

together with chronic ills provoked by altered conditions of living.

Until near the end of the nineteenth century the race remained close in most of the world's great cities. Growing urban centers that lagged in implementing sanitary reforms, like New York and most other American cities, actually saw a sharp increase in mortality.³⁹ But from the 1880s onward, a series of dramatic triumphs accrued to medical researchers who succeeded in isolating and studying the "germs" of one infectious disease after another. Careful study usually allowed experts to devise effective ways of checking infection, whether by synthesizing new drugs or devising immunizing injections, introducing new sanitary practices, altering older patterns of human encounter with insects, rodents, or other alternate hosts for the disease in question, or in some other fashion contriving to interrupt the established patterns of disease transmission. International organization supplemented urban and national measures aimed against infectious diseases, so that by the first decades of the twentieth century preventive medicine began to make a dent in the epidemiological experience of Asian and African as well as European and European-descended populations.

Success was sufficient so that by the second half of our century, professionals seriously proposed the global eradication of a number of mankind's most formidable infections, and thought it a feasible goal for the near future.⁴⁰ But as is their wont, such massive and fundamental successes in altering humanity's experience of disease carried within them a potential nemesis: population crises on a continent-wide scale seemed likely to supplant the localized population crises afflicting the new industrial cities with which nineteenth-century medical reformers had to cope. The race between skills and ills was thus by no means decisively won—or lost; and in the nature of ecological relationships is never likely to be.

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The first and in many ways most significant manifestation of the altered disease relationships created by industrialization was the global peregrination of cholera. This disease had long been endemic in Bengal, and spread thence in epidemic fashion to other parts of India and adjacent regions

from time to time. It was caused by a bacillus that could live as an independent organism in water for lengthy periods of time. Once swallowed, if the cholera bacillus survives the stomach juices, it is capable of swift multiplication in the human alimentary tract, and produces violent and dramatic symptoms—diarrhea, vomiting, fever, and death, often within a few hours of the first signs of illness. The speed with which cholera killed was profoundly alarming, since perfectly healthy people could never feel safe from sudden death when the infection was anywhere near. In addition, the symptoms were peculiarly horrible: radical dehydration meant that a victim shrank into a wizened caricature of his former self within a few hours, while ruptured capillaries discolored the skin, turning it black and blue. The effect was to make mortality uniquely visible: patterns of bodily decay were exacerbated and accelerated, as in a time-lapse motion picture, to remind all who saw it of death's ugly horror and utter inevitability.

The statistical impact of cholera was occasionally severe: in Cairo about 13 per cent of the total population succumbed in 1831 when the disease first affected that city.⁴¹ But this was unusual, and in European cities losses were never anything like that great. But this did not diminish the unique psychological impact of the approach of such a killer. Cholera seemed capable of penetrating any quarantine, of bypassing any man-made obstacle: it chose its victims erratically, mainly but not exclusively from the lower classes in European towns. It was, in short, both uniquely dreadful in itself and unparalleled in recent European experience. Reaction was correspondingly frantic and far-reaching.

The disease first came acutely to European attention when an unusually severe outbreak of cholera developed in the hinterland of Calcutta in 1817. Thence it spread to other parts of India, and soon transgressed the boundaries that had previously confined it to the subcontinent and immediately adjacent regions. What seems to have happened is that an old and well-established pattern for spreading cholera across the Indian landscape intersected new, British-imposed patterns of trade and military movement. The result was that

the cholera overleaped its familiar bounds and burst into new and unfamiliar territories, where human resistance and customary reactions to its presence were totally lacking.

From time immemorial, it appears, Hindu pilgrimages and times of festival had drawn great crowds to the lower Ganges, where cholera was endemic. Consequently, the celebrants had been liable to pick up cholera along with other infections. Those who did not succumb on the spot were liable in turn to carry the infection back home, where it ran an accustomed if nasty and sometimes demographically destructive course.⁴² The association of cholera with pilgrimage and holy days in India continues to the present⁴³; and prior to 1817 one may safely assume that well-defined custom pretty well confined the dissemination of the infection to the range of Hindu pilgrimage, i.e., to India proper. Nevertheless, from time to time cholera infection reached as far afield as China, traveling by ship. This is attested by the fact that when cholera penetrated China early in the nineteenth century, the Chinese did not regard it as a new disease, even though it had not been seen on the China coast for some time previously.⁴⁴

In 1817, however, when an unusually severe cholera epidemic started to re-enact its familiar pattern, English ships and troops were also on the scene; and their presence and movement to and from the primary focus in and around Calcutta, carried the infection to completely unfamiliar ground.

The expansion followed two routes. One was overland, and of relatively limited range. British troops fighting a series of campaigns along India's northern frontiers between 1816 and 1818 carried the cholera with them from their headquarters in Bengal, and communicated the disease to their Nepalese and Afghan enemies. Far more dramatic were the movements by sea. Ships carried cholera to Ceylon, Indonesia, the southeastern Asian mainland, China, and Japan between 1820 and 1822. Muscat in southern Arabia encountered the disease when a British expeditionary force, intent on suppressing the slave trade, landed there in 1821; and from Muscat the cholera filtered south along the east coast of Africa, following the slave traders. The infection also entered the Persian Gulf, penetrated Mesopotamia and Iran, and continued north into Syria, Anatolia, and the Caspian shores. There it stopped

short, more perhaps because the winter of 1823-24 was unusually severe than because of any action by either Russian, Turkish, or Persian authorities. It lingered longer in China and Japan; indeed it is not clear that the disease had disappeared from China before the second epidemic wave got going in 1826.⁴⁵

The episode proved only a foretaste of the far more extensive wanderings of the cholera bacillus in the 1830s, making the disease genuinely global. A new cholera epidemic emerged from Bengal in 1826 and quickly retraced its previous path into southern Russia. Military movements connected with Russia's wars against Persia (1826-28) and Turkey (1828-29) and the Polish revolt of 1830-31, carried the cholera to the Baltic by 1831, whence it spread by ship to England. In the next year it invaded Ireland; and Irish emigrants carried the disease to Canada, whence it filtered southward into the United States (1832) and Mexico (1833).

More enduringly important than this first sally into the European heartlands was the fact that cholera established itself at Mecca in 1831 at the time of the Moslem pilgrimage.⁴⁶ The inevitable result was the re-enactment of the patterns of epidemic dispersion long familiar within India, but this time on a much expanded geographic scale as followers of Muhammad headed homeward, whether west to Morocco or east to Mindanao, or to points between. Thereafter until 1912, when cholera broke out in Mecca and Medina for the last time,⁴⁷ epidemics of this dread disease were a common accompaniment of the Moslem pilgrimage, appearing no fewer than forty times between 1831 and 1912, or every other year on the average.⁴⁸

As cholera thus added the Moslem pilgrimage to its older Hindu pilgrimage dispersal routes, the exposure of peoples beyond India's borders to the new disease became chronic. On top of this, after mid-century the swifter movement of steamships and railroads became increasingly able to accelerate the global diffusion of cholera from any major world center. As a result, cholera deaths beyond India's borders certainly totaled millions in the nineteenth century, although no precise calculation seems feasible. In India itself the disease was and remains important, causing far more deaths than

plague⁴⁹; but cholera in India, being thoroughly familiar, excited no special alarm or surprise.

It was otherwise, however, beyond India's borders. Moslems had long been resigned to plague and found European quarantine efforts rather amusing. But the unfamiliar, dreadful, and sudden nature of cholera deaths created among the population of Egypt and other affected Moslem lands almost the same alarm that prevailed in Europe. Neither Moslem medical nor religious traditions were able to cope. The popular fright cholera aroused helped to discredit traditional leadership and authority within the Moslem world, and opened the way for reception of European medicine.⁵⁰

In Europe, to be sure, there were a few localities where memories of former visitations by the plague remained sufficiently vivid that public and private responses to the emergency could find fitting if somewhat archaic expression. This was the case in much of Mediterranean Europe, where a combination of religious supplication and medical quarantine had been built into public law ever since the sixteenth century. Thus in Marseilles, where annual commemoration of the plague of 1721 had kept memories of that disaster very much alive, the cholera became an occasion for renewal of Christian piety.⁵¹

In northern Europe, however, traditional guidelines for behavior in time of epidemiological crisis were less well defined. To be sure, chronic tensions between social classes tended to find overt and even ritualized expression in places as diverse as St. Petersburg and Paris⁵²; but such symptoms of social strain did not easily convert into concrete and definite programs of action. People had therefore to improvise, argue, and flee, as well as plead, threaten, and pray. In other words, there was a wide spectrum of behavior from which to choose the most effective way to cope with what everyone agreed was a real and present threat to life and society. From these perturbations, refreshed at frequent intervals during the rest of the nineteenth century, came the major impetus to improvements in urban sanitation and public life regulation.⁵³

To begin with, cholera added new urgency to long-standing debates between rival schools of thought about epidemics. Since the days of Hippocrates, some European doctors had

held that sudden outbreaks of disease were caused by a miasma, emerging perhaps from dead corpses or other rotting matter in the earth. When the miasma encountered appropriately weakened constitutions, these theorists believed, disease resulted. Wherever encounters with malaria and other insect-borne diseases remained important, the miasmatic theory had a firm and satisfactory empirical basis—or seemed to.

The rival germ theory of contagion had been clearly advanced as early as 1546 by Girolamo Fracastoro. This provided the theoretical justification for the sort of quarantine regulations that had become standard in the Mediterranean against plague. But early in the nineteenth century the germ theory was put on the defensive. The occasion was the disaster that came to French troops sent to Santo Domingo in 1802 to suppress rebellion led by Toussaint L'Ouverture. Within a few months, yellow fever and other tropical diseases utterly destroyed a force of 33,000 veterans, and the resulting setback to Napoleon's imperial ambitions (among other things) made him willing to sell the Louisiana Territory to the United States in 1803. This dramatic demonstration of the power of disease to blunt European military force overseas gave a special fillip to study of tropical diseases among French doctors; and when yellow fever broke out in Barcelona in 1822, they seized the opportunity to make a definitive test of the contagionist as against the miasmatic school of thought. French experts, led by Nicholas Chervin, organized systematic and careful study of how the disease occurred. They concluded that there was no possibility of contact among the different persons who came down with yellow fever in Barcelona. Thus contagionism seemed to have been fully and finally discredited.

For the next fifty years medical reformers set out to dismantle the long-standing quarantine regulations of Mediterranean ports, arguing that they were mere survivals from a superstitious age. Lacking any empirical base—for no one as yet imagined that insects might be carriers of disease—the germ theory seemed destined for the scrap heap of history.⁶⁴ British liberals, in particular, saw quarantine regulations as an irrational infringement of the principle of free trade, and bent

every effort toward the eradication of such traces of tyranny and Roman Catholic folly.

Yet in 1854 a London doctor, John Snow, neatly demonstrated how cases of cholera that broke out in a district of central London could all be traced to a single contaminated source of drinking water. But Snow's argument was merely circumstantial⁵⁵; and since contagionism had been so recently and so definitively discredited by Europe's most meticulous and celebrated medical experts, Snow's interpretation of his data commanded little attention. Then in the 1880s the microscope abruptly reversed the balance of medical opinion with the dramatic discovery of disease-causing "germs."

The first such germs to be detected were the bacilli of anthrax and tuberculosis, discovered respectively by Louis Pasteur, between 1877 and 1879 and Robert Koch in 1882. Since neither of these infections spread in a dramatically epidemic fashion, their identification did not upset the miasmatic theory, which had come into existence to account for epidemics. It was otherwise when in 1883 Robert Koch claimed to have found a new bacillus responsible for cholera, for if Koch was right the miasmatic theory was wrong—at least in explaining cholera.⁵⁶

Since many learned and respected doctors had committed themselves to the miasmatic theory as explanation of epidemic, it is not surprising to find that Koch's explanation for the cause of cholera met stout resistance among experts.⁵⁷ As late as 1892, a famous German doctor drank a beaker full of cholera bacilli to prove the falsity of the germ theory—and gleefully informed his professional rivals that he had experienced no ill effects.⁵⁸ No doubt he was lucky; but his act dramatized the uncertainties that still surround the question of what factors affect transmission of cholera infections. Perhaps in the professor's case, anger and nervousness provoked an extra charge of stomach acids which sufficed to kill the bacilli he swallowed.⁵⁹

Long before Koch's microscope thus provided doctors with an empirical base for the modern view of how cholera spreads, the alarm it created in American and European cities provided essential leverage for those reformers who sought to improve urban sanitation, housing, health services, and water

supply. Models of what to do and how to do it were readily at hand, for during the eighteenth century European governments discovered that soldiers' and sailors' lives were much too valuable to squander needlessly, when simple and not overly expensive measures could check the ravages of disease.

The most famous and significant of these health measures was the use of citrus juice to ward off scurvy. This disease haunted European ships on long voyages, when crews for weeks or months on end ate food that lacked essential vitamins. Its peculiar pattern of incidence provoked an abundant medical literature; and as early as 1611 the use of lemons and oranges as a cure was recommended in print, and repeated thereafter by respectable and important medical writers. But other cures were no less warmly recommended, and a supply of citrus fruit was often hard to come by. Hence the superior effectiveness of the cure was not clearly recognized until the end of the eighteenth century.

Indeed, even after a British naval surgeon, James Lind, published the results of his carefully controlled experiments that proved the efficacy of fresh lemons and oranges in curing scurvy (1753), the Admiralty did not act. The reason was partly pecuniary: citrus fruit was expensive and scarce and could not be stored for very long. Partly too, the naval authorities believed other cures were suitable, e.g., the sauerkraut Captain James Cook fed his crews in the Pacific. Moreover, when in 1795 the Admiralty did decide on citrus juices as the best preventative for scurvy and prescribed a daily ration for all sailors on shipboard, the result was imperfect. The species of limes grown in the West Indies lacked the essential vitamins; but it soon proved that West Indian limes were cheaper than Mediterranean lemons, with the result that the British navy soon was drinking the almost valueless lime juice that gave them the nickname, "Limeys." As late as 1875, therefore, outbreaks of scurvy occurred on British naval vessels, despite the daily dose of lime juice prescribed by regulations.⁶⁰

In spite of such confusion and inefficiency, James Lind and other medical men in the British navy pioneered a number of other significant improvements in health administration during the latter decades of the eighteenth century. Lind was in-

and faster ships plying between South America and Europe could traverse the tropics without allowing their holds to heat up past the critical temperature which the fungus could not survive.

39. One calculation concluded that one out of every forty-six New Yorkers died annually in 1810, whereas one out of twenty-seven did so in 1859. Cf. Howard D. Kramer, "The Beginnings of the Public Health Movement in the United States," *Bulletin of the History of Medicine*, 21 (1947), 352-76. In Paris the death rate increased between 1817 and 1835 from 31 to 34 per thousand. Cf. Roderick E. McGrew, *Russia and the Cholera, 1823-1832* (Madison and Milwaukee, Wisconsin, 1965), p. 6.

40. Cf. Aidan T. Cockburn, *The Evolution and Eradication of Infectious Diseases*, p. 196: ". . . there is no reason why the last case of smallpox should not disappear within two or three years." (1963).

41. Laverne Kuhnke, *Resistance and Response to Modernization: Preventive Medicine and Social Control in Egypt, 1825-1850* (Unpublished Ph.D. dissertation, Chicago, 1971), p. 51.

42. A number of casual references to sudden outbreaks of lethal disease in southern and western India that sound like cholera punctuate European records from the days of the first Portuguese residents at Goa. Cf. R. Pollitzer, *Cholera* (Geneva, 1959), pp. 12-13. C. Macnamara, *A History of Asiatic Cholera* (London, 1876), discovered no fewer than sixty-four such references dating between 1503 and 1817.

43. Cf. the bar graphs in Pollitzer, *op. cit.*, p. 80.

44. Cf. C. H. Gordon, *An Epitome of the Reports of the Medical Officers of the Chinese Imperial Customs from 1871 to 1882* (London, 1884), p. 124.

45. Pollitzer, *op. cit.*, pp. 17-21; McGrew, *Russia and the Cholera*, pp. 39-40; Norman Longmate, *King Cholera: The Biography of a Disease* (London, 1966), pp. 2-3; Hirsch, *Handbook of Geographical and Historical Pathology*, I, 394-97.

46. Estimates of mortality ranged from 12,000 to 30,000. Cf. Laverne Kuhnke, *op. cit.*, p. 66.

47. There was a minor recurrence in 1930, but nothing

was reported from Mecca itself on that occasion. Pollitzer, *op. cit.*, p. 63.

48. Norman Longmate, *King Cholera*, p. 237.

49. Between 1910 and 1954, 10.2 million died of cholera in India according to official tabulation; to these should be added nearly 200,000 deaths in Pakistan since 1947. Pollitzer, *op. cit.*, p. 204 and *passim*.

50. Kuhnke, *op. cit.*, p. 204 and *passim*.

51. Asa Briggs, "Cholera and Society in the 19th Century," *Past and Present*, 19 (1961), 76-96.

52. McGrew, *op. cit.*, pp. 67, 111, 125; Longmate, *King Cholera*, pp. 4-5; Louis Chevalier, ed., *Le Cholera, la Première Épidémie du XIX^e Siècle* (La Roche sur Yon, 1958).

53. Cf. Charles E. Rosenberg, "Cholera in 19th Century Europe: A Tool for Social and Economic Analysis," *Comparative Studies in Society and History*, 8 (1966), 452-63.

54. Erwin H. Ackerknecht, "Anti-contagionism between 1821 and 1867," *Bulletin of the History of Medicine*, 22 (1948), 562-93.

55. Reprinted as *Snow on Cholera, being a Reprint of Two Papers by John Snow, M.D.* (New York, 1936).

56. According to Norman Howard-Jones, "Cholera anomalies: the Unhistory of Medicine as Exemplified by Cholera," *Perspectives in Biology and Medicine*, 15 (1972), 422-33, an Italian named Filippo Pacini anticipated Koch by some thirty years in identifying the "vibrio" as causing cholera; but his theory attracted almost no attention at the time and it was thus Koch's "discovery" that mattered as far as medical opinion and practice are concerned.

57. The motivation behind Charles Creighton's monumental book, *The History of Epidemics in Britain*, 2 vols. (Cambridge, 1891, 1894) was a passionate wish to disprove the germ theory of epidemic infection.

58. Longmate, *King Cholera*, p. 229.

59. Pollitzer, *Cholera*, pp. 202-372, offers a careful discussion of the complex factors that are currently believed to affect cholera infections.

60. It has long been customary to ridicule the way the Admiralty handled scurvy. On the surface it certainly looks like a classic case of bureaucratic bungling. When effective cure

and prevention had been published by respectable medical men as early as 1611 and several times thereafter, how could official command wait till 1795? Cf. John Woodall, *The Surgeon's Mate or Military and Domestique Surgery*, 2nd ed. (London, 1639), p. 165. "Of the Cure of the Scurvie," which reads in part as follows: "The use of the juyce of lemons is a precious medicine and well tried, being sound and good, let it have the chief place, for it will deserve it. . . . Some Chirurgeons also give this juyce daily to the men in health as a preservative, which course is good if they have store, otherwise it were best to keep it for need."

Yet it is a defect of historical perspective to assume from passages such as this that *the* cure for scurvy was apparent in London before the very end of the eighteenth century. For explanation of the reasons for delay and misinformation, see John Joyce Keevil, *Medicine and the Navy, 1200-1900*, 4 vols. (London, 1957-63) I, 151; Christopher Lloyd and Jack S. Coulter, *ibid.*, III, 298-327.

61. On sanitation in European armies in the eighteenth century, see Paul Delaunay, *La Vie Médicale aux XVI^e, XVII^e et XVIII^e Siècles* (Paris, 1935), pp. 84ff, 275-80 and *passim*; Charles Singer and A. E. Underwood, *A Short History of Medicine* (New York, 1928), pp. 169-71; George Rosen, *From Medical Police to Social Medicine: Essays on the History of Health Care* (New York, 1974), pp. 120-58, 201-45; David M. Vess, *Medical Revolution in France, 1789-1796* (Gainesville, Florida, 1975). On Frank, see Henry E. Sigerist, *Grosse Ärzte*, 4th ed. (Munich, 1959), pp. 217-29.

62. Cf. R. A. Lewis, *Edwin Chadwick and the Public Health Movement, 1832-1854*, (London, 1952, pp. 52-55 and *passim*. Chadwick's proposal for using urban sewage for fertilizer was not a new idea. It had, in fact, been projected as early as 1594. Allen G. Debus, "Palissy, Plat and English Agricultural Chemistry in the 16th and 17th centuries," *Archives int. hist. sci.*, 21 (1968), 67-88.

63. Cf. C. Fraser Brockington, *A Short History of Public Health* (London, 1966), pp. 34-43.

64. Cf. Charles E. Rosenberg, *The Cholera Years: The United States in 1832, 1849 and 1866* (Chicago, 1962), pp.