Homeopathy and the "Bacteriological Revolution" 1880–1895

The Reception of Germ Theory and Bacteriology in 19th-century England and Germany

Carol-Ann Galego

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Introduction

The present study considers the responses of homeopaths in Germany and England to developments in bacteriology between 1880 and 1895, fifteen fruitful years of the "bacteriological revolution" that overlap with the fifth cholera epidemic of the nineteenth century, which ranged from 1881 to 1896.[1] During these formative years, the convergence of bacteriologists' isolation and cultivation of microbes with medical efforts to quell the ravages of cholera gave rise to the now predominant understanding of infectious disease as an invasion of pathogens. At the time, however, such an antagonistic response to the threat of infectious disease was anything but unanimous; alternative understandings and approaches continued to abound and their excavation sheds critical light not only on historical approaches to infectious disease but also on their contemporary counterparts.

But, first, a little historical context is in order. Of all the infectious diseases that challenged nineteenth-century Europe, cholera, albeit not the deadliest, had a unique capacity to elicit fear and haunt the public imagination as a foreign invader.[2] For centuries, the disease had been confined to a relatively small region within India, its dreadful symptoms recorded extensively by British physicians stationed in the subcontinent but otherwise unknown to most Europeans. Then, in 1817, a particularly virulent form of the disease began to spread westward, advancing slowly but relentlessly.[3] Although the European medical community was generally at a loss when it came to treating cholera or containing its spread, by the early 1830s its medical cartography was advanced enough that "both governments and public[-]health officials could literally

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[1] Dhiman Barua and William B. Greenough III, eds., Cholera (New York: Plenum Medical Book Company, 1992), 14; Paul Blake, "Historical Perspectives on Pandemic Cholera," in Vibrio cholerae and Cholera: Molecular to Global Perspectives, ed. Kaye Wachsmuth, Paul A Blake, and Ørjan Olsvik (Washington: ASM Press, 1994), 293.

[2] Michael Biddis and Frederick Cartwright, Disease and History (London: Rupert Hart-Davis, 1972), 114; Richard Evans, Death in Hamburg: Society and Politics in the Cholera Years; 1830–1910 (Oxford: Clarendon Press, 1987), 230; Pamela Gilbert, Cholera and Nation: Doctoring the Social Body in Victorian England (Albany: State University of New York Press, 2008), 2; Vincent Knapp, Disease and Its Impact on Modern European History (Wales: The Edwin Mellen Press, Ltd., 1989), 134.

[3] Biddis and Cartwright, Disease and History, 114; Knapp, Disease and Its Impact, 123; Richard Ross, Contagion and Prussia 1831: The Cholera Epidemic and the Threat of the Polish Uprising (Jefferson: McFarland Publishing, 2015), 4.

watch cholera gaining ground from one country to another."[4] Ill equipped to handle its arrival, they could do little more than follow its gradual, seemingly inevitable, approach to their communities, as if watching a slow-motion train crash.

The disease first entered the European continent in August of 1829, by way of the Russian city of Orenburg on the Ural River, reaching Moscow in the autumn of 1830 and St. Petersburg in June of 1831.[5] By the summer of 1831, most of the major cities and ports of central and Eastern Europe had been completely overwhelmed by the disease. While British authorities initially remained hopeful that the island would remain unaffected, their efforts to enforce quarantine did not suffice:[6] On October 12, danger became acute when cholera appeared in Hamburg, a port of regular communication with the British Isles.[7] Later that month, the first English cases appeared in Sunderland; by winter, all of the British Isles had also been affected.[8]

Before it eventually exhausted itself in most areas of the continent by the end of the nineteenth century, five pandemics had completely ravaged Europe, leaving behind approximately ten million casualties in its wake: "Not since plague had a single major European disease produced such high mortality in such a short period of time."[9] Although less fatal than its medieval predecessor, cholera was nevertheless an extremely frightening, fast-acting disease. In contrast to the agonizing anticipation with which the disease could be tracked as it slowly stretched across the continent, when cholera did finally strike, it took tens of thousands of lives almost instantly.[10] Indeed, one of the most terrifying aspects of the illness was its sudden onset and rapid development.[11] Persistent vomiting and diarrhea quickly evacuate the bowels and leave the body completely dehydrated. In turn, the lack of fluids brings on terrible cramps of the limbs and abdominal muscles, and the body's efforts to empty an already empty stomach result in continual retching and hiccupping.[12] Needless to say, death from cholera was anything but beautiful.[13] Richard Evans writes:

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[4] Knapp, Disease and Its Impact, 121.

[5] Knapp, Disease and Its Impact, 123–124.

[6] Knapp, Disease and Its Impact, 125.

[7] Biddis and Cartwright, Disease and History, 116.

[8] Biddis and Cartwright, Disease and History, 114; Stanley Warren, "Preface," in Cholera and Conflict (Leeds: Medical Museum Publishing, 2009), i.

[9] Knapp, Disease and Its Impact, 132.

[10] Knapp, Disease and Its Impact, 122.

[11] Biddis and Cartwright, Disease and History, 114.

[12] Biddis and Cartwright, Disease and History, 114; Knapp, Disease and Its Impact, 125.

[13] Evans, Death in Hamburg, 228.

> The blue, "corrugated" appearance of the skin and the dull, sunken eyes of sufferers transformed their bodies from those of recognizable people, friends, family, relatives, into the living dead within a matter of hours. Worse still, the massive loss of body fluids, the constant vomiting and defecating of vast quantities of liquid excreta, were horrifying and deeply disgusting in an age which, more than any other, sought to conceal bodily functions from itself.[14]

Owing to its quick onset, most cases went untreated, and one out of every two cases resulted in death within a few days or sometimes even hours.[15] According to Vincent Knapp, a good deal of the fatalism expressed by most Europeans in the face of cholera "was the result of the medical profession's inability to stop the ever-increasing vomiting and purging that characterized this disease and which ultimately led to the death of so many patients."[16] And when medical treatment was available, it generally did more harm than good: Most notably, doctors bled their patients,[17] and prescribed opium and calomel.[18] Compounding a general distrust of the medical community, the sudden onset of cholera resembled arsenic poisoning; in some areas, rumours began to spread that doctors were poisoning victims on behalf of the rich in European capitals.[19]

In addition to the varied opinions and overall lack of confidence surrounding the treatment of cholera, its etiology was also highly contested. Later identified as a waterborne disease,[20] transmitted by a "rather fickle microorganism

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[14] Evans, Death in Hamburg, 229.

[15] While this is a frighteningly high mortality rate, in Disease and Its Impact (135), Knapp reminds us: "When one considers the number of nutrients being lost to both vomiting and purging, what is truly remarkable is that up to half of those who were afflicted actually recovered on their own."

[16] Knapp, Disease and Its Impact, 127–128.

[17] For a detailed account of the practice of bleeding cholera patients, see Wilhelm Ameke, History of Homœopathy: Its Origins; Its Conflicts, trans. Alfred Drysdale, ed. R. E. Dudgeon (London: E. Gould and Son, 1885), 235–245.

[18] Karl-Friedrich Scheible, Hahnemann und die Cholera (Heidelberg: Karl F. Haug Verlag, 1994), 25.

[19] Ross, Contagion and Prussia, 11–12.

[20] According to Alfred Evans, "Even today there is still controversy over the major routes of spread of cholera. In some settings and for some strains transmission by water seems most important, whereas for other strains personal contact and carrier states are more likely means of spread." "Pettenkofer Revisited: The Life and Contributions of Max von Pettenkofer (1818–1901)," Yale Journal of Biology and Medicine 46 (1973): 174, https://pdfs.semanticscholar.org/1b98/75eef0dc838c1ad90cc4b17bb946430430d4.pdf.

[that] is almost totally dependent on environmental conditions,"[21] cholera did not adhere to the familiar understanding of contagious diseases, which were thought to spread by touch, contact with contaminated clothing and goods, or, in exceptional cases, by breathing infected air. It was widely acknowledged that the spread of the disease followed shipping routes, though not all localities and individuals were affected, and on numerous occasions quarantine proved ineffective. In view of this complex picture, physicians and hygienists tried to reconcile observations of cholera with existing understandings of infectious disease.

Before cholera even arrived in Europe, medical opinion was divided on the causes of this devastating disease. In their most polarized forms, the two competing theories adhered to different causal factors: the environment versus the germ. On the one hand, environmentalists (also referred to as miasmists, localists, and anticontagionists) tended to the local conditions, such as unsanitary living quarters, dirty water, and malnutrition, which they believed negatively impacted one's predisposition to disease. They posited that cholera was caused by miasma, that is, by particles of rotten material that polluted the air and infected all who inhaled them. Environmentalists accordingly focused on broad social reforms and sanitary measures such as draining stagnant water, separating humans from their excrement, building more spacious housing, and providing clean drinking water, healthy food, and warm clothing. On the other hand, contagionists insisted that the disease was spread by contagious material and accordingly sought to interrupt chains of transmission through disinfection and quarantine.

Even during the fifth epidemic, no universally convincing evidence supported either of these two conflicting theories. Adding to the controversy, when cholera entered Egypt in the early 1880s, it was "the first outbreak to occur after the completion of the [Suez] canal, and the cause of that epidemic was alleged by some of the European powers to be English ships coming into the Mediterranean Sea from India via the canal,"[22] prompting Britain, France, and the German territories to dispatch research commissions to study the disease,[23] thereby providing "an opportunity to turn the new tools of bacteriology to account."[24] At the time, Louis Pasteur and Robert Koch were still enjoying recognition for

[21] Knapp, Disease and Its Impact, 128.

[22] Mariko Ogawa, "Uneasy Bedfellows: Science and Politics in Refutation of Koch's Bacterial Theory of Cholera," Bulletin of the History of Medicine 74, no. 4 (Winter 2000): 672.

[23] Ogawa, "Uneasy Bedfellows," 673.

[24] W. F. Bynum, "The Rise of Science in Medicine: 1850–1913," in The Western Medical Tradition, vol. 2., 1800–2000, ed. W. F. Bynum et al., (Cambridge: Cambridge University Press, 2006), 129.

their recent achievements, namely Pasteur's development of an anthrax vaccine in 1881 and Koch's isolation of an organism associated with tuberculosis in 1882. But the expedition was more challenging than anticipated. The French aborted the mission following the death of one of their team members, and Koch believed that he had identified a pathogen that was found consistently in cholera victims, but he needed to pursue his investigations further in India. In late 1883, he identified a curved bacterium in the stool of sufferers and, in early 1884, published his observations of the "comma" bacillus, named after its shape but otherwise known as the cholera vibrio.

His announcement was received in Germany with much more fanfare than in the rest of Europe and, altogether, it took years and in some cases decades for his proposed etiology of both cholera and tuberculosis[25] to gain general acceptance among practitioners and public-health officials. At the time of this discovery, Koch was still in the process of formulating his famous postulates for establishing that a particular pathogen is the cause of a disease,[26] and his inability to infect an animal with the cholera vibrio meant that he had failed to meet his own requirements for establishing claims of infectious causality,[27] which "became the main bone of contention between him and his critics."[28] More generally, it was difficult for physicians to abandon their previous understanding of the disease.[29] In any case, the debate surrounding the causation of cholera certainly did not subside after Koch isolated the vibrio,[30] and in some cases it even intensified.

In the fall of 1884, the British secretary of state sent Emmanuel Klein and Heneage Gibbes to India "to ascertain the nature, origin, and propagation of cholera, the microscopic organisms connected with it, and their relations – causal or otherwise – to the disease."[31] Before their departure in December of

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[25] Bynum, "The Rise of Science in Medicine," 129–130.

[26] Ogawa, "Uneasy Bedfellows," 673.

[27] Bruno Atalic, "1885 Cholera Controversy: Klein versus Koch," Journal of Medical Ethics: Medical Humanities 36 (2010): 43; and Ogawa, "Uneasy Bedfellows," 673. Christoph Gradmann notes that "when some authors realized that Koch's own methodology was not always in line with his postulates, they devised the explanation that Koch had modified his postulates later in his career to accommodate the concept of carrier-state epidemiology, of which he was the inventor." "A Spirit of Scientific Rigour: Koch's Postulates in Twentieth-Century Medicine," Microbes and Infection 16 (2014): 891.

[28] Ogawa, "Uneasy Bedfellows," 685.

[29] Bynum, "The Rise of Science in Medicine," 130.

[30] Ross, Contagion and Prussia, 256.

[31] E. Klein and Heneage Gibbes, "An Inquiry by E. Klein, MD, F. R. S., and Heneage Gibbes, MD, into the Etiology of Asiatic Cholera," in Cholera: Inquiry

that same year, the two-man research team purported to have found sufficient grounds for systematically criticizing Koch's observations and, more critically, his conclusions. In particular, they noted the lack of direct contagion, Koch's failed animal experiments, and the observation that many villagers who consumed water contaminated with the feces of cholera victims did not contract the disease.[32] In June 1885, the British secretary of state for India convened a committee of thirteen British medical celebrities to assess to what extent Klein and Gibbes' report, "An Inquiry into the Etiology of Asiatic Cholera," supported or refuted the evidence for the germ theory presented by Koch. After three meetings, the committee published "The Official Refutation of Dr. Robert Koch's Theory of Cholera and Commas" in the *Quarterly Journal of Microscopical Science*.[33] As indicated by the title, the committee concluded that "the evidence that had been adduced by Klein and Gibbes seemed to warrant the inference that the comma-shaped bacillus was not the cause of cholera."[34]

While this verdict undoubtedly gave Koch's critics more fodder, circumstances outside of England were not generally favourable to this position. At the Second Cholera Conference held in Berlin in May 1885, for example, "Koch refuted the localist views of his archrival,"[35] Max von Pettenkofer, a once-prominent Bavarian hygienist and one of the most well-known representatives of "contingent contagionism" in the German-speaking world.[36] That said, it would be inaccurate to present the controversy surrounding the etiology of cholera as one between England and Germany. Even within the German-speaking world Pettenkofer's views were prone to die hard.

As early as 1869 – fourteen years before the cholera vibrio was identified by Koch – Pettenkofer acknowledged the existence of an infectious element in the spread of the disease. Unlike Koch, however, he insisted that more than exposure to the pathogen was required to produce the disease. In addition to the specific germ, he also emphasized the importance of local, seasonal, and individual conditions. More specifically, he posited that the cholera germ coming from India, which he referred to as the *x* factor, could not by itself produce cholera. In order to become infective, he argued, it needed to interact with *y*, a substrate found in the soil under suitable conditions, and, in doing so, would

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by Doctors Klein and Gibbes, and Transactions of a Committee Convened by the Secretary of State for India in Council (London: 1885): 12, quoted in Ogawa, "Uneasy Bedfellows," 701.

[32] Ogawa, "Uneasy Bedfellows," 697.

[33] Ogawa, "Uneasy Bedfellows," 701.

[34] Ogawa, "Uneasy Bedfellows," 700.

[35] Ogawa, "Uneasy Bedfellows," 697.

[36] Evans, Death in Hamburg, 238.

produce *z*, "the real cholera poison."[37] According to Pettenkofer's "soil theory," it was impossible for Koch's cholera vibrio alone to infect an individual, *even if the individual was susceptible.*

The strength of Pettenkofer's conviction was boldly displayed in 1892, when the cholera epidemic reached its apex in Hamburg, the "last and most devastating outbreak in Germany,"[38] where it took "a frightening toll on those living in the port city of Hamburg (...) [and] wiped out nearly 16,000 people in a matter of just a few short weeks."[39] Called in to assess the situation, Koch traced the epidemic back to an infected water source and pointed compellingly to the epidemiological evidence that Altona, which received its water from another source, had remained unaffected. Pettenkofer, however, remained unconvinced. He denied Koch's argument just as he had rejected John Snow's theory of cholera's being waterborne, which had been widely publicized in London in the 1850s despite having received little acceptance.[40] Pettenkofer was so confident in his position that on October 7 of the same year, at the age of seventy-four, he neutralized his stomach acid with sodium bicarbonate[41] and then, in the presence of witnesses, swallowed one cubic centimetre of a culture of cholera vibrios that he had obtained from Professor Gaffky. He reportedly suffered mild diarrhea and a "pure culture" of vibrios could be found in his stools, but he remained otherwise unscathed and convinced that his self-experiment had effectively disproved Koch's contention that the cholera vibrio was the necessary and sufficient cause of cholera.[42]

As indicated by this brief history of cholera and its controversies, homeopaths in England and Germany had a variety of "official" opinions to help them

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[37] Evans, "Pettenkofer Revisited," 170. Norman Howard-Jones observes that by 1892, Pettenkofer's "trinity had undergone a curious metamorphosis. Koch's comma bacillus was still x, but y was "temporo-spatial disposition" while z was "individual disposition." "Gelsenkirchen Typhoid Epidemic of 1901, Robert Koch, and the Dead Hand of Max von Pettenkofer," British Medical Journal 1 (1973): 104.

[38] Ross, Contagion and Prussia, 3.

[39] Knapp, Disease and Its Impact, 134.

[40] Ogawa, "Uneasy Bedfellows," 681.

[41] This was to address the observation, acknowledged by Koch, that cholera bacilli cannot survive the acidity of the stomach.

[42] For further details of Pettenkofer's "soil theory" and his self-experiment, see Evans, "Pettenkofer Revisited," 170–172; Evans, Death in Hamburg, 237–238, 493–498; Howard-Jones, "Dead Hand of Max von Pettenkofer," 103–104; and Alfredo Morabia, "Epidemiologic Interactions, Complexity, and the Lonesome Death of Max von Pettenkofer," American Journal of Epidemiology 1, no. 11 (2007): 1234–1236.