilians living in the vicinity of Etaples or Aldershot, which is what you would expect if purulent bronchitis had been the precursor of the pandemic virus. Having said that, if a time capsule can turn up once it can surely turn up a second time; in this case, what would be needed is pathology material from the purulent bronchitis outbreaks containing genes of the 1918 H1N1 influenza virus.

In the meantime, the continuing mystery of the influenza’s origins—and what it might take to trigger another similarly catastrophic pandemic—is good news
for medical researchers, historians, and broadcasters. After all, what better way to unlock money for gain-of-function virus research or trials of vaccines and antiviral drugs than by raising the spectre that disaster could strike again. And what better way for a budding writer to make their name than by retelling the story of the influenza in the centenary year of the pandemic.

Indeed, if the first wave of influenza scholarship was marked by Crosby-style national histories, each asserting the social and cultural—though rarely the political—significance of the pandemic for the UK, Australia, New Zealand, Canada, and South Africa (in the first wave of influenza scholarship, the UK and its former dominions tended to lead the way), the latest wave has seen researchers embracing a global perspective.

Despite the passage of time and the acres of ink that has been spilled on the subject, astonishingly these researchers are still turning up fresh perspectives and leads. At the UK’s Imperial War Museum in London, Hannah Mawdsley—with whom I am collaborating on Going Viral, a new podcast series about the pandemic—has unearthed a vast treasure trove of correspondence—some 1700 letters in all—collected by the military historian Richard Collier in the course of researching his 1974 book, The Plague of the Spanish Lady. Dispatched from every corner of the globe, the letters in the Collier Collection are testimony to the pandemic’s searing impact on those individuals who survived, particularly women who comprised two-thirds of Collier’s correspondents. Far from being excised from personal memory, it would seem that the pandemic was an experience that, in the words of one Scottish woman, she “could never forget”. This sentiment is echoed again and again in the Collier Collection. “I can remember very well the cortège was on its way to the church”, wrote another woman, recalling the double funeral of her mother and 7-year-old sister in Coventry on the same day as the Armistice. “Bells, hooters and all sounds of celebrations. It was raining but how silent people stood...I would like to tell you I am 64 years of age now but that period of my life I will never forget.”

In the latest addition to the influenza’s burgeoning historiography, Pale Rider: The Spanish Flu of 1918 and How It Changed the World, the science writer Laura Spinney travels far from the Western Front in search of places where the influenza was not overshadowed by war. In countries distant from the European theatre she argues the influenza had lasting political and social impacts, pushing “India closer to independence, South Africa closer to apartheid, and Switzerland to the brink of civil war”. Far from being ignored, in these places the 1918 influenza pandemic left “humanity transformed”. Whether or not you agree with such sweeping claims—and many historians do not—it is hard not to see Spinney as the heir to Crosby, who set similar historical hares running with his controversial claim that by sickening US President Woodrow Wilson during the Versailles peace negotiations the influenza resulted in the US delegation abandoning Wilson’s 14-point peace plan, opening the way to harsh reparations against Germany, thereby sowing the seeds for the rise of Nazism and the second great conflict of the 20th century (indeed, Crosby’s book was originally titled Epidemic and Peace; it was only in the wake of the AIDS pandemic, that in 1989 it was reissued as America’s Forgotten Pandemic).

However, there is a sense in which Spinney is correct to say that the 1918 influenza pandemic transformed humanity, for the genes of the 1918 virus continue to circulate in human and pig populations to this day. Some of these genes are direct descendants of the 1918 H1N1 influenza virus; others have reassorted with subsequent pandemic viruses, such as the 1957 “Asian” influenza A H2N2 virus and the 2009 swine influenza A H1N1 virus, to produce new influenza subtypes. From a genetic point of view, they can all be seen as belonging to a single pandemic era that began in 1918. In this sense, argue Taubenberger and David Morens, the “Spanish” influenza pandemic of 1918–19 can be considered the “mother” of all pandemics. Even 100 years later it still has the capacity to chill us and thrill us.

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The Going Viral podcast is by Blakeway, part of Zinc Media Group, and is supported by the Wellcome Trust. Mark Honigsbaum’s book The Pandemic Century will be published by W W Norton & Company in 2019.

Further reading
Spinney L. Pale rider: the Spanish flu of 1918 and how it changed the world. London: Jonathan Cape, 2017