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Infection, contagion and causality in Colonial Britain: the 1889-90 influenza pandemic and the *British Medical Journal*

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SUMMARY

The influenza pandemic of 1889 was the first truly global flu outbreak in scope. Characterised by high morbidity and low mortality, it spread rapidly across Europe and the rest of the world along trading routes. It reached mainland Britain in December 1889. The responses of medical practitioners in Britain and the British colonies to the pandemic were heavily featured in the British Medical Journal and reveal a confusing picture around causality, contagion and infection. Cases from the colonies (Cape Town, India, Australia, Samoan Islands, Hong Kong) as presented in the journal are explored in an attempt to reconstruct the mainstream medical belief of the time. The evidence sadly shows a lack of confidence in contagionism, almost complete absence of monocausalism and a vague picture of the epidemic constitution. Original case studies from colonial medical officers as well as editorials triggered a debate in the pages of the BMJ. In this context, the journal succeeded in playing a key role in recording the first thoroughly documented attack of influenza. In a world that was only learning to be interconnected, the BMJ became the point of reference for the British medical establishment, which ranged from London to Scotland and from Africa and India to Oceania.

Keywords: causality, epidemics, history of medicine, infection, human, influenza pandemic.

INTRODUCTION

The influenza pandemic of 1889 (usually studied along with its successive waves between 1890 and 1892) is the first flu outbreak that can be demonstrated to have been truly global in scope (Figure 1). It followed major socio-economic changes of the mid-19th century in Europe and parts of North America and was far better documented than any of its predecessors [1].
Originating in Russia, the pandemic spread rapidly across Europe and reached mainland Britain in December 1889. By February 1890 cases of infection were recorded across the country [1]. The first wave in early 1890 was relatively mild; although morbidity was high, with 20-30% of the population affected, mortality remained low [2]. According to modern research, the pandemic of 1889-90 had case fatality rate that ranged between 0.1% and 0.28% and was most possibly caused by an H3N8 virus [3]. Yet, even in this relatively benign early arrival of the pandemic, a clear pattern of transmission was evident in Britain, with the importance of urban hierarchy and of accessibility for local diffusion rate being illustrated.
This pattern of transmission, which moved from large cities to smaller ones before eventually arriving in rural towns and villages was evident to contemporary observers. It was also noticeable

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that the pandemic similarly seemed to spread from Britain to its colonies with a regular periodicity. Indeed, influenza had reached the British African colonies by the end of January 1890, the Australian in March and the Asian later in the same year. A second wave attacked Scotland and rural England in January 1891 while a third wave spread across South Australia in September and reached northern England and London in November 1891 [1].

In this paper, response of medical actors in Britain and the British colonies to the influenza pandemic of 1889-90 will be explored by examining the various theories and case reports that appeared in the *British Medical Journal* in the years immediately following the arrival of pandemic influenza. Thus, based on a number of primary sources and in the context of a specific instance of disease with an uncertain aetiology, the medical professions attempts to negotiate a collective understanding of disease causation will be examined. These pieces, published as part of an ongoing and historically important debate into the cause (s) of infectious diseases, will be used to illuminate both the various theories of pandemic influenzas epidemiology and theories of disease causation in general. In this way this paper contributes to both our understanding of an important episode in 19th century health while at the same time reflecting on broader currents in the historical sociology of medical diagnosis and the role of one of the most widely read medical journals.

Infectious disease, contagion and causality in the 19th century

The 1889 pandemic came as a particular surprise to a medical profession which had not been faced with a major epidemic since 1848. In the intervening years the medical establishment and the nature of medical knowledge had changed considerably. Germ theory, though to some extent it remained a vague and ill-defined cluster of ideas, was becoming more common in medical aetiologies. The number and professionalism of doctors and their journals had grown enormously, and a critical and quantitative atmosphere began to pervade the literature [1]. However, the notion of a unified scientific medical establishment does not accurately characterise this period, and even among similar schools of training notable differences in theory toward infectious disease remained common. Indeed, we must be cautious to distinguish what is called the germ theory in historical accounts from the germ talk that was common throughout the 19th century. Furthermore, the discovery of germs not only changed little in the practice of medicine, it had only a minor impact on the medical understanding of illness [4]. After all, many diseases we

Figure 1 - The Dutch minister Bergansius and Hendrik Pieter Tindal visit an influenza hospital populated with representations of the countries of Europe. Reproduction of a lithograph by J. Braakensiek, 1889. Attribution: Wellcome Library, London (under the Creative Commons Attribution only licence CC BY 4.0).

now recognise as having a bacterial or viral cause were already understood as contagious and had been elaborately described in terms of their causes and consequences.
Nonetheless, in the earlier decades of the century, major transformations had occurred in the medical description of disease and it is not possible to understand the *rhetoric* employed in the *British Medical Journals* discussion of pandemic influenza without paying attention to this.

Cases from the colonies: pandemic influenza in the BMJ

Cape colony

In 1890 Dr William Scholtz reported on the outbreak of influenza in Cape Town, South Africa, then a part of the Cape Colony (between the years 1806 and 1910) [5].
Scholtz started by describing the symptoms and the demographics of the epidemic. Interestingly, he commented on how *much* contagious the disease was characterising it "*feebly contagious, of a largely miasmatic influence, distributing itself more rapidly through the air,* [thus] *personal contagion being a subordinate factor in the diffusion of the disease*".
In another intriguing comment, he attempted an association with travellers coming in to town. As a colonial-service doctor working in Cape Town during the nineteenth century, who no doubt considered the city to be an outpost of civilisation in a savage land, it is no surprise that he suspected travellers.
He then moved on to a brief description of two case reports (a woman and his own experience) aiming to make assumptions on the diseases incubation period. Finally, he described the weather and climatic conditions in the area. Among his conclusions, stands his striking remark that the atmosphere should play an important part in conveying a "*poison vapour, rather than the supposed growth and development of a microbe*".
It is not clear in his brief article whether Dr Scholtz thought of the disease to be caused by the poison vapour or is distinct from it. Moreover, he did not expand on the levels of contagion and if these were inherent to the pathogenic vapours themselves or rooted in the bodies of the patients. Thus, his various assumptions may be attributed to a relative lack of training or different school of thought incorporating some contemporary elements (like the idea of contagion) to the previously well established ideas of atmospheric and astronomical influences.

British Raj

In an April 1890 correspondence in the journal, morbidity from the pandemic in India was described [6]. India was then known as the colony of British Raj (that lasted between the years 1858 and 1947).
The author, who was a military medical officer, recorded the spread of influenza in certain parts of India, especially among British military corpses. He vaguely suggested a contagious nature of influenza as he recognised that it spread more rapidly in the bazaars and crowded cities, as well as among soldiers. Despite not making any further assumptions on causality, he made an interesting point on infection and mortality. He mentioned that if the outbreak had occurred during the cold season "*when there were some 2000 men under canvas*", the mortality would have certainly been higher.

Western Australia

Referring to a November 1891 outbreak of influenza in a Perth asylum, Dr Frank Hay discussed the disease in detail based on his observations from this area of the British colony (1832-1900) of Western Australia [7].
In his article, Dr Hay discussed many different aspects of the outbreak, including symptoms, incubation period, onset, progress, complications, mortality and treatment. Among his interesting contributions were facts that morbidity in females was greater than in males, the existence and impact of a recent history of influenza history among some of the cases, and the observation that symptomatic treatment did not systematically affect the course of the illness.
The medical officer of the James Murrays Royal Asylum did not delve into the means of transmission of the disease, but stated that despite the occasional differences in characteristics and symptoms the cases were undoubtedly due to the same infection. This, though somewhat vague, is an assumption towards monocausalism.
Another intriguing reference in this paper is the mentioning of insanity as attributed to a complication of influenza; a common conception during this pandemic. Dementia, episodes of mania, and imbecility were all recorded here as *sequelae* of influenza.

Samoan Islands

In his January 1892 correspondence, Dr Davies from Samoa discussed the spread of the second wave of the influenza pandemic in Australia, focusing on the Samoan Islands, a British colony between the years 1889-1900 [8]. He described the symptoms, mortality rates and therapeutic approaches among native Samoans who regarded this epidemic as a calamity from a new disease.

However, the most interesting part of his article is the tracking of the spread of the outbreak. He noted that an epidemic resembling that in New South Wales and Victoria appeared first in Sydney, in the harbour of Apia, and spread from there to the South Sea Islands. This course led the author to suggest that the epidemic spread due to the steam communication between Sydney, New Zealand and the Samoan Islands. Influenza also appeared in the *Targan* or *Friendly Islands*, some 400 or 500 miles south of this region, whose residents believed to have been brought by steamers from Sydney or New Zealand.

This comprehensive spreading of the epidemic is what makes Dr Davies believe in the infectious character of influenza. He did not venture a guess on monocausalism though.

Hong Kong

In two articles divided just by a few weeks in early 1892, the influenza outbreak in Hong Kong was briefly described. Hong Kong was a British colony between 1843 and 1941.

In a January 1892 piece discussing the potentiality of Hong Kong being the primary point of emergence of the pandemic, interesting remarks on the means of spreading of the disease were made [9]. Dr Atkinson from the Government Civil Hospital in Hong Kong discussed the influenza morbidity in the city between 1888 and 1890 and noted discrepancies on previous accounts. He especially criticised earlier authors who had not made clear how the spread of the disease occurred for in his own belief it could not had originated in Hong Kong for two reasons: "*the north-east monsoon prevailing at this period of the year* [sets] *atmospheric contagion out of the question;* [and,] *further, there are no direct lines of communication that would account for the transmission of contagion by human intercourse*". Once again, quite intriguingly, a doctor integrated two types of transmission; human to human and atmospheric.

In a February 1892 correspondence, the respiratory nature of the disease was highlighted [10]. Interestingly, this short article also attempted to criticise views of the influenza outbreak originating in China, but the authors primary argument for that is that the "*Chinese have no name for the disease*". In this context, the British physician correspondent distinguished, in fact, himself for being aware of the disease having previously followed training in *European* medicine.

DISCUSSION

By 1889, Western medical opinion was being won over, although not completely, to the idea that microorganisms (bacteria, or germs) caused diseases. So the 1889-90 pandemic stimulated a search for its causative organism. An eminent German bacteriologist, Richard Friedrich Pfeiffer, claimed to have discovered such a bacillus in 1890, but his subsequent efforts to demonstrate the necessary connection between his bacillus and actual cases of influenza were not entirely convincing. In fact, for many the cause of influenza remained unknown, although there seems to have been wide agreement that some microorganism must be responsible. It was also generally agreed that influenza was very contagious. Its spread along routes of human traffic clearly argued that infected people carried it, and passed it to others through the air. The relatively mild character of its symptoms usually did not prevent people from travelling [11].

However, contagion and causality are two different issues. Initially, while the epidemic was generally called *influenza*, neither public health professionals relying on epidemiological knowledge nor medical practitioners relying on clinical knowledge could agree on its actual nature. Disputes over influenzas epidemiology though, as well as efforts to reach decisions on its clinical characteristics, were also rooted in competing medical epistemologies. A priority for public health professionals was to explain how influenza had spread so rapidly and appeared so suddenly. At first, disagreement reigned. Some observers invoked theories based on the original meaning of *influenza*, which presupposed an external *influence* that conspired to excite an epidemic. The notion of an *epidemic constitution*, was widely used to ar-

gue that the epidemic was the product of one or a number of changes in temperature, moisture, air pressure, ozone levels and the nature and density of fogs. A long history of associating influenza with the weather had wide appeal because it resonated with popular perceptions of its apparent affinity for colder and damper months of the year [12].

All of the above are evident one way or another along the lines of the presented primary articles. Though, on the one hand, it is clearly established that influenza is a contagious disease, none of the authors is firm on monocausalism. This tentative stance permeates the whole 19th century. While certain illnesses were generally conceded to be transmissible ever since the first decades of the century, doubts were voiced especially as cholera in the 1830s did not appear to spread solely by means of personal contact. During the heyday of an environmentalist stance contagionism was seen as an outmoded, old fashioned and conservative approach to disease that denied its obvious causes in filth and squalor. However, far from vanishing, contagionism celebrated a triumphant return with the bacteriological revolution at the end of the century when Louis Pasteur (1822-1895), Robert Koch (1843-1910) and others vindicated the insight that disease, caused by specific microorganisms, was often transmitted among humans and that, whatever the effects of predisposing factors, certain illnesses spread independently of social and local circumstances. A strictly binary view of either aetiology would, however, be a distortion. The basic building blocks of epidemiological theory (local factors, individual predisposition, contagion) were multiply and mutually permeable. Miasmas could be regarded as localist, contagionist or both, seen as emanations produced by environmental causes, other times as the vehicle by which disease spread from one place to another [13].

As already shown, the *British Medical Journal* had a central role in this dialogue that seemed to reach new levels during the pandemic. In January 1890, an editorial argued that: "*like other epidemic diseases, influenza is spread by a contagium, and must be due to a living organism, a microbe*"; but there remained dispute over whether the stunning diffusion of influenza was the product of *microbes carried in the air*, microbes spread from one person to person or a combination of the two [14]. Contemporary author, Henry Franklin Parsons, remarked that the absence of records of a major epidemic of influenza for 43 years before 1889 suggested that a change occurred in that year in the epidemiology of the disease and was able to map influenzas spread across the world [2,14]. His report was well received and the *British Medical Journal* noted that although "*the theory that influenza is mainly if not entirely spread by contagion is no new one ...* [it] *had needed to be born again*" [15].

This was not the closure, however, as during the course of the pandemic and its successive waves a number of other different views had appeared within the pages of the *British Medical Journal*. For example, in his April 1890 publication, Dr William Tibbles from Melton Mowbray, England, made the assumption that influenza is due to an unknown microbe, however, he denied human transmission and argued that soil and atmosphere play a key role in the germination [16]. Thus, while mentioning a microorganism as active in the production of disease, he was careful to distinguish his position from strict monocausalism. In the same year and journal, Scottish physician Dr John Haddon described influenza cases that he treated to state his belief that the current epidemic was an infectious disease [17]. To his eyes, influenza, a disease that *depends upon a bacillus*, was introduced to communities and families via other members. In another example from 1891, Dr Peter Eade from Norfolk, though uncertain on the exact microbe, considered that the influenza "*bacillus is present awaiting demonstration, inasmuch as a contagious and multiplying germ must be a living one and have a material presence*" [18]. Regarding the communicability of the disease in particular, he accepted recent reports and dismissed any further discussions on its *contagiousness* or *infectiousness*.

The above serve to mostly contradict the ideas proposed in most of the papers coming from colonial settings. These striking and repeating differences in the approaches to influenza in colonial and mainland Britain go further than simple terms of personal observation and insight. The impact of various other factors, like clinical experience, peer-to-peer relationships, field training, resources, has to be taken into account. Indeed, over the next few years, in the reverberation of the pandemic, colonial medical policy underwent some serious changes. By 1900 the new specialism of tropical medicine rose to prominent levels.

Perhaps identifying discrepancies like the ones noted in the herein presented articles, the London School of Tropical Medicine was founded with its principal motive the desire of the Colonial Office to provide for the better training of Colonial Medical Officers and the expansion of their service. As long as colonial medical men were trained and serviced the needs of expatriate communities, tropical medical policies could be deemed a success. In addition, the new Liverpool School of Tropical Medicine was also promoted as an investment in increased colonial trade. Thus, with the memory of the global pandemic still fresh, in declaring that it was improving general public health, colonial medical policy (with its alternatives) targeted at the stabilisation of the British policy towards its colonial empire and the economic benefits gained from the colonies themselves [19].

Figure 2 - An annoyed patient surrounded by dancing politicians and doctors (corruption), skeletons playing music (commonly connected to the plague), and prostitutes dressed in clothes referring to common antipyretics (frequently associated with syphilis). Wood engraving by Pépin (E. Guillaumin), 1889. Attribution: Wellcome Library, London (under the Creative Commons Attribution only licence CC BY 4.0).

The various pieces of the puzzle came gradually in to place in the years that followed as the 1889 influenza pandemic in Britain was apparently the impetus to drive towards the mapping of a new influenza. Professional agreement on its nature and identity involved aligning different forms of knowledge produced in the field (public health), in the clinic (metropolitan hospitals) and in the laboratory (bacteriology). While there existed different *influenzas* in the wake of the 1889-90 pandemic, the problems, interests and practices of public health professionals, clinicians and laboratory pathologists were made increasingly commensurable, such that by the early 1900s influenza was generally characterised as a specific infectious disease. The late 19th century definition of influenza was based on what was later shown to be the wrong microbe. Rather than a bacillus, its primary causative agent was now identified as a virus. However, both the bacillus and bacteriology had a crucial part in the realignment of clinical and epidemiological knowledge of the disease [12].

Until then, in the 1890s the nosological identity of influenza was far from fixed (Figure 2). Instead, the disease was regarded as a protean somatopsychic infection that could even present with very similar symptoms to neurasthenics: namely, anxiety accompanied by insomnia, fatigue, and depression [20]. People also had to deal with an epidemic of universal scope and appreciate the role of industrialisation in its spread. As is mentioned in some of the primary sources, distant connections through rail networks and steamships were dramatically recognised to have a role in the diffusion of the disease [11]. Revolutionary developments in transportation allowed influenza to move from city to city and into the countryside in unprecedented speeds. Better roads and canals played a role, but the crucial innovation was the railroad network that covered Europe from the Urals to London and extended from coast to coast in North America. Equally important for transmission to port cities and between continents was the enormous increase in the volume and speed of maritime commerce [1].

Therefore, the world in general, and the British

mainland and colonial population in particular, came up against a disease with partly unknown nosology, on the transmission of which many different opinions kept being formulated, that was spreading in unrecognisable speeds within a newly industrialised environment. Even though late 19th century imperialism was forging much stronger links between Europe and the other continents, the colonies still seemed to operate under different variable statuses. In a world that was only learning to be interconnected, the *British Medical Journal* became the point of reference for at least the British medical establishment that ranged from London to Scotland and from Africa and India to Oceania. Thereafter, it is not unexpected that it served as a tribune to the various different representatives of the immature medical knowledge, including among its pages all the variable perspectives on infection, contagion and causality. The 1889-1890 influenza outbreak, the first one that was clearly worldwide in scope, was the perfect means to encompass all the emerging and revisited aspects, and in consequence became, not unexpectedly, the first thoroughly documented attack of influenza.

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