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Second Opinions: Final Response

Epidemics and Infections in Nineteenth-Century Britain

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We would like to thank Graham Mooney and Andrew Noymer and Beth Jarosz for their responses to our 'Second Opinion' on 'Infectious Disease and the Epidemiological Transition in Victorian Britain'.[1] Mooney offers a robust attack on our general claim that the importance of infectious diseases as a cause of death in the nineteenth century has been overstated, while seeming to accept what we say about epidemics, while Noymer and Jarosz take us to task on what counts as an infectious disease and also provide a critique of the specific limitations of our claims when applied to New England. We are pleased that our piece generated such reactions and hope that the debate will continue.

Mooney was mightily offended by our apparent neglect of the work of historical demographers, and on reflection we regret not giving this aspect more attention, including the excellent articles and chapters that Mooney has published over the years.[2] For this, we apologise. However, historical demographers were not our target, for we are well aware of the work they have done and the nuanced picture they have now presented of changes in mortality patterns; indeed, we would have been unable to present our piece without this work. That said, one of our key points was that the complex picture they have produced has yet to find its way into mainstream social and economic history books, or popular understandings of Victorian Britain. Why this is so we can only speculate.

Part of the reason may be that the work of historical demographers, with very notable exceptions, such as that of Wrigley, Schofield and Woods, has not been accessible to less numerate colleagues, who seem to prefer dramatic contrasts between disease and death

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[1]Mooney 2007; Noymer and Jarosz 2008.
[2]Mooney 1997*a*; Mooney 1997*b*; Mooney 1999; Mooney 2002; Mooney *et al.* 1999.

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then and now.[3] As we noted, one example of this is representations of the 'epidemiological transition' that show the great majority of deaths switching from infections to degenerative diseases. Mooney states that these are 'clumsy', an interesting term for presentations which are wrong. We feel there is still work to be done in ensuring that the new findings on nineteenth-century mortality patterns are taken up in the wider histories of Britain that are written for students and the public.

The increase in average longevity in Western industrialised countries is dated from the early nineteenth century and continues to dominate historical demography. There is a strong consensus, from which we do not dissent, that the main reason for this was a decline in child and early adult mortality, due to a decrease in deaths from infectious diseases in children and pulmonary tuberculosis in young adults. While these changes were demographically important, leading to more people reaching middle age and then old age, deaths amongst children and young adults only accounted for one in three of all deaths. Some historians have assumed that what happened in relation to childhood mortality was repeated at other ages.

For example, J. C. Riley, whose studies of patterns of morbidity have been exemplary, recently wrote in the context of Omran's 'second stage' of the mortality transition, that 'adult death rates also declined, presumably because adults, too, died, less often from communicable diseases'—note the word 'presumably' and crucially that the point was not referenced.[4] It is surprising that after kicking over the 'rubble strewn' in our article, Mooney's conclusion about the prevalence of infectious diseases is not all that different from our own estimate. We suggested that 33 per cent of deaths were from infectious diseases in the mid-nineteenth century. Mooney suggests 40 per cent as 'a reasonable approximation'. Noymer and Jarosz cite United States data that show the proportion of deaths from infectious diseases at around 40 per cent in 1900 and suggest that earlier it would have been higher. We have no problem in accepting these figures; indeed, they are supported by the work of Armstrong, Conn and Pinner, though these authors' concern is with twentieth-century epidemiological changes.[5]

Mooney takes us to task principally over our claims about infectious diseases. We must first make it clear that, contrary to his suggestion, we never wrote or implied that 'infection was *not* part of the common disease "experience" in Victorian Britain' (emphasis in original).[6] This would be as absurd a claim for the nineteenth century as it would be for today, when colds, influenza, athlete's foot, and so on, are so prevalent. Thus we are not clear what point Mooney's charting of the many reports of infectious diseases in primary sources and the secondary literature is making. The only illustration that seems to be relevant relates to the growing number of isolation hospitals in the Victorian era. Yet we would see this development as supporting our argument because, as many historians have shown, they were built and used quite reluctantly, in large part because of doubts amongst doctors and the public about their value.[7]

[3]Woods 2000; Woods 2007; Wrigley and Schofield 1989.
[4]Riley 2001, p. 22.
[5]Armstrong *et al.* 1999.
[6]Mooney 2007, p. 598.
[7]Pickstone 1985, pp. 156–83; Eyler 1987.

The main point of contention between Mooney and ourselves is over what should count as 'infectious diseases'. We are accused of adopting a definition that is 'misleadingly narrow'. Our usage may be 'narrow' in comparison to his wide and ahistorical usage, but it is only 'misleading' if one accepts as 'infectious' any disease in which a microorganism plays a role. Thus his characterisation spans everything from highly contagious smallpox, to opportunistic, secondary conditions that occur in relation to pneumonia. Indeed, he seems to want to add a number of cancers to the category. Our use of the term follows that of nineteenth-century doctors and public health officials, who reserved it for conditions that are directly or indirectly communicable. Hence, we count smallpox as an infectious disease, but not pneumonia. As we explain below, this is for both historical and contemporary medical reasons.[8]

One of the aims of our article was to direct greater attention to contemporary nineteenth-century perceptions of infectious diseases. Previous discussions along these lines have focused on the accuracy and reliability of cause of death certification; we were adding to this an interest in the meanings of contemporary disease categories. Mooney, like most demographic historians, has a different agenda: to trace changing patterns of infectious disease according to criteria accepted in the late twentieth and early twenty-first centuries. That said, his work is amongst the most historically sensitive. The task of translating nineteenth-century data on causes of death into current notions of the causes and nature of disease is, as everyone acknowledges, fraught with danger.[9]

For example, we were surprised at the confidence with which Noymer and Jarosz assured us that, 'Croup *is* caused by parainfluenza virus and by respiratory syncytial virus' (our emphasis).[10] In the nineteenth century, as William Jenner explained in lectures at the end of 1874, croup was a non-specific, variable inflammatory condition. It was understood to be mainly caused by cold and damp, and difficult to differentiate from spasms of the larynx, diphtheria and other laryngeal conditions.[11] The complexity, which included nervous croup, was evident in the correspondence that followed his lecture.[12] As noted above, historians cannot assume that the nineteenth century witnessed diseases as defined today.

Categorisations of infectious diseases that are projected back on to the early Victorian period are particularly problematic, as it makes no sense to think about diseases categorised as 'miasmatic'—produced by 'noxious and infectious vapours'—as microbial or

[8]The term 'infectious diseases' has no agreed meaning in current medical discourse. The most common use, exemplified in the work of bodies like the World Health Organisation and the Centers for Disease Control and Prevention is in relation to 'communicable diseases' in public health contexts. However, there is also the notion of 'clinical infectious diseases' which focuses on patients with microbial conditions in primary care and hospitals, including pneumonia. The different sites of practice are important, since with clinical infectious diseases there is less interest among doctors in communicability, as their focus is on the management and treatment of disease already present.

[9]For example, in a 2002 article on mortality during the urban epidemiological transition in Victorian London, Mooney keeps to Registrar General categories, although he does surprisingly translate 'zymotic' to 'infectious'. Mooney 2002, p. 30.

[10]Noymer and Jarosz 2008, p. 574.

[11]Jenner 1875.

[12]Lancet, 1875, I, pp. 216, 252, 285–6, 321, 353–5, 387–8, 423–4, 456–7, 592, 662–3, 705–6, 776.

communicable. Miasmatic diseases were said by contemporaries to be 'zymotic', a term that linked disease to fermentation, which suggested either chemical or biological processes that produced and spread morbid poisons. Indeed, one link between infectious diseases and insanitary conditions was that the latter could produce morbid poisons and that they weakened the whole body making it more susceptible to low doses of poison and/or to their spontaneous generation.

Obviously, no disease could have been accepted by doctors or the public as caused by microorganisms until after 1870, and the coming of bacteriology, and even then aetiologies and pathologies were debated for many decades, as was the interplay of infection and immunity.[13] These changing assumptions shaped the nosologies developed to classify and record causes of death, and the actions of the doctors who filled in the forms and those who aggregated and analysed their data. The apparent confidence Mooney, Noymer and Jarosz seem to have in translating this complexity into a single category of 'infectious diseases' is surprising.

It is worth dwelling a little longer on the term 'infectious' since we also want to respond to Mooney's points about our discussion of lung diseases. To do so, we need to say more on the notion of infection in medicine then and now. In the nineteenth century, there was a commonly drawn distinction between contagious diseases that were spread by contact, like smallpox, and infectious diseases, like cholera, that were spread indirectly via some medium, such as air or water. Among pathologists, there was an important distinction between 'infection' where a morbid poison entered the body from without, and 'infective processes' where inflammation and morbid conditions spread within the body. In this context, cancer was seen as an infective disease, as was tuberculosis; in both cases seemingly spontaneously generated changes in tissues spread contiguously and by metastasis. Mooney suggests that if we are to work with historical actors' categories then we ought perhaps to regard cancer as an infectious disease, because some late nineteenth-century doctors thought it was caused by protozoa. However, the single reference to cancer in Worboys's *Spreading Germs* makes it clear that the notion had very few supporters and was anyway short-lived.[14]

The need for historical sensitivity is evident in Mooney's failure to understand our view of pneumonia and bronchitis. He regards our unwillingness to regard these two diseases as infectious as a 'bizarre exclusionary manoeuvre'. Of course, it depends on what is meant by 'infectious', then and now. Let us start with now. The current understanding of pneumonia is that it is produced by a variety of microorganisms, but that in the vast majority of cases, the disease only develops in people with a pre-existing medical condition, or in specific vulnerable groups.[15] For example, it is most likely to be a cause of death as a secondary condition in the very old, in those with other chronic lung conditions and in those who smoke, and in patients weakened by long-term illnesses, especially those that compromise the body's immune responses.

Pneumonia used to be called 'the old man's friend', as it brought a rapid, often painless death to those with a terminal illness. Indeed, modern public health doctors use the term

[13]Worboys 2000, *passim*; Worboys 2007.
[14]Worboys 2000, p. 246.
[15]Karetzky *et al.* 1993, *passim*.

'community acquired pneumonia' or 'walking pneumonia' for the disease for the small number of cases in which the disease is 'caught' by a previously healthy person. In addition, at least 10 per cent of healthy adults in industrialised countries today have the pneumococcus present in the nasopharynx region. The important point is that it is wrong to operate with a simple model of the 'presence of a microorganism equals disease'. Infection is a far more complex process in which one needs to think ecologically about environment–host–pathogen interactions.

In the Victorian period, doctors understood the pathology of pneumonia as involving the inflammation of lung tissue, which could be prompted by predisposing, inherited and acquired factors, by exciting causes such as damp, cold, dusts and by other lung diseases. Only after 1880 were such inflammatory diseases linked to microbes, and then only for a few such diseases. More generally, the condition of the soil (the lung tissue) was seen to be more important than the presence of any seed (irritant or microorganism). This was then confirmed by bacteriological investigations, which showed that many healthy people harboured the pneumococcus and other pneumonic organisms in their bodies without developing pneumonia. As Worboys shows in *Spreading Germs*, most late nineteenth-century doctors also worked with an ecological view of infectious diseases.[16]

Similar points can be made for bronchitis. In most cases, bacteria or viruses play a role, but there is no specific bronchitis microorganism, a fact demonstrated by the continuing pathological basis of the term: inflammation (*-itis*) of the bronchi. Microbial involvement can be both primary and secondary, although chest physicians rarely see the disease as communicable. Rather they stress predisposing factors such as prior disease, smoking, allergy, immune deficiencies, emphysema, tuberculosis, air pollution and adverse weather. The views of late Victorian doctors were quite similar. They also emphasised predisposing factors. In their case, these were age, constitution, climate, alcoholism and blood diseases, and exciting causes such as cold, vapours, dust and other diseases, especially colds and influenza. Again, in no sense was bronchitis seen as communicable. Thus it seems to us untenable to do as Mooney does in his Table 1, and place pneumonia and bronchitis, along with asthma(!) as infectious diseases.[17]

While we are on the lungs, we may restate our historical points about respiratory tuberculosis. First, for most of the nineteenth century, the disease was understood and experienced by contemporaries as an inherited or constitutional disease. The move towards accepting some degree of communicability only began in the 1880s, and still in terms of some type of predisposition of an inherited or acquired weakness or vulnerability. It took many years for pulmonary tuberculosis to be added to the list of diseases covered by the Infectious Diseases Acts in Britain. Second, the evidence from tuberculin skin tests in the 1890s was that the *Tubercle bacillus* had infected over 90 per cent of urban populations in Britain.[18]

However, only around one in ten of those infected would develop respiratory tuberculosis. This medical finding confirmed the widely held view among doctors and the public

[16]Worboys 2000, pp. 278–86.
[17]Mooney 2007, p. 600.
[18]Worboys 2000, p. 232.

that the development of pulmonary tuberculosis depended on a complex interaction between seed and soil. Even when the role of the microorganism was accepted, poor diet, other illnesses, occupational conditions, damp, cold and nervous tendencies, among many factors, were seen as necessary to turn infection into disease. Similarly, in current medicine there is a distinction between the communication of the *Tubercle bacillus* and the development of the disease. Is it not problematic to use the same aggregate category for, say, cholera and tuberculosis, two diseases for which the term 'infection' has meant very different things over time?

In conclusion, we wish to highlight two fundamental differences between our critics and ourselves. First, they take an expansive view of the category of infectious diseases as all diseases in which microorganisms are today understood to have a causative role. Our approach is different in three senses. It focuses on diseases that are directly and indirectly communicable. It recognises that communication of a pathogenic microorganism does not always lead to disease. And it acknowledges that microorganisms that are present in the body without causing disease can become pathogenic when conditions in the body change, as in secondary pneumonia.

Our more complex, ecological view of infectious diseases seems to us more in harmony with medical and public views of disease in the nineteenth century and, interestingly, with the most recent medical and epidemiological work. Second, we regard the practice of using modern disease categories to analyse sources that were constructed using radically different notions of disease to be flawed. This is fine so long as such histories seek to serve and inform the present, as Thomas McKeown and many others used to attempt to do. But if history is supposed to make sense of the past, we feel that historically sensitive categories cannot be avoided, even if they complicate the job for historians. This aspect of the discussion is obviously part of a larger, historiographical debate about historical categories and master narratives.

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