

History of epidemics informs modern science

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Soldiers from Fort Riley, Kansas, ill with Spanish influenza at a hospital ward at Camp Funston. Credit, Wikimedia Commons.

As well as telling us more about earlier societies, the study of diseases in the past is proving an invaluable tool for modern science, as a new book by the historian of medicine Mary Dobson reveals.

Diseases such as bubonic plague, smallpox, or scurvy, killed so many and



caused such misery that they are still household names today, even if for most of us they are things of the past. From an historical perspective, they are also fascinating. By studying these illnesses and their impact, we can understand more about the people for whom such horrors were commonplace and real. Moreover, remarkable and inspiring tales of scientific endeavour are often part of the story of how they were controlled, treated, in some cases cured, and, in the case of smallpox, even eradicated from the globe.

Yet the study of these conditions is about more than simply gleaning historical information from a cabinet of increasingly distant medical curiosities. As time goes by, scientific knowledge is not just informing the work of historians; it is being informed by it. Many modern-day historians of medicine are operating more and more like pathologists and epidemiologists in their efforts to understand what caused the most disastrous pandemics of previous centuries, and how and why they spread. Their work is providing vital new information in the fight against modern-day "plagues", such as cancer and dementia. More worryingly, it has started to highlight cases where millions died for medical reasons that remain obscure, raising some pressing "what-if" scenarios about our future.

In a new book published this week, Murderous Contagion, the historian of medicine Mary Dobson examines 30 of the biggest killers in the history of humankind, from scourges like the Black Death of the 14th century, to modern epidemics such as HIV/AIDS, and the still-developing Ebola crisis. Rather than simply focusing on the gruesome history of disease itself, however, or the often agonising treatments administered to earlier generations of patients, the study also shows how modern science and the history of medicine have come to depend on each other.

For one thing, historians are now able to take advantage of a growing



body of scientific knowledge for their research. We know more than we ever have, for example, about how diseases "jumped" the barriers between species and spilled over from bats, birds, wild and domestic animals to humans, often adapting in their hosts as they spread. New techniques for recovering and analysing ancient DNA are also making it easier to identify past pathogens that were previously mysteries.

As we understand more about historical outbreaks, however, we are also learning more about human susceptibility to certain diseases, and how they might be prevented from recurring. History is increasingly capable of providing modern science not just with a record of what happened, but with information about why.

"Historians of medicine are moving closer to modern science as they come to understand more about the origins of disease," Dobson, who is based at St John's College, University of Cambridge, explained. "It's vitally important to get on top of where and how diseases originate before they have a chance to spread, and history can play an important role in this work. Understanding these stories is important for stopping diseases in their tracks, and fundamental to the goals of advancing global health in the present and future."

Dobson's book features striking examples of cases in which historical knowledge has, unexpectedly, become relevant to modern medical practice. In the 1950s, for example, two British epidemiologists decided to investigate the cause of a surge in cases of lung cancer that had become apparent during the previous decades. They predicted that this would most likely turn out to be exposure to car exhaust fumes, or possibly the tarring of roads.





Policemen in Seattle wearing masks made by the Red Cross, during the Spanish Influenza epidemic, December 1918. Credit: Wikimedia Commons

As an outside possibility, they also considered smoking as another potential reason for the spike in cases. Some scientists thought this unlikely, but speculations about a link dated back as far as the 17th century, when James I himself had warned that tobacco was "hateful to the nose, harmful to the brain [and] dangerous to the lungs". As we now know, smoking was indeed discovered to be the main cause of this emerging tragedy, and public health campaigns on the subject have become more plainspoken and forthright ever since.

The potential for BSE, better known as "Mad Cow Disease", to infect humans as variant Creutzfeldt-Jakob Disease (vCJD), might similarly never have become evident without historical knowledge of an obscure condition known as Kuru. This disease, characterised as "the trembling death", had first been observed in the early 20th century among the indigenous population of Papua New Guinea. Symptoms included involuntary tremors, jerks, uncontrollable outbursts of laughter, loss of co-ordination, wasting and eventually death. The brains of people who



had died from the disease were found to be riddled with holes. Extensive research eventually posited a link with ritualistic cannibalism, and thanks to the ending of such rites in the mid-20th century, Kuru all but disappeared, vindicating the theory.

These unusual symptoms were also apparent in some animal diseases, such as scrapie in sheep and in BSE in cows. In each case, the brain was found to have become "spongiform", or Swiss-cheesed with holes. Scientists eventually linked these conditions to a new agent of infection; rather than a virus or bacterium, Kuru, scrapie and BSE were caused by an aberrant protein called a prion.

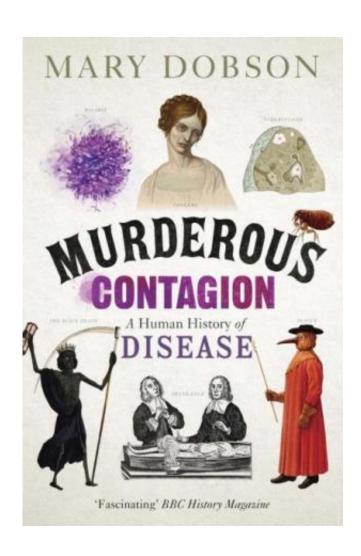
The cannibalistic connection that had led to these symptoms emerging in Kuru opened up the disturbing possibility that BSE had emerged due to "high-tech" cannibalism, in the form of cattle feed made of proteins derived from sheep and cattle. By the 1980s, it was not just clear that this had happened, but that scrapie may have made an inter-species jump to become BSE in cows. If that was true, then it was equally possible that a similar leap could occur between cows and humans, particularly when cases of 'new variant' CJD started to emerge in young people.

This realisation formed the basis of the mid-1990s scare over BSE and vCJD in Britain. The feared large-scale epidemic of vCJD has not materialised, however, partly because offal had already been removed from cattle feed and tight controls put in place to keep infected meat out of the food chain. The link to Kuru, and subsequent discovery of prions, was critical: "What had begun as a mysterious disease in Papua New Guinea and an esoteric discussion in scientific circles about the cause of a rare class of animal and human neurological disorders has led to the revolutionary discovery of a new biological principle of infection in the form of prions," Dobson writes.

In recent times, the story has taken a fascinating new twist as this



research has begun to be linked to modern work on neurodegenerative disorders such as Alzheimer's and Parkinson's diseases. Although these diseases are not infectious, they are, it has emerged, like the Kuru-vCJD family, associated with the "misfolding" and malfunctioning of proteins. There is also growing evidence that the mechanisms by which these diseases progress could indeed be very similar. Even the recent upsurge in cases of type 2 diabetes appears to be linked to this "misfolding" phenomenon.





Elsewhere, the book highlights cases where the identity of the historical causes of a <u>disease</u> could prevent similar disasters occurring. Following the recent outbreaks of avian flu and swine flu, the mystery that surrounds historical pandemics of influenza is of especial concern. In particular, the cause of Spanish Influenza, which killed at least 50 million people between 1918 and 1920 – the highest death toll of any pandemic in human history – was unknown at the time.

Despite being one of the most deadly diseases of all time, Spanish Flu was until recently so little-studied it was sometimes characterised as a "forgotten" pandemic. Now scientists and historians are joining forces to understand how it began, how it spread, and why it was so lethal, especially in young adults. The traditional theory that it was disseminated by troop movements during and after World War I fails to explain why some of the worst-affected countries, such as India and Samoa, were far from the main theatres of conflict.

To date, there has been no subsequent global influenza pandemic of such lethality. H5N1 (bird flu), while potentially dangerous, has displayed limited capacity to "jump" the species barrier as initially feared, thereby, hopefully, eliminating any possibility of widespread human-to-human infection. H1N1 (swine flu) in 2009 was a more worrying example of "reassortment", a process by which different types of flu combine into new strains, but was probably effectively contained through careful screening, quarantine programmes and efficient drug delivery – although not until it had claimed perhaps as many as 200,000 lives.

Both cases, however, demonstrate the urgency with which historians need to understand what caused the far more devastating Spanish flu pandemic. Researchers are now investigating the subject in the hope of finding more answers, and have even gone so far as to exhume the remains of victims from the permafrost to comprehend its cause, with the latest findings suggesting that it might, indeed, have been a novel



form of bird flu.

Nonetheless, there are still questions to be asked and solved: "We still don't know why and how Spanish Flu went global," Dobson said. "But if we want to stop virulent flu pandemics from happening again, we really need to know more about why they happened in the past."

Murderous Contagion: A Human History Of Disease by Mary Dobson is published by Quercus on March 6th, 2015.

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