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Curing Cholera: Pathogens, Places and Poverty in South Asia

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In this paper I will seek to provide a new understanding of endemicity of disease in India. Through a study of cholera research in the twentieth century I will argue that disease and its endemicity has to be understood in biological factors as well as within a wider social and economic context. I will discuss the medical efforts at locating the causality of cholera from the nineteenth century in Indian climate, water bodies and human anatomy to show that cholera is no more a biological phenomena than water is an ecological or environmental problem. Both are essentially political and economic questions.

Why cholera? Cholera remains one of the most common and yet complex disease in South Asia. As Mark Harrison described, ‘No disease was more, important, and no disease so little understood, as the “epidemic cholera” ’ (Harrison 1994: 99). Cholera was clearly identified geographically and climatically to India. It was called ‘Asiatic Cholera’ as the Gangetic basin was believed to be the ‘home’ of the disease. In doing so, the focus was on the climatic and environmental conditions of this region in which cholera was believed to thrive (Jameson 1820; Corbyn 1832). At the same time, cholera enjoys a strong European history, as it affected countries like Germany, Britain and Russia in the nineteenth century. It was believed that in 1817 this Asian pestilence moved out of its homeland and spread to Europe and America in a pandemic form causing widespread death and devastation. It had a profound effect on society on both sides of the Atlantic. The epidemics of the 1830s, 1840s and 1860s have been identified as causing popular unrest, accentuating social conflict, and providing a catalyst for municipal reform and the development of public health. By the middle of the nineteenth century cholera had become worldwide, the most dreaded scourge, often referred to as the ‘cholera years’ (Rosenberg 1962). This has given rise to vivid cultural and social imagery around it in Europe and it received great medical attention throughout the nineteenth century. As a result we know a lot about the cultural, epidemiological and ecological history of cholera, but we know very little about its bacteriological history beyond Koch and Haffkine. Moreover, medical and historical interest in cholera, so dominant in the nineteenth century, seemed to suddenly fade away in the twentieth, which was dominated by malaria.

CHOLERA AND THE POLITICS OF CAUSALITY

Cholera enjoys a unique etiological legacy. Its causality was explored simultaneously both inside and outside the laboratory. Historically cholera germs had been located in two different sites, the environment (water) and the human intestine. Throughout the nineteenth century, European medical men and scientists devoted considerable time and energy to study the disease. They conjectured variously, that it was caused by the effect of climate on the soil, by providing the right condition of germination of the cholera ‘seed’, by contagion or by transmission by water. Various theories about the causality of cholera were thrown up, linking cholera outbreaks to lunar influences and atmospheric changes. In 1849, the London doctor John Snow showed that cholera was a waterborne infection (Snow 1849). This gave rise to traditions of identifying water as the carrier of disease, which retained its relevance even in the post-bacteriology era (Hamlin 1991). This identification was ideological as well. Cholera outbreaks were critical in English efforts at providing purified water for the general public from the mid-nineteenth century. The water-borne theory also appeared to locate the

disease in India. In 1893 British Medical Journal reported that they had received a 'spirited defence' of the water borne theory from India, particularly from Ernest Roberts, the late acting Deputy Sanitary Commissioner North-West Provinces and Awadh. They noted that 'Reliance is no longer placed on theories of "air currents" and "pandemic waves" to explain the prevalence of cholera' (*BMJ* 1893: 600-1). By the end of the nineteenth century, when the waterborne theory was firmly established in India, these earlier explanations were ridiculed as 'relics' of the middle ages which 'attributed epidemics to comets, meteors, earthquakes, volcanoes, or sidereal changes ...' (Moore 1885: 270-3). However, this apparent shift from air to water did not change the main focus of British understanding of occurrences of cholera in India. British medical men continued to see cholera as essentially a disease of the 'locality', endemic to the Gangetic basin (Bryden 1874).

Ideas of environmental endemicity of cholera became popular in India and it expanded theoretically and geographically. H.W. Bellew, an Indian born British medical officer asserted that 'a state of endemicity' existed, not only in Bengal and the adjacent areas, but also in the interfluvial tracts of the Godavari, Krishna and Cauvery rivers in Madras, in the southern coastal districts of the then Bombay Presidency, in Awadh and the southern Gangetic districts of the north-western provinces, and even in parts of the Punjab, in other words any region 'characterized by a low-lying alluvial soil, which is more or less supersaturated with ground water in a state of stagnation ... and which is subject to periodical inundations or water-logging by the seasonal floodings of the great rivers'. According to him these conditions of the ground coupled with the 'moist and hot tropical climate' provided the ideal breeding grounds of cholera (Bellew 1884). Therefore, the whole of India appeared to be endemic to cholera.

But the opposing point of view about the causality of cholera was gaining ground as well. The German bacteriologist Robert Koch discovered the *cholera morbus* as the causal bacterial in 1884 (Coleman 1987). The German Cholera Commission headed by Koch arrived in Calcutta in 1883 and collected evidences and found vast quantities of a particular bacterium in the intestines of the people suffering from the disease. Koch's discovery of the cholera bacillus strengthened the contagionists voices and demands for quarantine. Armed with Koch's theory of the *cholera morbus*, bacteriologists in India found reasons to question the waterborne theory. Ernst H. Hankin, in Agra found that *cholera vibrio* could not survive in the raw water of Ganges and Jumna rivers for even two hours due to the presence of volatile acid substances (*BMJ* 1895: 312).

Interestingly, the theory of cholera and its habitation in the human biliary tract in India was older than the waterborne theory. India was subjected to intense pathological mapping from the eighteenth century and the centrality of morbid anatomy was more strongly established in India than in Britain itself. James Johnson, James Annesley and William Twining were particularly eminent in the attempt to produce a pathological 'map' of India. The intestine and biliary tract had been seen as the main site of tropical diseases, particularly cholera and fevers. The human body was viewed as a microcosm of its environment, and the social and environmental factors were seen to be reflected in bodily decay. The pattern of decay appeared to manifest itself differently in the bodies of Indians and Europeans, according to their habits, customs and constitutional traits (Harrison 2009: 173-95). James Johnson placed great emphasis on excessive (or sometimes deficient) biliary secretion as a cause of many diseases including hepatitis, dysentery, cholera and certain types of fevers. He found evidence of vitiated bile wherever these disorders occurred, appearing in post-mortem dissections as a mass of black or dark-green fluid. Johnson believed that biliary disorders were endemic to Europeans in India because their bodies were poorly equipped to cope with extreme heat (Harrison 1999).

Koch's assertions were met with strong reaction both in Europe and in India. Following his discovery of the cholera bacterium in 1884; Max Von Pettenkofer (1818-1901) asserted that the composition of the soil and its interaction with groundwater was equally important to the activities of the pathogen. Pettenkofer's belief was that the cholera germ had to transform or 'ferment' under these favourable circumstances before it could become contagious and cause an epidemic. Isolated from these circumstances, the cholera germ could not, in his view, cause disease (Murray 1873: 219-21). Pettenkofer's ideas were in continuity with the nineteenth-century British medical traditions and Pettenkofer was received with great enthusiasm in British India (Isaacs 1998: 281-2).

The British scientific community was particularly keen on refuting Koch's claims. A paper published in the *Quarterly Journal of Microscopical Science* in February 1886, entitled 'The Official Refutation of Dr. Robert Koch's Theory of Cholera and Commas', gave an interesting view of the British response by bringing the relationship between bacteriology and the social, economic, and political situation of the time into sharp focus (*Quarterly Journal of Microscopical Science* 1886). In 1884 in accordance with directives from the India Office, the Indian government set up India's first medical laboratory to officially refute Koch's claims. D.D. Cunningham was appointed director of the laboratory and he was one of the strong believers of 'local causation'. Cunningham claimed that bacilli in the intestine of cholera patients might assume several forms, varying according to temperature, acidity and so on and it undermined Koch's hypothesis. He argued that the comma bacillus was a comparatively feeble organism unable to survive for long outside the body. Thus without really disproving Koch's theory it placed a far greater emphasis on the environment necessary for the generation of cholera. He launched his further critique on contagionists, stressing that the spread of cholera was entirely subjected to 'local' conditions. Cunningham was able to square growing international acceptance of the role of the comma bacilli with the Government of India's insistence on the importance of local factors in the production of cholera.

This was the ideological and political schism when a Russian Jewish scientist, Waldemar Mordecai Haffkine working in the Pasteur institute in Paris entered the scene. Haffkine began to study cholera in October 1890, shortly after his arrival at Roux's laboratory. In 1892 he demonstrated that immunity could be induced in animals by inoculating them with attenuated cholera bacilli. He conducted successful experiments on human subjects, including himself, thus raising the possibility of preventing one of the most dreaded diseases of the nineteenth century. News of Haffkine's work spread quickly and soon came to the notice of Lord Dufferin, a former viceroy of India. Dufferin wrote to the Secretary of State for India requesting that Haffkine be permitted to pursue his studies in what was regarded by many as the 'home' of the disease. Haffkine arrived in Calcutta in 1893. After injecting himself and four Indian doctors, he was able to induce some villagers in the cholera belt of Bengal to come forward for inoculation. However, here he was faced with active hostility from the government medical officers opposed to his ideas and dubious about the efficacy of his inoculation. Medical scepticism was reinforced by racial prejudice and Haffkine's Jewish-Russian origins made him a suspect in the eyes of the British-Indian establishment. He was even denounced by some newspapers as a Russian spy. Haffkine was forced to travel to north India where cholera was not endemic but where he could conduct his inoculations relatively peacefully.

In India Haffkine was faced with another dilemma, 'the question which has always been most difficult to answer was, how I could collect my observations, and, when collected, what possible conclusions could be drawn from them. In a city like Calcutta, with over 800,000 inhabitants, was I to inoculate half of the population for the purpose of comparing inoculated with non-inoculated and of testing my method?' ... 'It was, indeed impossible for

me to inoculate whole cities' (Haffkine 1895: 39). That opportunity came in Calcutta in 1894, when he inoculated 116 of 200 people in a *bustee* or slum. All 116 escaped infection; there were 9 cases, several fatal, among the 84 uninoculated population. The vaccine continued to acquit itself similarly throughout the year that followed and on the tea gardens of Assam, where Haffkine spent the winter and spring of 1894-5. This policy of inoculating labourers had its own problems; the inoculation produced a reaction and fever which made physical labour impossible, and the planters were reluctant to lose the work time. In addition, the labourers formed a highly mobile population, unused to having their movements tracked, which made the collection of results extremely difficult.

He sought therefore for ways to make the best possible demonstration of the effects of his inoculations. He started conducting his experiments in well-defined populations, like prisoners with carefully selected control groups. Haffkine's experiments in Gaya and Darbhanga jails were his most important innovation in preventive medicine. The results were encouraging and after his Darbhanga and Gaya jail experiments, he said, 'Few operations which I made within the walls of a laboratory exceeded in precision the one just described. Its results confirmed some essential conclusions which I had deduced from previous observations; and henceforth I accepted those conclusions confidently as a guidance for my future work' (Haffkine 1913). His cholera vaccine was the first serious attempt at large-scale immunization with a human vaccine (Löwy 1992: 272).

The plans for the future were now clear, as he added, 'The next step was to transfer the operations to the East [Bengal], where their efficacy could be first of all tested in cholera-affected localities and, if ascertained to be favourable, put into practice' (Haffkine 1913: 68-9). But he had to soon leave India due to ill-health leaving his work incomplete and on the eve of his departure he added, 'The complicated problem of introducing the inoculations, in proportion to their protective power, as an adopted prophylactic among the populations of the East, remains practically untouched' (Ibid.: 50). From 1896 outbreak of plague in Bombay, a major trading centre diverted government attention. The question of cholera inoculation and cholera vaccine research were shelved.

After Haffkine, the most important bacteriological research in India on cholera was conducted by Major E.D.W. Greig, Assistant Director of Central Research Institute (CRI), between 1914 and 1916. Greig made an important discovery, actually a confirmation of Hankin's earlier claim. He found that the life of the cholera vibrio outside the human host in natural conditions of India was very short, certainly too short to cause epidemics, thereby negating the climatic theories to a great extent. Greig instead focused on the biliary tract of the human body as the site of cholera infection. Leonard Rogers in his famous work on cholera has rejected the biliary tracts as the site of the cholera infection. Greig found that the cholera vibrio could enter and reside and even remain dormant in the bile for long periods and that was how the chronic cholera carrier was produced. It was the human body which was the 'chronic carrier' of cholera. And that was how people continued to get infected, from human contact. This was Greig's theory of cholera causation, the 'human factor' as opposed to the ecological one:

The death rate of the cholera vibrios outside the human body was extremely high and in a comparatively short time it becomes extinct. On the other hand, scientific research has shown that in certain portions of the body of man, namely the biliary passages, the organisms find conditions suitable in all respects for continuing its life. The bile is an excellent medium for the growth of the cholera vibrio

(Greig 1913-14: 42-6)

He added that earlier the measures for tackling cholera in India were based on the idea that the virus of the disease existed outside the human host, particularly in the water, which was

misguided and had thus failed, so the bacteriologist needed 'concentrate his attack on the human element'. However, in 1916, Greig was withdrawn for wartime vaccine production duties at CRI. This put an abrupt end to the researches in cholera on humans, leading to the years of epidemiological research on cholera in India.

The Indian Research Fund Association (established in 1911), which organized and coordinated medical research in India took up epidemiological research in the interwar period and its surveys produced detailed and fantastic epidemiological maps of cholera in India. These also confirmed the Gangetic belt as the permanent and natural 'home' of the disease. The surveys established that cholera tended to recur repeatedly in river deltaic tracts, the main endemic areas were the delta regions of all the major Indian rivers, the Ganges, Brahmaputra and Cauvery. The outbreaks commenced in the towns or villages lying on the banks of these and other rivers, the infection then rapidly and systematically spreading down the waterways:

... there is no question that, in endemic areas, cholera spontaneously appears, year after year, in the same villages and towns. In other areas, per contra, it is necessary for other favourable conditions to be present before cholera becomes diffused, e.g. overcrowded and insanitary conditions associated with religious fairs and festivals. (Russell 1927: 131)

Not just cholera, in malaria too the focus at this period was on producing epidemiological maps, locating the disease in certain so called endemic areas (Hehir 1927).

A report by J. Taylor (Director, CRI) in 1940 summarized all the cholera research in the inter-War period. He sought to reiterate the other important nineteenth-century link, between cholera and water, or 'Bengal waters' as it often referred to (Taylor 1941). In this report Taylor summarized the epidemiological studies under the IRFA between 1920 and 1940. His purpose was to identify the 'permanent endemic areas which form a source for spread to other areas and eventually to other parts of the world'. It repeated earlier conclusions, even the language, stating that 'year after year' 'The same villages and towns' showed a regular incidence of cholera (ibid.: 24). The main focus of Taylor's report was on the question of the survival of the *V. cholerae* in Bengal waters. Taylor challenged Greig's and Hankin's work to suggest that the *V. cholerae* persisted in Bengal waters up to sixteen days thereby tilting the balance firmly in favour of the water borne theory (ibid.: 28). But how did he arrive at such a conclusion? The 'field enquiries' that Taylor referred to tested large samples of water sources from tanks, open water resources of Bengal but they did not test the same sample of water after sixteen days for the presence of the *vibrio*. Rather the water sources of immediate vicinity of a cholera case were tested and were found to contain the germs for up to 16 days after the onset of that particular case (ibid.: 27). The other method was to identify the conditions in which the germs survived in water. It was found that the germ did not survive without a minimum salt and organic matter content, both of which were found to be high in the 'Bengal waters' (ibid.: 23). However, water from any other geographical region was not subjected to the same test.

After establishing the link between cholera and Bengal waters, Taylor questioned the 'carrier' thesis of Greig. He suggested that the 'carrier' was unlikely to transmit infection at any prolonged interval after primary infection since the germ had a 'very short persistence' in the intestinal tract of the convalescent (ibid.: 26). He insisted that the period in which a person might be infective was 'a few days only'. This conclusion was not based on morbid anatomy or laboratory research but on statistical epidemiological analysis. He drew evidence from a 'field enquiry' conducted in the Khulna district of Bengal where in the majority of cholera convalescents the *V. cholerae* could not be isolated from the stools later than the fifth day from the onset of the attack. The maximum period of persistence was thirteen days

(ibid.: 27). However, what was not clear was that if, as according to Greig, the *V. cholerae* survived in the intestine for a long period in a dormant state it would continue to pass through the stool. But it helped Taylor to draw the most important conclusion, that it was water rather than the human intestine that was the home of cholera, the ‘persistence [of *V. cholerae*] is longer in water than in the intestinal tract’ (ibid.: 27).

The report stressed both ‘home’ and ‘permanence’ of cholera in Bengal. He described Bengal as the ‘endemic centre’ where cholera was ‘permanently present’ (ibid.: 29). The measures of tackling the problem were thereby sanitation in Bengal, and the report made no recommendations for large-scale vaccination (ibid.: 31). It is important to point out here that by this time local sanitation in India was not a responsibility of the imperial government. In 1919, the Montague-Chelmsford reforms introduced the concept of ‘Dyarchy’, transferring functions like education, public health, sanitation and agriculture (referred to as ‘transferred’ subjects) to provincial legislative bodies, while retaining others like finance, revenue and home affairs as ‘reserved’ or ‘imperial’. This indirectly increased the number of elected Indian members in district boards and municipal corporations, since the authority to regulate local government bodies was placed in the hands of the popularly elected ministers, whose constituents naturally wanted more devolution of power (Harrison 1994: 60-98). GOI’s cholera policy continued to stress epidemiological mapping and in effect mapping became an end in itself. Little progress took place in the identification and control of the microbe itself.

What is interesting is that unlike in the nineteenth century, in the twentieth century, despite the advances in bacteriology and laboratory medicine, there was a greater acceptance of these climatic theories of diseases. In the nineteenth century, anticontagionists had adopted miasmatic theories in the face of growing international criticism and the fear of the spread of cholera from India to demonstrate the localized nature of the disease. With the advent of germ theory and laboratory research by the end of the century, these ideas were challenged. However, in the twentieth century, there was a return to climatic theories in understanding diseases, particularly those of the tropics.

EPIDEMIOLOGY AND POWER

Why this focus on epidemiology by the colonial state? Epidemiology provided cartographic visibility and statistical clarity to the disease, which was essential to the purposes of power and governmentality. It also provided the moral continuum by linking colonial diseases with climate and culture. The tradition had started in mid-Victorian England, when cholera offered opportunities for an assertive public health movement promoting state intervention into the lives of the unhealthy urban poor leading to John Snow’s famous cholera maps and their apparently decisive demonstration of the water-borne nature of the disease. This acted as a legitimization of specialist medical authority and cholera marked the emergence of medical geography in Britain (Gilbert 2004). Epidemiology was adopted by the colonial state, in the face of growing international criticism and fear of the spread of cholera from India to demonstrate the localized nature of the disease. These traditions of localizing disease and suffering have become integral to modern approaches to diseases of the poor. Epidemiology has been adopted by postcolonial states and organization like the WHO to similarly localize disease problems. The WHO report of 1959 on cholera marked the epidemiological dead end. It discussed survival of the cholera vibrio in different rivers of the world, from the Rostock to the Shanghai in several substances like earth and dust, cloth and cotton, leather and rubber (six hours), paper (‘20 hours on a postcard’, tests were also conducted on Chinese banknotes, four hours), metals (silver coins), tobacco (24 hours), meat (in winter one to two weeks), fish and shellfish, whey, butter, salt, sugar and honey, bread and cakes, cereals, potatoes, onions and garlic, green vegetables (particularly in cucumbers),

fruits, beverages (coffee, beer, wine, tea, cocoa), etc. (Pollitzer 1959: 172-80). Along with this WHO position of the 1950s, cholera was seen as crucial to discussions on 'medical geography' in creating the 'convergence' of pathological and geographical factors, which even led to the coinage of a new term; 'geogens'. Geogens are those 'pathological complex' which consisted of both physical environments and cultural habits (May 1950). Along with that the policy of the government of India has been in locating cholera in the rivers and water bodies of India.

Consequently, little progress took place in the identification and controlling of the microbe itself. It was only in 1959, that Shambhu Nath De, who worked among dead patients in the pathological laboratory in Calcutta, demonstrated that cholera bacteria secrete enterotoxin in the intestine (De 1953,1959,1961). De provided a new definition of the locality of cholera, '... cholera should be regarded as a *local* infection with the bacteria confined to the superficial mucosa of the small intestine' (De 1961: 105). This discovery eventually promoted research to find a treatment aimed directly at neutralizing the cholera enterotoxin. It was based on his work that the next generation of vaccines was developed in the 1960s by South East Asia Treaty Organization (SEATO) and used in East Pakistan. This led to the development of oral vaccines (Heyningen and Seal 1983).

The historical backdrop is important because even in the contemporary world cholera continues to be linked to the environment. Statistical researches of the twentieth century have re-established cholera as a disease of 'locality', with the Gangetic-Brahmaputra basin identified as the endemic region or the 'reservoir'. Recent studies on climatic influences of cholera have renewed some of the lost links between environment and cholera. Such studies have also been shaped by the growing concerns over the effects of climate change and environmental deterioration on disease dynamics. Cholera by its recurring presence in certain climatic zones has been a particularly important epidemic for such studies. These have reiterated the Gangetic and Brahmaputra basin as the 'native habitat' of cholera. Cholera seemed to be more prevalent in lower deltaic belts than cooler higher locales and appeared to wax and wane in cycles from 3 to 6 years. Earlier this pattern was explained, as discussed above in climatic and lunar patterns, particularly connected with rainfall patterns. In recent times these reasons have been stressed again connected with a new factor El Niño which initially showed remarkable links with cholera outbreaks in South America, and later with those of Bengal (Pascual, Bouma, Dobson 2002).

The pathogen itself is seen to have a 'life' of its own in these localities. In 1961 a new strain of cholera vibrio, haemolytic EI Tor, was discovered which spread through Asia, Philippines, Hong Kong in 1962, Burma and Bangladesh in 1963, India in 1964, Pakistan, Afghanistan, Iran, Iraq, South of USSR in 1965-6 and finally to Europe and Africa in 1970. In 1992, large parts of Bangladesh and India, particularly coastal areas of Tamil Nadu and West Bengal were ravaged by a previously unrecognized strain, which was subsequently designated as O139. Since then, this strain has been isolated from 12 other Asian countries. The resurgence of O139 serogroup in September 1996 in Kolkata and the coexistence of both the O1 and O139 serogroups in much of the cholera-prone areas of India and elsewhere, suggested that the O139 serogroup had come to stay and would be an entity to contend with in the coming years.

CHOLERA AND POLITICS

The epidemiological focus has continued to highlight the problem with local water, despite the previous evidences against it and in the 1960s WHO started the massive project of installing deep tube wells in Eastern India and Bangladesh. This has led to disastrous consequences. In Bangladesh tube wells were installed to provide 'pure water' to prevent

morbidity and mortality from gastrointestinal disease. This led to widespread arsenic poisoning produced from the deep tube wells, which kills more people today in these parts than cholera. The contamination of groundwater by arsenic in Bangladesh is the largest poisoning of a population in history, with millions of people exposed (Smith, Lingas, Rahman 2000).

The debate about locating the cholera in the human body or in water has been ultimately a political debate. Cholera resided neither just in the small intestine nor in the water. More than any other, it was a disease borne by poverty. The causality of the continued presence of cholera among the poorest of the population is as much connected to the new strains of Cholera vibrio, the El Niño, as to the dwindling water resources of the poor as evident in parts of Africa and Asia today. Despite its historically complex causality, its recurrence in some of the poorest parts of the world has been the reasons why increasingly it has been defined as the 'the disease of poverty'.

Haffkine while on his vaccination drives in Calcutta noticed that 'the intellectual part of the population almost does not suffer from cholera at all, and the part which chiefly suffers from it is exclusively composed of classes whose fate cannot be traced afterwards, who move constantly from place to place and are soon lost sight of' (Haffkine 1895: 39). Cholera was in nineteenth-century India and continues to be in contemporary India, not just a disease of a locality, or even the *Vibrio cholerae*, it was a disease of poverty. Water is a resource access to which is economically determined. In the twenty-first century in the era of privatization of water, this acquires particular significance. The privatization of water in India is going ahead in spite of mounting evidence, especially from Africa, that it leads to epidemics. In The dolphin Coast in South Africa, local councils had, in 1988, commercialized water and compelled local residents to pay the full cost of drinking water. In August 2000, when cholera broke out in Dolphin Coast, civic officials dismissed it as just a sporadic outbreak — one among many in the area. But it turned out to be the worst cholera epidemic in South Africa's history, infecting over 25,000 people and killing about 300 before it wound down in January 2002. According to David Hemson, a social scientist at the Human Sciences Research Council (HSRC) of South Africa commissioned by the government to investigate the causes of the cholera outbreak, making people pay the full cost of drinking water was 'the direct cause' (Hemson 2008). Millions of poor people simply could not afford to pay the rate charged for drinking water and were, therefore, compelled to find water in streams, ponds and lakes polluted with manure and human waste.

A report generated after a year-long investigation undertaken by the International Consortium of Investigative Journalists (ICIJ), a project of the Centre for Public Integrity, has blamed the outbreaks of cholera in Asia and Africa on 'an aggressive group of utility companies, primarily European, that are attempting to privatize the world's drinking water with the help of the World Bank and other international financial institutions'. The investigation revealed that the world's three largest water companies, France's Suez and Vivendi Environment and British-based Thames Water, owned by Germany's RWE AG, have since 1990 expanded into every region of the world. Their revenue growth has grown in conjunction to their overseas expansion. Vivendi universal, the parent of Vivendi Environment, reported earning over \$5 billion in water-related revenue in 1990; by 2002 that had increased to over \$12 billion. RWE the parent of Thames Water increased its international water revenue by a whopping 9,786 per cent — from \$25 million in 1990 to \$2.5 billion in fiscal 2002 (Ravindran 2003).

The position of WHO, following the arsenic disaster and SEATO antitoxin vaccine research, has been to promote cholera vaccination. It now states that personal hygiene, food safety and sanitation are although important, drastic improvements in these fields are difficult to

achieve in most cholera endemic areas, which are also poverty stricken. The WHO policy paper admitted that a vaccine was available, it 'has not for many years been recommended by WHO'. At the moment though, it has accepted that 'There is an urgent need for cholera vaccines that are efficient against the different epidemic types of *V. cholerae*' (WHO 2001). The WHO continues to search for a cholera vaccine and scientists reiterate that an effective vaccine for cholera is around the corner, particularly since the genes controlling production of cholera toxin are now defined which is assisting in the design of more effective vaccines (Lagos 1999). In the twenty-first century, disease eradication policy and medical research continues to revisit the nineteenth- and twentieth-century problematic of locating causality of disease in either the environment or in the human body and to that extent either through 'field enquiries' or laboratory research. The history of the search for the causality of cholera highlights the problem in identifying disease in the binary opposition of the field and the laboratory and the need for new conceptual frameworks.

CONCLUSION: CHOLERA AND POVERTY

Did epidemiological studies liberate cholera research from the confines of the bacteriological laboratory? The answer to this question lies in the fact that in medical terms, the tropical environment was as structured and confined a space as was the modern laboratory. Cholera research in the twentieth century has struggled for cognizance between the environment and the laboratory. The epidemiological focus has continued to identify cholera with the tropics, with local water, despite evidences against it. By these intensive mappings cholera was not only located to its 'home', but also dislocated from the problematic of Europe, along with colonial poverty. The failure of the laboratories to devise means of eradicating cholera was a failure of this medical paradigm.

Shambhu Nath De, who discovered the cholera toxin, raised some interesting questions about the endemicity of cholera. He demonstrated through historical records that in the eighteenth and nineteenth centuries, cholera appeared with the same periodicity in both Asia and Europe. He found that 'cholera should not be regarded as something natural and innate to the soil and atmosphere of any particular place...'. He instead suggested a theory of 'local revival after long quiescence' and showed that there have been sudden and sporadic outbreaks in Europe as well in the twentieth century. To explain these outbreaks he moved away from the environmental causality and suggested that the dormant pathogen which inhabits the human body flares up in situations of bodily stress, particularly in times of war and famine when 'the resistance to infection was lowered' (De 1961: 39). This provides us with a third perspective on cholera; the relationship between stress, poverty and cholera.

If anything, cholera is endemic to poverty. Cholera and other diarrheal diseases currently account for 11.3 per cent of all deaths among the poor globally, making such illnesses the second leading cause of deaths among the poor. The optimism about eradicating cholera of the 1970s has now disappeared (Anbarci, Escaleras, Register 2006). The poor are asked to wait for the next vaccine and clean sources of water. Cholera policy of the WHO had oscillated constantly between sanitation measures and vaccine research. In the 1960s the WHO, encouraged by the epidemiological studies and the Pollitzer report adopted sanitary measures as the mainstay of its fight against the disease. It began a massive project of installing deep tube wells in Eastern India and Bangladesh. This has led to disastrous consequences. It led to widespread arsenic poisoning produced from the deep tube wells, which kills more people today in these parts than cholera (Smith, Lingas, Rahman 2000). On the other hand, the development of effective treatment against cholera has remained elusive (Anbarci, Escaleras, Register 2006). Little research was conducted in India after Haffkine on the cholera vaccine or toxin. In the 1990s the WHO actively discouraged the use of cholera vaccines 'under any circumstances' (WHO 1992). The optimism about eradicating cholera

in the 1970s has now disappeared. The WHO has now stated that hygiene, food safety and sanitation are although important, drastic improvements in these fields are difficult to achieve in most cholera endemic areas, which are also poverty-stricken (WHO 2001). It has reiterated the need for a new vaccine for the successful eradication of the disease. This is evident in the seemingly shifting home of cholera in the twenty-first century. In 2007 about 62 per cent of cholera cases and 56.7 per cent of cholera deaths were reported from the WHO African Region (Kirigia, Sambo, Yokouide, Soumbey-Alley, Muthuri 2009).

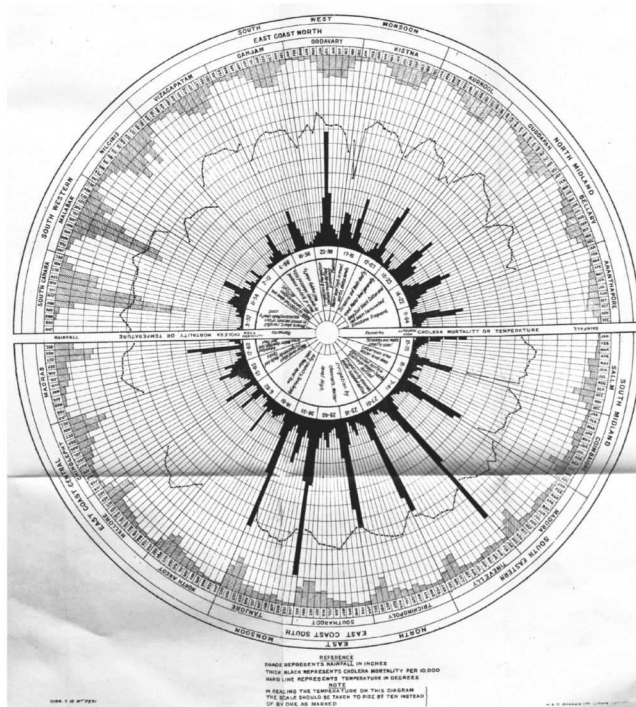
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