CHAPTER III

Time Trends of Cholera in India : An Overview

This chapter provides an overview of the history of cholera epidemics and reviews literature that brings out the disease dynamics over time with changing biological nature of the agent. This chapter is divided into three sections. The first section reviews cholera pandemics (caused by the classical biotype) in India from 1817 upto the arrival of *Vibrio cholerae* El tor in 1964. The determinants responsible for the spread of cholera in the region have been examined in detail. History of cholera in India has been closely linked with fairs and the state response to it. History of ideas regarding causality of cholera and its preventive strategies have also been reviewed. The second section begins with a review of the progress of the seventh cholera pandemic (due to *Vibrio cholerae* El tor) across the world. India was affected in 1964 by El tor cholera. Its spread across the Indian states has been described with particular emphasis on the determinants that emerged from different epidemiological investigations. While Bengal was the home of classical cholera, the El tor strain established itself in other states of India and acquired a new endemicity pattern. The third section examines the situation arising out of the introduction of yet another 'novel' strain in 1992-93 and the forecast of a possible eighth pandemic. But the new strain (O139 Bengal) failed to prevail over El tor which soon regained its primacy.

PANDEMICS OF CHOLERA (CLASSICAL BIOTYPE)

In the colonial history of India, cholera occupied an important place not only as a public health challenge but also as a political issue. This political disease was a matter of concern to the British rulers and was a major issue for the interface of the colonial state and the indigenous society. It is interesting that Hospital Boards were established in Madras and Calcutta in 1786 as prior to that year 'no regular reports on the incidence of cholera among the Europeans and the native soldiers were available' (Pollitzer, 1959).

The West was not much concerned about cholera till the invasion of Europe by cholera during 1830s and 1890s when it was treated with the same concern as the import of yellow fever from West Africa and the West Indies (Arnold, 1989). However, collection of data did not begin until late 1860s; figures available for reported deaths during the 19th century are therefore not considered to be accurate. Monitoring of cholera during nineteenth century and

first half of twentieth century was by deaths and/or annual mortality rates. This was on account of high case fatality rate of cholera. Better statistics was available from 1870s. Mortality data began to be collected systematically with the epidemic of 1865 in Bengal. Between 1817 and 1865, an estimated 15 million deaths occurred in British India and a further 23 million deaths is estimated to have occurred between 1865 and 1947 (Arnold, 1993). During the last quarter of the 19th century, on an average, annual cholera mortality rate of British India was 1.75 per thousand population. In 1874, this rate was at a low of 0.16 per thousand. In 1877, 1892 and 1906 mortality due to cholera exceeded 3 per thousand. The year 1900 was badly affected by cholera, mortality peaked to 3.70 per 1000. Though it is customary to describe cholera history in terms of pandemics, different writers disagree regarding the dates of onset and duration of successive pandemics (Pollitzer, 1959) and it is difficult to validate any of these due to paucity of information. Our focus is on the sixth and seventh pandemics as for the initial pandemics either the data is scanty or their spread was largely outside India.

First Pandemic

During the last quarter of eighteenth century, cholera was prevalent both in eastern and western coastal areas of India and beyond the sub-continent as well. The First Pandemic originated in the year 1817. It was first reported on 23rd August of that year by the Civil Surgeon of Jessore (now in Bangladesh). The hinterland between Ganga and Brahmaputra rivers experienced storms and very heavy rainfall in early August. Calcutta was affected at about the same time and the disease spread beyond the borders of Bengal. This was confirmed by the Calcutta Medical Board. Macnamara (1876) described the epidemic having affected 195,935 square miles of Bengal Province and that 'not a single village or town was spared'.

Later, during the year cholera was reported outside Bengal. The army of the Marquis of Hastings then camping at Bundelkhand was severely affected. The General entered in his diary on 17th November, 1817 (as quoted by Macnamara) that 'it is ascertained that above 500 have died since sunset yesterday', signifying a high case fatality rate. In 1818, cholera

spread to northern India. Agra and Delhi were affected in 1818 while Punjab reported its first case in 1820.

No overall estimates of mortality are available for the epidemic of 1817-21. For the period 1817-1831, Moreau de Jonnes, a French physician calculated that cholera affected one-tenth of the population of British India and killed one-sixteenth, on the basis of mortality figures for certain areas of Bengal and among troops of the East India Company – a methodology that has been criticised by others. He postulated an average annual mortality of 1.25 million annually and a total of 18 million deaths for the period. Other estimates put the total cholera deaths for India at 40 to 50 million.

Whatever the correct approximations may be, the 1817 epidemic came nearly three decades after the last recorded epidemic of 1780s; the population was immunologically vulnerable leading to a high attack rate and mortality rate. It is important to recognise that 1815 and particularly 1817 was marked by extremely heavy rainfall, severe floods and harvest failures. 1816 was extraordinarily hot and dry (Pollitzer, 1959). Though the most severe epidemics have later been associated with famines there was no famine in Bengal or other parts of India during that period (Arnold, 1993).

The poor and the undernourished were the worst affected in the 1817-21 epidemic. Though European residents were alarmed at the spread of cholera, they were not seriously affected. They were protected partly by relatively healthy living conditions and later (by 1850s) the emphasis on clean drinking water and personal hygiene. Mortality among slum dwellers of Calcutta was high, in comparison to the "higher classes of Native and Europeans." This pattern was also confirmed from observations in Bombay and Madras (Arnold, 1993).

Second and Third Pandemics

The Second Pandemic also originated in Bengal in 1826 and spread in upstream regions of the rivers Ganga and Yamuna. It reached Punjab after affecting United Provinces (UP) and

Delhi in 1827. During a later resurgent phase of the Second Pandemic, cholera again struck Delhi by an indirect yet interesting route.

Cholera was raging in southern Bengal in 1840. Troops were being sent from Calcutta (and Madras) to China. These troops from Calcutta were considered to have 'exported' cholera from Bengal to the Straits Settlements and then into China – first affecting Chushan Island in July, 1840 outside Shanghai and then over the next two years, various parts of the mainland. Cholera spread to areas far and wide following the trade routes from China and affected Kashgar, Yarkund, Kokand and Bukhara in succession. 'It lasted for a few weeks in each place and people died by hundreds every day' (Macnamara, 1876). Cholera reached Kabul in 1844 from Bukhara, which had earlier been affected in 1829 following a direct spread from India. In 1844, however, it returned to India, reaching Punjab from where it extended south-westwards to Karachi and south-eastwards to Delhi, affecting both these cities in 1845.

The third pandemic, following a recrudescence in India, was conspicuous for its spread to countries that were till then not seriously affected by the disease. During the later phase of the pandemic in 1859, there was a spurt of cases in Bengal. The disease spread to Persia, Mesopotamia and Arabia and thereafter to Russia.

Fourth Pandemic

The fourth pandemic began in 1863. There was a major epidemic in Mecca in 1865. Some considered that the infection was imported from India by Haj pilgrims. Others disagreed and opined that the outbreak was a result of a recrudescence of infection already present in Mecca. However, all authors agree that from Mecca the infection spread to a large number of countries. The earliest instance of Kumbh Mela giving rise to a cholera epidemic was recognised for the first time during this pandemic. The April 1867 Kumbh Mela at Hardwar has been considered to be responsible for the epidemic spread of cholera in different provinces of northern India (Arnold, 1993).

1877 was an epidemic year in India. In Madras Presidency in 1877, mortality due to cholera was 12.20 per 1000. In four districts famine was also raging at the same time; in those districts mortality due to cholera rose to 20-25 per 1000. Overall, at the height of the epidemic cholera accounted for about 10% of the annual mortality[1] (Whitcombe, 1993).
The poor were worst affected when cholera epidemics coincided with famines. This was evident in the second half of the nineteenth century, a period of frequent and widespread famines. This was observed in Madras Presidency in 1866 and 1877 and in Bombay Presidency in 1877 and 1900, when mortality due to cholera peaked.

In Madras Presidency, the cholera epidemic of 1869-71 resulted in nearly 100,000 deaths. Cholera reduced to low levels in 1873 (440 deaths) and 1874 (313 deaths). In 1875, there were 94,546 deaths and the situation got worse in 1876 with 148,193 deaths. Drought and famine began in the Presidency at this stage of the epidemic and annual mortality rose to 357,430 in 1877. Though the famine reached its peak in September 1877, cholera mortality was already showing a downward trend from February. But undoubtedly, cholera mortality tended to peak when the famine was at its most severe form. In Madras Presidency, during the 1877 famine and epidemic, in the ten districts worst affected by famine cholera mortality was 18 per 1000, whereas in the six districts that were least affected, cholera mortality was 4.6 per 1000 (Arnold, 1993).

Hot and dry conditions though not conducive to the survival of *Vibrio cholerae* were nevertheless responsible for scarcity of drinking water. As a result, villagers accessed fewer available sources of water that were liable to contamination and act as sources of infection. Consumption of roots, leaves and other surrogate foods in desperation during famine conditions was generally responsible for diarrhoeal diseases. Mobility of famine affected communities in search of food and their concentration in relief camps facilitated the spread of the disease. Lack of adequate state medical relief failed to compensate for family and community care that collapsed in such disaster situations. Association of famine and high

[1] Plague accounted for an estimated 10 million deaths between 1896 and 1921 and malaria nearly twice that number.

cholera mortality emphasised that issues of poverty, food availability, hygiene and medical relief were not handled well by the colonial regime.

Fifth and Sixth Pandemics

The fifth cholera pandemic caused considerably less havoc than the previous four pandemics. It is during this pandemic (customarily stated to last from 1881 to 1896) that Robert Koch, studying the outbreaks in Egypt and Calcutta proved in 1883-84 that cholera was the result of a gastro-intestinal infection. There were epidemics in India in 1881 with particularly severe outbreaks in Punjab, specially in Lahore. In 1891, there were 169,013 deaths in the Kumbh Mela at Hardwar. Rogers (1928) considered that following the fair, cholera spread to the United Provinces and Punjab and then spread by overland route to Afghanistan, Persia, southern Russia and finally Europe in the 1892-95 phase of this pandemic. A similar situation occurred in 1894 following the Allahabad Kumbh Mela.

The sixth pandemic lasted for nearly twenty five years. Cholera cases in India had begun rising since 1899 and there were major outbreaks in Calcutta and Bombay in 1990. Madras was also affected. Since 1900, cholera had begun spreading from India to other countries. There was a resurgence of cholera in some countries between 1908-1910; some authors have linked this to the increase in cholera cases in India during 1905-08. There were 189,955 cholera deaths in India in 1904. Between 1905 and 1908, there was an average of about 526,000 deaths each year; there were nearly 150,000 deaths in the 1906 Kumbh Mela at Allahabad during this phase and the mela is also considered to have contributed to the epidemic. In 1909, deaths declined to 227,842.

Yacob (1944) and Pollitzer (1959) have linked the high incidence of cholera in Punjab in this phase to spread of infection from melas at Hardwar. Zurbrigg (1992) linked malaria in Punjab not only to climatic factors but to the importance of the role of famines[2] (hunger and malnutrition). Epidemics of cholera in Punjab in 1901, 1906 and 1908 can be correlated with the famine conditions. In fact in 1908 there were no fairs; yet there was an increased incidence of cholera.

Status of Cholera after 1924

The sixth pandemic ended in the early 1920s. Following this there was a general decline in mortality due to cholera.. Between 1900 and 1920 annual mortality rate reduced from about 112 per 100,000 to about 50 per 100,000. The decline continued; in 1947 there were about 30 cholera deaths per 100,000 population and by 1963 there were 4.3 cholera deaths per 100,000 population (Patnaik and Kapoor, 1967). The phase of 1920-63 of cholera in India is characterised by an endemic situation in different states with some fluctuations. There were two peaks in this phase – 1927-31 and 1941-45. El tor cholera began to be detected in India in 1964.

Epidemic increase of cholera cases in 1924 occurred simultaneously in Bengal, Bihar, Orissa, Assam and United Provinces (UP) as early as March-April. With the onset of monsoon, cases peaked in Bihar and Orissa. Punjab was affected in July but the epidemic did not proceed to North Western Frontier Provinces.

1925 and 1926 were not epidemic years in India and cholera remained generally confined to Bengal. Meanwhile from Punjab, cholera spread to Kashmir in April 1925, where the epidemic continued till October with North Kashmir province being the worst affected. In south India, the months of January in both the years (1925 and 1926) reported maximum cases; the infection spread to Ceylon as well.

Cholera began to rise again since April 1927 in Bengal and Madras Presidencies followed by the United Provinces, Bihar and Orissa. Soon it spread to Punjab, Central Provinces, Hyderabad and Bombay Presidency. November 1927 and March 1928 recorded peaks in Bengal and cholera affected Bihar, United Provinces and Punjab earlier than the usual months in 1928. Madras Presidency was affected in June 1928, with south-eastern districts being the worst affected. 90% of the deaths in 1928 were reported from United Provinces,

[2] She linked famine conditions to prices, but at the same time acknowledging its limitations

Bihar, Orissa, Bengal and the Madras Presidency. During 1930, cholera remained at high levels in Bihar, Orissa and United Provinces but Bengal registered a decline. This epidemic began in Bengal and by April had affected the United Provinces (and Bihar and Orissa). It also spread westward into the Punjab. The epidemic of 1927 lasted in western India (Bombay and Central Provinces) till 1931, though at a lower level of deaths.

Cholera declined sharply in 1932 and a further decline in 1933 was of particular interest considering the fact that the Kumbh Mela[3] was held at Hardwar after a period of 12 years. An epidemic in Bengal, Bihar, Orissa, United Provinces and Central Provinces was responsible for the peak in 1934-35.

The 1938 Kumbh Mela at Hardwar has been considered to play a major role for epidemic situation in Punjab and other parts of northern India. Though the mela started in February, cholera cases began to rise only after summer set in during April. The first case was reported from Punjab on 9th of April. In the next week, 11 districts upto Lahore were affected and 16 other districts in the week after that. From there it took just one more week for cholera to reach North West Frontier Provinces.

In Punjab, there were 70,622 deaths against 6,341 deaths in the previous year. Bengal also reported a large number of deaths in 1938 – 71 ,133 deaths in contrast to 32,700 in 1937. Central Provinces reported 45,332 deaths whereas there were only 1,107 deaths in the previous year. The Public Health Commissioner of Government of India chose not to blame the Hardwar Mela, perhaps because the administration was already cautious of taking anti-religion positions in the 1930s (Arnold, 1993); the epidemic was attributed to floods. The fact remains that floods occurred in Bengal in October 1938 (Pollitzer, 1959)!

Cholera cases were reported from the Ganges valley during 1941 from where the disease spread further to Punjab, Sindh and North West Frontier Provinces. 1942 also showed a similar pattern. 47% of the deaths occurred in Bengal. Madras (25%), Bihar (11%) and

[3] There were only 1,915 deaths – the lowest ever figure in the series of Kumbh and Ardh Kumbh Fairs from 1897-1948 (Bannerjea, 1951).

Assam (3%) were the other affected provinces. In contrast to the general trend, cases in Bengal did not decline during the monsoon (July-August); instead they continued to rise and reached a peak in October. The tail of the epidemic continued to 1945.

The 1941-45 epidemic coincided with the second World War and has been attributed to severe famine conditions in Bengal and food scarcity in other areas. Famine was precipitated by serious shortage of food on account of war; food meant for civilian consumption was stockpiled for the army. Another factor of serious concern was that civilian medical officers had joined the army in large numbers thus leaving gaps in the health services infrastructure. Further, medical and sanitary stores were diverted to the army (Pollitzer, 1959).

Since 1948, cholera in Punjab showed a consistent downward trend. There were epidemics in Bihar in 1950 and 1952-53, in Madras in 1948 and 1950 and in Uttar Pradesh in 1948 and 1952-53.

There were epidemics in Bengal in 1948-50 and thereafter there was a general decline. The mean annual death rate declined steadily from 1891-1915, when it ranged between 2 and 3, to about 1 in 1935 and thereafter to 0.29 by 1955. District level analysis showed that the reduction was slowest in the endemic districts (Pollitzer, 1959).
Incidence rate and death rate was at a low of 3.68 and 1.99 per 100,000 persons respectively in 1955. Incidence rate registered an increase in 1963 to about 12.66 per 100,000 in 1963 (Patnaik and Kapoor, 1967). The first El tor case was reported from India in 1964 (Barua, 1974). The seventh pandemic caused by *Vibrio cholerae* El tor had begun in 1961 in South East Asia and by 1963 had begun to affect countries of mainland Asia.

Regional Patterns

Between 1910-1954, five states – Bengal (29%), Bihar and Orissa (25%), Uttar Pradesh (17%) and Madras (14%) – accounted for 85% of the deaths (Patnaik and Kapoor, 1967).

Russell (1928) in his presentation at the Conference of the Far Eastern Association of Tropical Medicine in Calcutta divided Indian provinces on the basis of cholera mortality :

(a) Assam, Bengal, Bihar, Orissa and the United Provinces ~ where the average incidence was high and variation was less from year to year.

(b) Bombay Presidency, Central Provinces, Punjab and the North West Frontier Province (NFWP) ~ sudden peaks of cholera occurring at infrequent intervals. Infection was not endemic in these areas and was imported from other areas from time to time.

(c) Madras Presidency ~ the Northern and Central groups of districts were epidemic areas while the Southern group was an endemic area.

Pollitzer (1959), however, classified the Indian states into two groups on the basis of prevalence of cholera.

(a) Regions where epidemics occurred occasionally and remained free from infection for considerable periods. These included Punjab, Delhi, United Provinces (Uttar Pradesh), Central Provinces (Madhya Pradesh), Hyderabad, Mysore and Bombay.

(b) Areas where the disease was maintained at a high level (endemic) and at times assumed epidemic proportions. These consisted of Bengal, coastal Orissa and some districts of Assam and Bihar.

Swaroop (1951) computed average mortality rate from cholera for 10 years with lowest mortality, for 1901-1945, and used the magnitude of this average as a rough measure of endemicity. According to his observations, the principal regions in which cholera was highly endemic in India were :

(a) Delta region in lower Bengal – formed by the rivers Hooghly, Damodar and Rupnarayan

(b) At or around the confluence of the rivers Ganga and Brahmaputra and Meghna in east Bengal

(c) In the deltaic zone of the four rivers of Orissa – Mahanadi, Brahmani, Baitarini and Subarnarekha.

(d) Northern Assam and Brahmaputra valley.

(e) In Patna and Gaya districts of Bihar where Gandak, Sone and Gogra rivers join the Ganga.

(f) In the low-lying river basins of United Provinces (Uttar Pradesh), beginning from the junction of the Ganga and Jamuna at Allahabad and extending through Varanasi to the points where the Gomti and the Sone join them.
(g) In Madras Presidency, in the Cauvery Delta
(h) In Madras Presidency, in the delta formed by Krishna and Godavari rivers around the Colair Lake.

Swaroop (1951) noted several characteristics of these endemic belts ~

- located generally around rivers
- areas of high population density
- low-lying land tracts, not more than 500 feet above sea level
- areas of high absolute humidity and rainfall

Cholera generally spread from Bengal through United Province (Uttar Pradesh) and Punjab north-westwards to Afghanistan. This route of spread was described by Bryden (1874) as the 'northern epidemic highway'. Similarly a 'southern epidemic highway' was also described along which the disease spread from UP through central India southwards to Madras and Bombay.

Seasonal Trends

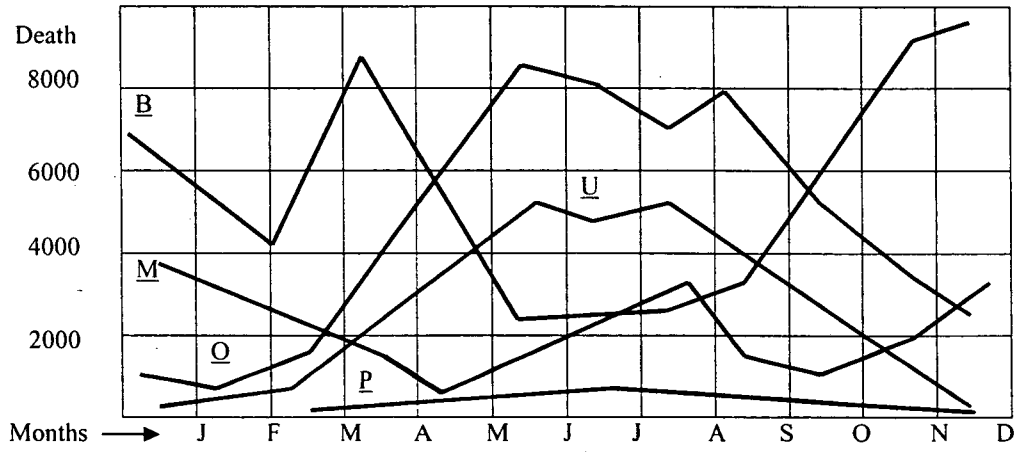
Pollitzer (1959) noted shift in seasonal trends in cholera as one proceeded westwards from Bengal, the endemic home of cholera, on the basis of mortality figures between 1900-20. He showed that cholera peaked in Bengal between September and January followed by a second peak during March-April and incidence fell to a minimum during the months of June, July and August. As the dry season set in cholera appeared in a mild epidemic form in Bengal. But storms and tidal waves that brought saline water inland were associated with cholera epidemics, as in 1876 and 1897. There was, however, considerable variation within different regions of Bengal. Winter was found to be unfavourable for spread of cholera in Bihar and United Provinces (Uttar Pradesh). Seasonal peak was reached in these two states in spring. In

Punjab, peak incidence was during the monsoon (July and August). In Madras Presidency, there were two cholera peaks that were co-related to south-west monsoons in June, July and August and north-eastern monsoons during November and December.

Fry (1925), on the basis of district level data, demonstrated that change in seasonal incidence patterns took place in Bhagalpur Division of Bihar between Rajshahi and Patna. Purnea District was shown to have peak incidence during spring; for Bhagalpur that lay further west, summer and rainy seasons were the peak period. He concluded that not only did the seasonal pattern change in the Bhagalpur Division but the endemic pattern of cholera in East Bengal gave way to an epidemic pattern.

Graph 3.1 is based on the mean monthly cholera deaths for five Indian provinces and represents the seasonal trends.

Graph 3.1 : Mean monthly cholera deaths for selected provinces, 1925-1946



Province Code: B-Bengal. M-Madras. O-Orissa & Bihar. P-Punjab. U-United Provinces
Source : Pollitzer, 1959

Seasonal variation is of importance as epidemics originating in Bengal generally spread westward upto Bihar or Uttar Pradesh during the same year, subsiding during winter. The westward progress resumed only during the following year. This time table was generally followed unless the occurrence of epidemics in fairs or festivals accelerated the progress of

the disease. Following the recrudescence in United Provinces each year it followed the land route to Punjab.

Macnamara (1876) took a firm stand against the notion of the early researchers that prevailing winds exerted a direct influence in propagating cholera infection. He observed that, "cholera will not extend during the cold of a European winter, or even of our Punjab cold season." He emphasised the role of atmospheric moisture and rain. He postulated linkage between rain and cholera (in Bengal) – "cholera is at its height ... every year in March and April and again in September and October, and these are the very months in which we get heavy downpours of rain, washing the surface soil and its contents into wells and tanks from which we procure our drinking water; these storms are generally followed by intensely hot days. As soon as the regular rains set in, and we get a more or less continuous downpour for some three months, cholera ceases for the time, and in fact until the close of the year, when it breaks out again in the stormy weather which, with intervals of intensely hot days, succeed the rain."

Pollitzer (1959) argued that in north-western parts of India, including Punjab and United Provinces, moisture laden south-west monsoon promoted spread of cholera, that was imported through the boats sailing up the Ganges. Drought years were also equally prone to cholera. Survival of the *V. cholerae* showed a decrease rather than an increase *pari passu* with an increase of ambient temperature. Other factors were therefore obviously responsible for the increased prevalence of cholera during the summer months.

Flugge (quoted by Pollitzer) had postulated in 1893 that hot weather led to an increased consumption of raw water, other cold drinks, cold foods like fruits, salads and jellies – all of which were liable to be contaminated with *V. cholerae*. Lowering of the level of rivers, other surface water and groundwater in wells that occurred in late summer was also critical since if those waters became contaminated with *V. cholerae* they were liable to maintain a high level of vibrios which thrived better in concentrated water containing large amounts of organic matter.

Rogers (1928) studied the relationship between cholera and seasonal trends and concluded that rainfall and relative humidity could not explain the epidemiological pattern of the disease. He observed that cholera was minimum during south-west monsoon in Assam and Lower Bengal but maximum in Punjab during the same season. He argued that mean temperature showed a closer relationship to cholera incidence as cholera was at its minimum during winter in Punjab, United Provinces, Central Provinces and the Deccan area but not so in Lower Bengal. Russell (1928) was of the opinion that relative instead of absolute humidity was of epidemiological importance. At the same time he also stressed that the "clue to the cholera problem was not to be found in any individual climatic factor." Koch (Pollitzer, 1959) laid emphasis on the issue of rainfall being a crucial determinant of cholera. The rains washed the cholera infected faeces into tanks or ponds thereby contaminating the drinking water source.

Impact of Travel

Caravans, railway, ships and the travellers have been traditionally blamed for spreading cholera. Punjab remained relatively unaffected by cholera among the Indian provinces. The first reliably authenticated infection occurred in Punjab in 1820. In the next thirty five years, there were only five epidemics – in 1827, 1845, 1852 and 1855. Railway traffic was opened in Punjab in 1860. Large epidemics followed in 1861, 1862, 1865, 1867, 1869, 1872, 1875, 1879 and 1881. The population remained the same, and the meteorological conditions did not change; only the traffic with the cholera focus in Bengal was accelerated.

Whenever water and sanitation was taken care of, cholera reduced among the travellers accessing various modes of transport. Koch (reported by Pollitzer, 1959) observed in 1885 that, "the introduction of sanitary improvements, particularly the provision of pure waterworks water in place of *Hooghly* water in 1874 led to a marked decrease of the cholera incidence on ships leaving Calcutta with *coolies* (seasonal labourers) for Assam and other ports." It is interesting to quote Napier (1946) in this context ~ "...that normal railway travel on business or pleasure does not tend to spread the disease to any great extent on account of

the control that can be exercised over passengers, and that, though the sanitary arrangements are far from perfect, especially at the small stations, there are latrines and a safe water supply. Such travellers of course come from all grades of society, but even the poorest are seldom destitute, and the fact that they are travelling usually indicates that they can afford the ordinary necessities of life."

Cholera and the Military

In British India, cholera was one of the most important causes of sickness and mortality among the troops. Main causes of death among British soldiers in India, as reported by the Royal Commission on the Sanitary State of the Army in India in 1863, between 1830 and 1846 were dysentery and diarrhoea (32%) fevers (23%), diseases of liver (10%) and cholera (10%). In contrast, only 6% of European soldiers died in combat or of wounds. Simultaneously, troop movements were held responsible for spread across rural India. Large scale troop movements from Bengal to northern India was considered to have facilitated spread of the epidemic of 1917. During 1857-58, there was a repetition of this pattern on account of the Sepoy Mutiny. In the aftermath of the Mutiny, cholera posed a serious threat to the well being of European soldiers on whom the colonial rulers were heavily dependent. Cholera became the preoccupying consideration of the new sanitary commissioners in the 1860s. Investigations into cholera (and venereal diseases) established that sanitary and medical security of the military could not end at the barrack gates. Such measures had to be extended into adjacent urban and rural areas and to the indigenous general population. After 1861, cholera in cantonment areas and military camps was generally prevented and this has been considered to be a significant (if underlying) factor in the increased security of British power in India.

Cholera and Pilgrimages

Widespread dissemination of cholera pandemic that had its origins in the Indian subcontinent became a symbol of fear for the West. For the first time, Moreau de Jonnes in 1831,

identified religious pilgrimages and troop movements as two of the critical determinants for the spread of cholera in India. Faeco-oral transmission of *Vibrio cholerae* through reservoirs and watercourses, that provided water for drinking, washing and bathing, was the main source of infection in India. Mass bathing at the Kumbh and Ardh Kumbh melas at Hardwar and Allahabad and sipping of water as part of the ritual for worship and religious purification provided efficient routes of transmission. The practice of pilgrims bringing back Ganga water and ritual sipping of that water by relatives and friends ensured propagation of the infection. Congregation of people has consistently been considered a warning signal in the literature on cholera. What has been overlooked is the fact that safe water and sanitary measures could ward off such risks.

The Kumbh Mela at Hardwar in April 1867 was attended by nearly 3 million people. Only 19 people were treated for cholera during the mela. The disease was considered to have actually spread during mass bathing on the 12^{th} of April and was disseminated across northern India by the dispersing pilgrims. Similarity with famine conditions have been drawn in view of the poverty of the pilgrims, the conditions under which they travelled and their crowding together in insanitary conditions. Moreover, tanks and wells along routes of pilgrims also infected towns and villages. Nearly a quarter of a million people have been estimated to have suffered from the disease in this epidemic and about half of them died (Arnold, 1993).

Bannerjea (1951) demonstrated that, overall, average death rate from cholera in years during which no Kumbh or Ardh Kumbh fairs were held was 1.6 times lower than that during festival years. 116 fairs with a total gathering of about 250,000-300,000 were held in UP (most of them in eastern districts) during March and April, when meteorological conditions were favourable for the spread of cholera. Kumbh and Ardh Kumbh fairs were estimated to attract nearly three and two million people respectively at Allahabad and about one million and half million respectively at Hardwar. Bannerjea (1951) reported cholera deaths in Kumbh and Ardh Kumbh Fairs at Allahabad and Hardwar from 1879-1948 (Table 3.1)

Table 3.1 : Cholera Deaths in Kumbh and Ardh Kumbh Fairs, 1879-1948

Hardwar			Allahabad		
Year	Fair	Deaths	Year	Fair	Deaths
1879	Kumbh	35892	1882	Kumbh	89372
1885	Ardh Kumbh	63457	1888	Ardh Kumbh	18704
1891	Kumbh	169013	1894	Kumbh	178079
1897	Ardh Kumbh	44208	1900	Ardh Kumbh	84960
1903	Kumbh	47159	1906	Kumbh	149549
1909	Ardh Kumbh	21823	1912	Ardh Kumbh	18894
1915	Kumbh	90508	1918	Kumbh	119746
1921	Ardh Kumbh	149667	1924	Ardh Kumbh	67000
1927	Kumbh	28285	1930	Kumbh	61334
1933	Ardh Kumbh	1915	1936	Ardh Kumbh	6793
1938	Kumbh	70622	1942	Kumbh	7662
1945	Ardh Kumbh	77345	1948	Ardh Kumbh	52604

Source :Bannerjea, 1951

Cholera in United Provinces (based on cholera death rates) generally increased during years in which Kumbh and Ardh Kumbh fairs were held. In some instances, rise in cases was during the year following the fair; as during 1880, 1892, 1910, 1913 and 1938. There were however exceptional years when fairs were held but incidence of cholera was low, as during 1933 and 1942. A similar exception occurred during 1888, at a time when cholera prevention measures were not well developed in the fairs. It is also important to note that in 1887 and 1908, cholera mortality in UP was particularly high despite the lack of religious congregations. While these figures establish the association between the religious fairs and a consequent increase in cholera mortality rates it also indicates that not each of these peaks is attributable to the fairs. In fact some of these increases have been attributed to the increased incidence during those periods in Bengal, from where the infection is likely to have been imported. Generally Punjab was also affected alongwith UP. However, epidemic rise of cholera mortality in Punjab in certain periods do not correlate with fairs. Rather they can be explained with the argument of Zurbrigg (1992) that food scarcity was associated with increase in deaths due to epidemic diseases as during epidemics of cholera in Punjab in 1901, 1906 and 1908.

To a lesser extent than the Kumbh and Ardh Kumbh Melas, the temple of Jagannath at Puri, pilgrimage sites at Nasik and Pandharpur in Maharashtra, Tirupati in Andhra Pradesh and Kanchipuram at Tamil Nadu have all been implicated at different times as being sources of epidemic cholera. The location of Hindu religious places along rivers and the periodicity of the major festivals contributed to the epidemicity of cholera in India. Initially, colonial reports on cholera did not look for associations between the disease and pilgrimages.

The International Sanitary Conference at Constantinople in 1866 made particular efforts to protect Europe from cholera (originating from Asia). Taking a strong contagionist line, reference was made to Hindu pilgrimages (with particular mention of Puri) and Haj congregations of Muslims at Mecca (as a second stage of relay). Sanitary commissioners, appointed in the aftermath of the Conference, were asked to investigate the causes of cholera epidemics. The Government of India (by then, directly under the Crown) instituted inquiries into the sanitary state of major pilgrimage and festival sites. Drawing on the case of the Hardwar outbreak of 1867, the reports pointed to a close association between cholera and pilgrimage. Epidemiological association between cholera and Hindu pilgrimage gave rise to expected antipathy among ruling classes.

Dr. David B. Smith, Sanitary Commissioner of Bengal, made scathing observations on the public health scenario of Puri. Similar reports emanated from other parts of India as well. The medical and sanitary assault on cholera took up an anti-Hindu stance. It not only condemned the pilgrims but also accused the Hindu priests of compromising on public health standards for pecuniary interest. In 1916, the Sanitary Commissioner to the Government of India recommended that preventive actions should be taken at fairs and also along the routes that the pilgrims took to the fair including major railway stations.

The pilgrims were categorised as a "dangerous class" who required special surveillance and regulatory measures. In the scientific/medical paradigm, this high risk approach made it appear possible to control cholera by controlling those most obviously implicated in its transmission. Asia's poor pilgrims were held at par with the slum dwellers of the industrial towns of Europe as far as the reservoir and transmission of the disease was concerned. Strict

control over pilgrims and pilgrimages including quarantines were instituted to protect cantonments and municipalities. Pilgrim tax was recommended to pay for additional sanitary measures. This was not implemented for the obvious political fallout, particularly after the promise of religious toleration of Queen Victoria's proclamation in the aftermath of the 1857 Mutiny. Pilgrimages thus came to be tolerated as a "necessary evil" with the hope that with the spread of western education and ideas, pilgrimages would lose their traditional appeal and Indians would value sanitation and hygiene. In practice, therefore, sanitary surveillance at the pilgrimage centres was tightened. J. M. Cunningham, Government of India's sanitary commissioner, with his anti-contagionist views, opposed the quarantines and sanitary cordons as he considered that they would not be effective.

In 1892, the Government of the North Western Provinces ordered the breaking up of the Mahavaruni Mela in Hardwar following an outbreak of cholera among those already assembled. Two hundred thousand pilgrims *en route* to Hardwar were turned back. This caused a pubic outcry. The proposed legislation on conferring powers on Government to disperse fairs and religious assemblies on sanitary grounds was quietly dropped by the Viceroy.

Lal (1937) pointed out that till the beginning of twentieth century it was considered inevitable that cholera would occur at fairs and pilgrimages. The responsibility of the state was not given due consideration. There was lack of funds, expertise and institutions for this job. Public health organisation at fairs and religious congregations was invariably left to a handful of sanitary inspectors.

Haffkine tested anti-cholera vaccine in India during 1893-96. The ruling medical establishment remained cautious. As with the banning of fairs and festivals, there was an element of fear of public reaction if compulsory anti-cholera vaccination was introduced. Before 1914, the vaccinees were soldiers, prisoners and tea-estate workers. Even as late as 1930, the suggestion of compulsory inoculation of pilgrims for the Allahabad Kumbh Mela was rejected by the government. At a time when Congress civil disobedience movement was at its peak, this was absolutely a political decision. In 1936, compulsory inoculation of

pilgrims was introduced at Pandharpur. The Central Advisory Board of Health recommended to provincial and state governments in 1940 to introduce vaccination in more festival centres. In 1945 it was employed as a compulsory prophylactic measure at the Hardwar Kumbh Mela.

Cholera Theory and Sanitary Policy

The spread of the first pandemic beyond the confines of the Indian subcontinent stimulated interest in theories of causation and evolving strategies of control. European theories of aetiology ranged from electrical state of the atmosphere, operation of the climate on the soil providing the right condition for the germination of the cholera seed and contagion to transmission of cholera germ in water [Snow's theory] (Harrison, 1994).

The debate acquired fresh importance after British troops were affected severely by cholera in the 1860s. The dominant view then was that cholera was basically a 'disease of locality' like malaria. At the same time Snow's theory was gaining ground in England. While the approach in England was to devise sanitary policies, the focus in India was to impose internal and maritime quarantines.

One of the first investigators was Dr. James L. Bryden, the first Statistical Officer of the newly formed Sanitary Department. His main tasks were to establish the limits of the geographical distribution of the disease, duration of the epidemics, influence of the meteorological conditions, routes of transmission and its control. Bryden's natural history approach is to be considered in the background of the Enlightenment notion. He adopted William Farr's Liebigian nosology and statistical methods. By 1866, Farr had concluded that cholera spread through contaminated water. Bryden on the other hand believed that there were two processes operating in the causation of cholera. He likened the pathogenic organism to a seed that underwent cycles of reproduction and decay. For epidemic spreads, he hypothesised, cholera seeds were transported beyond endemic area by monsoonal air currents. This theory was carried forward by James McNabb Cunningham, the Sanitary Commissioner from 1866-1884. The localist/atmospheric theory of cholera theory became a

powerful tool against advocates of quarantine and those who sought more direct interventions for the government (in public health).

Annesley Charles DeRenzy the then Sanitary Commissioner of Punjab was a critic of Bryden's hypothesis and the consequent inaction of the government. He himself was a believer of Snow's hypothesis. In fact, Bryden never thought that improvement of water supply was important. DeRenzy opposed huge expenditure that was incurred on the implementation of the Contagious Diseases Act. He advocated that resources should be better utilised through grants to municipal commissions and improvement of water supplies in the cantonments. During 1850s, Francis MacNamara, Professor of Chemistry at Calcutta Medical School initiated, and later improved, a large water supply scheme in Calcutta. Both DeRenzy and Macnamara did not receive any credit. DeRenzy was transferred from the civil medical services to a remote station in Assam.

The 1867 epidemic originating from the Hardwar Kumbh Mela led to an inquiry. Along with Bryden the other investigator was John Murray, Inspector General of Hospitals for North West Provinces. Murray concluded that the 'germ of epidemic cholera appears to reside in the evacuations of a person suffering from the disease.' Murray also recommended strict regulation of water supply and provision of camps for the pilgrims. His recommendations were reiterated by the MacKenzie Committee commissioned by the Madras government but was rejected by the Indian government on grounds of cost. By 1870, the Indian government decided to discontinue *cordons sanitaire*. In 1873, the government of India's Sanitary Commissioner, J. M. Cunningham, realising that success of sanitary measures lay in the co-operation of the local communities, advised that municipalities must form centres from which education in 'sanitary matters should spread among the people'.

At about the same time the sub-soil water theory of the German hygienist Max von Pettenkofer had achieved international recognition. The critical factor was considered to be the presence of a porous soil and abnormally high levels of ground water. In 1870, a scheme for registration of sub-soil water levels was instituted in Madras and was later extended to other provinces. No direct linkages could be established to incidence of cholera and

opposition was encountered to this measure. It was finally discontinued by the Army Sanitary Commission in 1879. The hypothesis became obsolete with Koch's discovery of *V. cholerae* and the proof that faeces of patients served as the immediate vehicle of transmission. Koch opposed Pettenkofer at the 1885 cholera conference. Virchow (1885) and Liebermeister (1896) also opposed Pettenkofer independently. Pettenkofer's theory today remains a historical curiosity though his thesis of epidemic spread of cholera promoted by suitable local and seasonal influences remains valid.

The notions of 'contagion' dominated the scenario till 1870s. The analogy with smallpox continued to prevail. The outbreak of the disease in certain localities and the absence of cases among those who tended the sick was construed as evidence against its communicability. In fact till the first half of 1880s, official medical doctrine in India continued to diverge from prevailing opinion in Britain. The German Cholera Commission with Robert Koch arrived in Calcutta in December 1883, after working for several months on cholera in Egypt.

The 'Egyptian' bacillus was isolated from a water tank; several people who regularly drank water from that source had been diagnosed to be suffering from cholera. While Koch demonstrated the comma bacillus, he failed to establish causality. What he demonstrated was a mere association between the presence of large numbers of the bacterium in the intestines of cases and its absence in persons free from the infection. Further, there was no conclusive evidence based on animal models. The thrust of research however shifted from the locality to the laboratory. Germ theory continued to gain acceptance amidst dissenting opinions.

Sir Joseph Fayrer, surgeon-general at India Office Council in London argued that proof of cholera's contagious character would imply strict international regulations, including quarantine, over India's overseas trade. A British two-member commission dispatched to Calcutta in 1884 ruled that Koch's bacillus was innocuous and could not be the cause of cholera. The Government of India stressed on controlling of local factors. Cholera inoculation developed by Haffkine also experienced limited success (Arnold, 1993).

Following Gaffky's analysis of the cholera epidemic of Hamburg, the critical role of water was firmly established. In 1894, Metchnikoff established causality by drinking water containing comma bacilli and inducing cholera in himself and, also produced the disease in rabbits. He succeeded where Koch had failed.

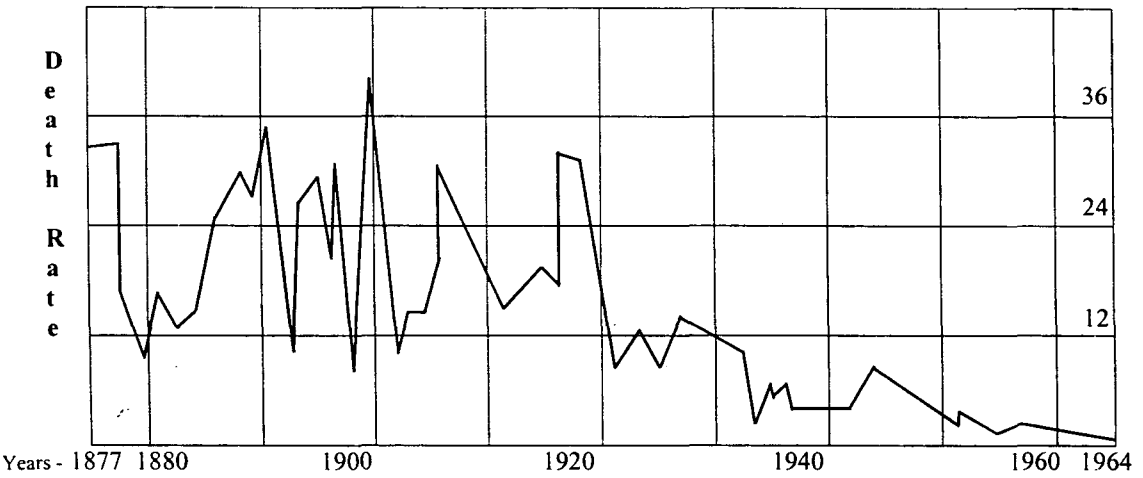
Meanwhile British India as the 'factory of cholera' was subjected to international censure and the plague epidemic of 1896-97 (Bombay Plague) put further pressure on the British administration to improve public health measures. The International Sanitary Conference in Venice called for a more interventionist role of the government. At about the same time bacillus theory gained more credence. Medical researchers and advisors argued the importance of climate, physical environment and social behaviour as contingent factors. There was this 'Orientalist' assumption that India was essentially different from Europe; those with long experience of the field in India could only comprehend its peculiarities and idiosyncrasies. The environmentalist school converged with their contagionist opponents in condemning the climate, the soil and the people of India for being responsible for cholera.

By 1890s, contagionist theory finally gained official acceptance; the state and its health officers opted for more active policies towards control of cholera. By the early twentieth century there was general agreement among public health authorities in India on the relevance of specific measures against cholera (particularly, water supply) but merely as part of a general sanitary programme.

Decline of Classical Cholera

The annual cholera death rate (per 10,000 population) between 1877 and 1964 has been presented in Graph 3.2 as reported by Pollitzer (1959) for the years 1877-1954 and Patnaik and Kapoor (1967) for the period 1955-64.

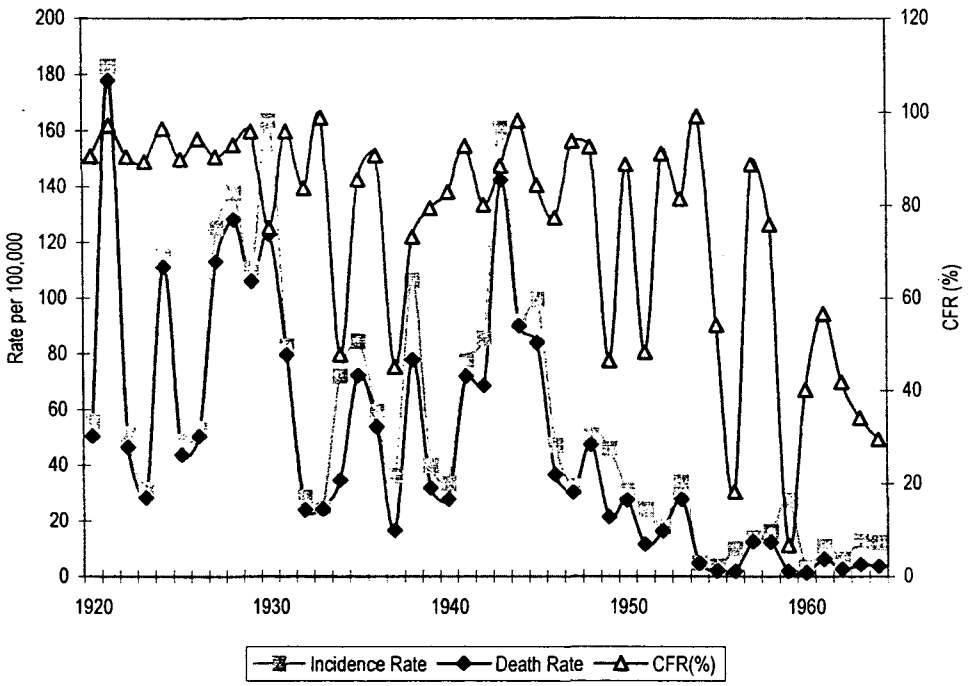
Graph 3.2 : Cholera Mortality Rate (per 10,000 population) : 1877-1954



Source : Pollitzer, 1959 (1877-1954) and Patnaik and Kapoor, 1967 (1955-1964)

Graph 3.3 presents incidence rate, death rate and case fatality rate of cholera in India during 1920 to 1964.

Graph 3.3 : Incidence Rate, Death Rate and Case Fatality Rate of Cholera in India, 1920-64



Data on reported cases and deaths is available for this period (Patnaik and Kapoor, 1967 and Bhatty, 1969). Data on population and decadal growth rates were available for the census years 1911, 1921, 1931, 1941, 1951, 1961 and 1971 (Census, 2001). Population figures for the intervening years was calculated by applying the decadal growth rate to the population of the corresponding census year. Incidence rate and death rate have been calculated per 100,000 population and case fatality rate (CFR) has been expressed as a percentage. The time trends and the decline of classical cholera will be examined in the light of the above two graphs.

Patnaik and Kapoor (1967) identified three distinct phases in the time trends of cholera in twentieth century India, from 1900-1963, before the onset of the seventh pandemic in 1964. They are represented in Table 3.2.

Table 3.2 : Epidemic Years and Average Inter-Epidemic Interval of cholera 1900-1963

Years	Epidemic Years	Average Inter-Epidemic Interval
1900-24	1900, 1903, 1905-06, 1908, 1910, 1915, 1918-19, 1921 and 1924	1.75 years
1925-47	1927-30, 1935-36 and 1941-45	2.25 years
1948-63	1948, 1953, 1957-58 and 1963	3.25 years

Source : Patnaik and Kapoor (1967)

The time trends of cholera in India correspond well with the global pandemic till the 1920s when the sixth pandemic came to a close. There was a sharp decline following the 1877 epidemic, during the fourth pandemic. The fifth pandemic began in 1881; the rising trend of 1879-80 has been explained by the increase in cases in United Provinces following the Kumbh Mela at Hardwar in 1879. Following the decline of this phase there was a rise in cases during 1885 possibly due to Ardh Kumbh Fair at Hardwar. The rising phase from 1889[4] and the peak in 1900 marks the beginning of the sixth pandemic; its resurgent phase is

[4] In 1891, there was a Kumbh Mela at Hardwar where 169,013 persons died and the epidemic spread to the United Provinces.

evident in the sharp rising trend of 1905-08. Cases increased in U. P. and Punjab in 1913 following Allahabad Ardh Kumbh in 1912; there were relatively fewer cholera deaths (18,894) during the Mela itself. There was a steady decline from that point till 1918 when following the Kumbh Mela at Allahabad there was a rise of cases in U. P. and Punjab. 1921 Ardh Kumbh Mela at Hardwar witnessed 149,667 deaths in the Mela itself and spread to adjacent areas of Punjab and U. P. resulting in a sharp rise of cases (460,000) and deaths (446,817) with a case fatality rate of more than 97% for the country as a whole. The sixth pandemic ended in 1923 (Graph 3.1)when cases and deaths were at the lowest of 80,000 and 71,504 respectively.

Though some of the epidemic increase in cases have been sought to be correlated in literature with Kumbh and Ardh Kumbh Fairs as has earlier been explained all fairs did not inevitably result in epidemics. There are several instances when despite fairs, there were no epidemics at all. Further, drawing on Zurbrigg's (1992) argument the Punjab situation in the first decade of the twentieth century can be correlated with overall food scarcity. Moreover, by the second quarter of the twentieth century, when the Independence Movement had gathered full steam, the colonial medical administrators refrained from associating Kumbh with the 1938. While undoubtedly some of the Fairs are associated with epidemics the fact remains that colonial medical history over-emphasised their role. Further, Melas resulted in epidemics because public health and sanitation was neglected for lack of funds, expertise and institutions (Lal, 1937).

Barua and Greenough (1992), commenting on the global situation of cholera during 1926-1960 observed that :

- cholera disappeared from Europe after 1925
- cholera, however, did not remain confined to the Indian subcontinent
- cholera kept occurring regularly in Asian countries
- the origin of the 1947 epidemic of Egypt remained undetermined but was not caused by pilgrims from Mecca nor by British troops returning from India

There was a general decline in the all-India trend; each successive epidemic upto 1938 appeared to reduce in magnitude, except in the case of the epidemic (1941-45) during the World War II. After 1919, cholera incidence during the inter-epidemic periods also became lower. Individual provinces, however, showed considerable heterogeneity in length and severity of epidemics.

In Europe and North America, cholera was endemic in industrial cities and urban slums. In India, cholera by and large remained a disease of the rural poor. During 1860s and 1870s, the model of sanitary reform in Britain was implemented to some extent in the major Indian cities, particularly with respect to improved water supplies. These made an impact on urban mortality but the slum dwellers received marginally little benefit from these initiatives. Between 1841 and 1865, the annual reported deaths due to choler in Calcutta ranged from 2,500 to 7,500. Sewage system started in Calcutta in 1865, followed by filtered water supply in 1969. Cholera deaths dropped sharply. Despite rapid expansion of the city, in the second half of the 19th century the annual cholera mortality figure reached 3,000 only once during 1895 and otherwise kept well below that level. Similar benefits were observed in Bombay and Madras. Despite these advances, in 1877 when cholera epidemic and famine coincided in Madras Presidency in 1877 (as mentioned earlier), and the rural poor migrated to Madras city in large numbers, urban mortality reached the highest levels. Piped water supply and sanitation did not improve in small towns and more so in villages; cholera therefore continued to affect these settlements.

The absence of major famines has been considered to be crucial to the reduction of cholera. There were no major famines after 1908. Cholera also gradually declined thereafter only to peak again with the Bengal famine during the Second World War. Cholera mortality began to decline in the early decades of the twentieth century. Population was growing and there was no evidence to suggest any remarkable socio-economic improvement for the majority of the people. It has been hypothesised that sanitary precautions and medical interventions, rather than a better standard of living or improved socio-economic standards, is the most plausible explanation of the decline. It must also be remembered at the same time that, globally, the sixth pandemic was on the decline and the declining trend in India should be considered in

consonance with that trend. After 1923, public health services were expanded or established in various parts of India, with efforts to primarily control epidemic disease. It is important to note that cholera disappeared from Europe at about this time (Barua et al, 1992), following the development of sanitary engineering services. Cholera declined in most countries of the world, including Asian nations, in mid-1920s thereby marking the decline of the sixth pandemic.

Harris (1913) reported that the provision of piped water supplies in the towns of the United Provinces led to a great reduction in cholera. He, however, added that the fact that cholera was not eliminated completely was because infections continued to be disseminated through unsafe sources of water supply and movement of pilgrims. The provision of pipe water supplies to towns in Bombay State resulted in a sharp decline in the incidence of cholera (Benjamin, 1949). Cholera prevailed in areas where the provision of water supplies was not adequate or communities were dependant on unsafe sources of water[5]. Incidence of cholera reduced sharply in district towns of Bengal where water supply was provided as compared to the rural areas of the corresponding districts (Subrahmanyan 1951).

Pollitzer (1959) pointed out that decline of cholera through the first half of the twentieth century was also the story of declining deaths. Arguing against the prevailing hypothetical notion that the decline was on account of reduced virulence, he attributed this phenomenon to several extrinsic factors and outlined them as follows :

- improvement of case reporting system
- detection of clinically mild cases
- prompt hospitalisation of those with severe dehydration and shock (cholera gravis)
- development of 'herd immunity'[6]

[5] Free provision of water to the entire population through public standpipes was able to eliminate cholera from every state of Malay Peninsula

[6] The notion of herd immunity is a controversial one; it was generally agreed by different authors of the period that herd immunity acquired through an epidemic protected for 3-4 years. It was argued by others that while an individual outbreak may fail to produce good herd immunity, that immunity may eventually be acquired through repeated cholera infections.

Case fatality rate (CFR%) has been calculated on the basis of reported cases and deaths (Patnaik and Kapoor, 1967 and Bhatty, 1969). Data constraints have been pointed out earlier in the chapter. Cholera statistics (and reporting) in India focussed on deaths and mortality rates due to cholera. Available data on cholera in Europe suggest that CFR did not exceed 50%. On the contrary, CFR calculated on the basis of available data in India (1920 onwards) suggest a very high CFR till the 1950s. Importantly, examining trends, it is observed that the declining trend begins during the mid-1950s. The decline was sharp and CFR reduced from nearly 89% in 1955 to 34% in 1963.

THE SEVENTH PANDEMIC

The outstanding feature of the current seventh pandemic of cholera is that its causative agent is *Vibrio cholerae*, biotype El tor. El tor was first isolated by Gottslich in 1905 and the claim of its identification with classical *V. cholerae* was overruled for two reasons:

- its haemolytic activity
- failure to establish association between these strains and clinical cholera or even diarrhoea; all such strains were recovered from wells, surface waters or stools of 'healthy' pilgrims returning from Mecca

The decision to absolve *V. cholerae* El tor from choleragenic activity was internationally accepted and continued to prevail in spite of repeated outbreaks (with El tor as the pathogen) that were clinically indistinguishable from cholera. The most conspicuous of these outbreaks and the ones that have a direct relationship to the present pandemic were the four outbreaks recorded in the island of Sulawesi (Celebes) in Indonesia in the years 1937-38, 1939-40, 1944 and 1955-57. The recurrence of these outbreaks in Sulawesi and the repeated recovery of this vibrio from the stools of persons with mild diarrhoea marked the island as an endemic focus for *V. cholerae* El tor infection.

El tor disease began to upsurge in an epidemic form in Macassar, its usual focus. Soon it spread to central and northern areas of the island. It spread to Java in May, thus flagging off

the first trip of the current pandemic. Pandemic, by definition, is a continuous show or record of sequential dissemination of a disease globally. The seventh pandemic of cholera is considered in three distinct geo-chronological phases which are (Barua and Greenough, 1992):

- First Period (1961-62): affecting all the states of South East Asia
- Second Period (1963-1969): affecting the states of mainland Asia
- Third Period (1970 to date): Middle East-Afro-European phase

Phase I

It is generally accepted that the January 1961 outbreak in Sulawesi was the precursor of the seventh pandemic of cholera. From there it spread to Java in May and Sarawak in July. By August, the disease was reported in Kalimantan (Borneo), Macao and Hong Kong. The Philippines experienced its first batch of cases in September and Sumatra and Timor were infected in November. Incidence of cholera in these territories in 1961 was reported to be 12,197 cases with 1,969 deaths and the causative agent in all these cases was *V. cholerae* El tor. In view of the prevailing situation, the World Health Organisation (WHO) convened a Scientific Group on Cholera Research to study the problem. Noting that the disease caused by El tor was clinically indistinguishable from that caused by *V. cholerae* classical, the Group recommended that cholera should be defined in the International Sanitary Regulations as including cholera El tor. This was subsequently ratified by the 15th World Health Assembly in April 1962.

The pandemic continued to spread further and by end-1962 all the states of South East. Asia were affected - Sabah in January, Taiwan in July and Irian Barat (formerly West New Guinea) in October. Incidence of El tor cholera during 1962 was 13,393 cases with 1,977 deaths. Sea borne spread of the infection has been hypothesised to be consistent with geo-placement of the affected territories. Initial cases in the concerned countries were in coastal areas or among fishermen/boatmen, who also showed a higher carrier rate than among the contacts of cases living permanently on land (Kamal, 1974).

The most critical question is why the disease began to spread outside Sulawesi (which was already an endemic area) in 1961 and not earlier. For cholera, a disease with man as its only 'source-vector', the factors necessary for pandemic flare ups are:

- volume of infection (the degree of prevalence of the disease) in the endemic area
- number of persons leaving the endemic area
- susceptibility of populations with whom these persons come in contact during the journey and at their destination.

The 1961 outbreak in Sulawesi had spread to different sections of the island. There was large scale troop and civilian movements within Indonesia; emigration was also on a higher scale than before. Adjacent South East Asian countries were free from cholera for at least a decade and a half. So, when the infection was introduced, it established a foothold in the susceptible population. At the same time, economic restructuralisation was going on in these countries with large scale introduction of mechanised high sea transportation and travel. The countries affected in 1961 reported an additional 11,611 cases and 1,457 deaths in 1962 and the pandemic was on its way to the Asian mainland in 1963 (Barua et al, 1974).

There were several microbiological characteristics that facilitated the spread of El tor strain. The proportion of mild and subclinical cases was high. Ratio of severe cases to mild or subclinical cases was reported to be 1:5 for classical cholera and 1:25 to 1:100 for El tor cholera (Masley, 1970). Carrier state also lasted longer, resulting in spread of inapparent infections. Further, viability of El tor outside the human body is high resulting in greater spread from infective material, than the classical variant .

Phase II

While the mainland of Asia was affected in 1963, all territories of Western Pacific Region that experienced the pandemic during 1961-62 were again infected. Malaysia was the first country to be affected in May, while all other countries of Indo-Chinese Peninsula (Singapore, Thailand, Cambodia and Burma) except Vietnam were invaded by December

1963. Vietnam joined the pandemic in January 1964. Two more El tor invasions were recorded that year – Republic of Korea in September and East Pakistan (Bangladesh) in December. It is interesting to note that all countries were invaded by ships except Cambodia where the infection was imported from Thailand by railways.

On the 1st of April 1964, Barua et al (Barua, 1974) diagnosed the first El tor cholera case in India from a patient in Calcutta. Mukherjee and Basu on the basis of phage typing of 2,970 vibrio strains isolated in different outbreaks in India during 1964-66 expressed their opinion that El tor cholera entered India in March 1964 through the port of Madras (Kamal, 1974). They opined that in both the cities (Calcutta and Madras), the infections were likely to have been carried by the sea-route by the Indian repatriates from Burma. They observed that although cholera El tor appeared in East Pakistan in December 1963, for several months it was confined to the eastern districts. Movement of refugees to India took place mostly from the western districts and was unlikely to be responsible for importing the infection.

West Pakistan was affected during the first week of June 1965 after freedom from the disease since January 1961. It is interesting to note that though Bangladesh (East Pakistan), India and Pakistan were the home for classical cholera, they responded quite differently to the El tor pandemic. In Bangladesh, the classical variety continued to dominate. In Pakistan, both biotypes continued to coexist and the classical type caused a major epidemic in 1968. In India, classical biotype was almost completely replaced by 1966. However, classical vibrio resurfaced in Calcutta, Assam and Orissa in 1968-69. Reasons for this differential behaviour is incompletely understood (Mackay, 1979).

El tor cholera began to spread westward from Pakistan. Afghanistan was affected in the first half of July 1965. In the next two weeks all four eastern provinces of Iran suffered heavily from El tor cholera. Iranian health authorities claimed that the infection was introduced by opium smugglers. Though the July epidemic in Afghanistan was a mixed one, only El tor travelled to Iran. On 21.08.1965, the USSR notified its first case and a year later Iraq became a part of the pandemic.

It is to be noted that notifications of cholera as per norms of the International Health Regulations ceased with the threat of a further westward spread. Unwarranted panic prevailed and some countries adopted measures that were reminiscent of the past centuries. The range of measures was incredible and included disinfection of mail and printed matter, prohibition of importation of tinned fruit, iron beams, carpets, teak, mineral ore etc., prevention of the entry of the westbound Orient Express with 70 passengers for more than a week, prevention from disembarkation of the passengers from a plane including a minister of health for many hours in the heat of a summer's day and finally the detention of cholera vaccine at an airport for three days because the plane sent to collect it could not get permission to land.

The result of these restrictions was the announcement of freedom from infection without adequate proof of surveillance and/or international refusal to notify the presence of the disease. Officially, a lull was reported in the spread of the pandemic from 1967 to 1969 as no new territories were affected except Laos in 1969. Some epidemiologists had expressed apprehension that such halt was unusual and not sustainable. In the history of cholera pandemics, a pandemic never retreated once it had crossed the Caspian Sea or Mecca, from where dissemination was rapid.

During 1967-69 El tor cholera continued to be reported from the previously affected areas, in many of which the disease had established endemicity. It reappeared in Malaysia and Singapore (1968) and in Hong Kong, Macao and the Republic of Korea (1969) after being quiescent in those territories for five years or more. During 1969, vigilant health authorities detected 8 imported cases in Japan and 1 in Australia.

Phase III

Africa was affected during the 1970s and the death rate was one of the highest in the world. The worst affected communities were the displaced and refugee populations in Somalia and Zaire (WHO, 1996).

Since 1973, sporadic endemic infections related to the seventh pandemic strain were reported in the United States along the Gulf Coast of Louisiana and Texas. These infections were traced to the consumption of contaminated locally harvested shellfish. Cases in other U.S. locations have been traced to consumption of shipped-in Gulf Coast seafood (Fauci et al, 1998).

The seventh pandemic reached Latin America in 1991. Beginning from the Peruvian coast in January 1991, the infection was carried by fishermen to Ecuador and Columbia. It then spread to all nations of South and Central America and also to Mexico. In the earliest affected communities, case fatality rate was to the tune of about 30% and it later dropped to an overall cumulative rate of about 1% (Fauci et al, 1998).

Exploring the patterns of cholera in Mexico during 1991-96, Boroto and Martinez-Piedra (2000) noted that cholera recurred each year during summer. Cholera incidence was 2.5 times higher in coastal areas than in interior areas. Cases typically occurred in less urbanised areas where access to safe water and sanitation was lacking. In low lying plains, hydrological conditions made it difficult to achieve and maintain high sanitation standards. The authors argued that hydromorphic soils hindered construction of appropriate sewage disposal systems. Low infiltration rates favoured overflow of soakage pits of septic tanks, specially if emptying and cleaning of soakage pits was not done regularly. Underground water was close to the surface (within 1 metre) in affected communities; thereby resulting in a high probability of groundwater contamination with faecal matter. Downstream residents were recognised as a particularly vulnerable group in this study wherever upstream pollution was high.

In a study of epidemic cholera in Ecuador, (Weber et al, 1994), the route of transmission was identified to be principally through contaminated drinking water and to a less extent through contaminated seafood. In a study of 63 cases and 126 controls, the most important vulnerability factor identified was the lack of potable water supply at the household level; in its absence communities accessed water by illegal tapping of municipal pipes or purchased

water from tankers. Storage containers with wide uncovered mouths were identified as risk factors as they were not only liable to contamination by hands but residual chlorine also evaporated rapidly. Consumption of beverages from street vendors was another critical risk factor.

Koo (1996) identified transmission during epidemic cholera in Guatemala in 1993 through *helados*[7] sold by street vendors. *V. cholerae* is not killed by freezing. 60% of *helados* samples had a pH of ≤ 4.5, the pH above which *V. cholerae* survives. 40% of the *helados* samples tested suggested the use of unsafe water.

During 1990s cholera affected the refugee camps of African nations that were ravaged with war and famine. Rwandan refugee camps around Goma, Zaire were badly affected. Cholera was reported in a major way from Romania and the Black Sea states of former Soviet Union where public health systems had broken down (Fauci et al, 1998). During the same time cholera outbreaks were reported from Karachi, Pakistan (Sheikh et al, 1997). Though peak incidence was reported from May to August and rainy season lasted from June to August, the outbreaks were observed to be independent of rainfall. The authors identified civil unrest and crumbling infrastructure as critical determinants of the outbreaks.

Spread of El tor cholera in India

We explore here the spread of the new strain to different areas of the country, the emergence of new endemic areas, time trends and seasonal trends. The various factors that contributed to the rapid spread of El tor infection inside India are considered to be (Mackay, 1979) :

- land travel as main route of spread
- ability of *V. cholerae* El tor to eliminate cases of classical biotype
- invasion of El tor cholera into areas in India which either had never been cholera-affected and/or had been free from the disease for years

[7] A food item prepared from crushed ice, similar to *chuskies* sold in the streets of Delhi and north India

- higher proportion of mild cases and carriers in El tor outbreaks as compared to the outbreaks of classical cholera
- longer persistence of carrier state for El tor strain than for the classical biotype
- longer viability of El tor outside the human body

Following the introduction in 1964, cholera spread rapidly across India. In 1965, cholera was reported from Delhi[8], Gurgaon, Surat and Madras. El tor cholera was reported from Gurgaon during July to September and spread upto to Rewari. Case fatality rate was 12.1% during the initial phase and later reduced to 3.7%. Contaminated well were identified as sources of infection (Arora and Misra, 1975). More than 1,800 cases of El tor cholera were reported from Uttar Pradesh (Patnaik et al 1967). Cholera was reported from Ahmedabad, Gujarat (Arora and Misra, 1975) and Andaman and Nicobar Islands in 1966 (Shrivastav, 1970). In 1968 , cholera was reported from Rajasthan – Sikar, Churu and Pali were the affected areas (Arora and Misra, 1975). Pal et al (1968) reported that strains isolated from Rajasthan in 1968 were similar to those isolated earlier from Delhi, indicating the spread from Delhi to the adjacent states.

In an epidemiological study of *Vibrio cholerae* El tor outbreaks in Indore city in 1968, Mittal et al (1969) observed that cases were reported from among communities without household water supply (accessing water from public taps) and sanitary latrines (defecation was being done in the open fields and to a lesser extent in public latrines). High fly index was also reported. Though flies may carry *Vibrio cholerae*, and is an indicator of poor sanitation, they are currently not considered to be vectors of importance, as far a cholera is concerned (Park, 2000).

Cholera was detected in Lucknow (Saxena, 1970) in 1969. The areas affected were densely populated, partially sewered and environmental sanitation was poor. The poorest sections of

[8] Classical cholera outbreaks were earlier reported during 1958 and 1960; routine testing of stool samples for cholera of patients admitted with acute diarrhoea in Infectious Disease Hospital, Delhi started from 1965.

the community were affected. Significant association was established between "bazaar purchased food" and cholera.

El tor cholera was reported sporadically from all municipal wards of Baroda in April-May, 1970. (Pal et al, 1972). Cholera was reported during June-August, 1972 from Aurangabad Town (Sen Gupta et al, 1974). Residents of *bustees* (slums) were the worst affected; the risk factors identified were inadequate water supply and improper drainage and sewage disposal.

Kashmir was affected in 1971. Simultaneously, in 1970-71, there were reports of classical cholera outbreaks from West Bengal, Madhya Pradesh and Orissa following the Bangladesh Liberation War and the arrival of hundreds of thousands of refugees from East Pakistan (Bangladesh) and their rehabilitation in different parts of these states.

In February-March, 1977, cholera outbreak was reported from Manipur, a state that was till then affected by the El tor invasion. The outbreak occurred among a group of about 2,500 labourers working in a river project. They were living in shanties and accessing well and river water for drinking purposes. Defecation was in the open and was considered to contaminate the drinking water sources.

Contaminated municipal piped water supply was incriminated in different outbreaks in Indore in 1980, Bhavnagar in 1987 and in Ahmedabad in 1988. In 1988, there was a major epidemic in Delhi which will be taken up later in the next chapter.

New Endemic States

Bengal was the home of classical cholera. Post-1964, the distribution of cholera within India underwent a change. States with low or zero incidence gradually became endemic for cholera. The severe epidemics (particularly following fairs and festivals) with high mortality had been replaced by endemic foci and low mortality (Shrivastav, 1970). Sen Gupta and Kapoor (1969) reported that large number of cases were reported from Assam, Gujarat,

Tamil Nadu, Mysore, Rajasthan and Delhi between 1964-68. While the attack rate declined during this period in Bihar, Madhya Pradesh, Orissa, West Bengal and Tripura, it increased in Andhra Pradesh, Assam, Gujarat, Kerala, Tamil Nadu, Maharashtra, Mysore, Rajasthan and Delhi.

Between 1965-75, 16 or more states were reporting cholera against 6-10 states that used to report cholera earlier. Cholera was reported from states that were free from cholera for a long time – Kerala, Gujarat, Haryana, Punjab and Rajasthan, or, from areas which had never known the disease – Andaman and Nicobar Islands (Sharma, 1975).

In the 25 years after the introduction of the El tor strain, Maharashtra, Tamil Nadu, Karnataka, Kerala and Delhi accounted for about 80% of the reported cases (GoI, 1980, 1988 and 1992). In 1994, 1995 and 1996 the incidence of cholera in Delhi was 45.10%, 57.81% and 20.47% of the all-India figures. In 1996, nearly half the cases were reported from Maharashtra after a lull of several years (indiastat.com, 2002).

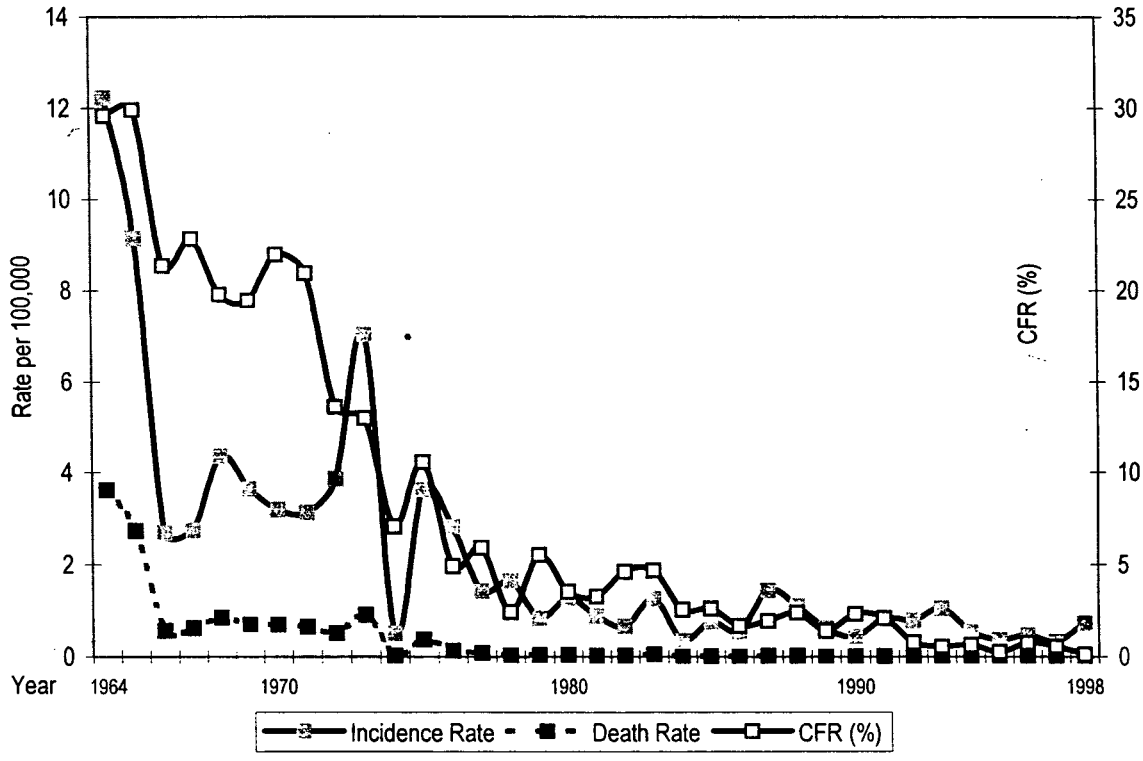
Time Trends

As already mentioned, cholera declined in India by the mid 1950s and in 1955, 14,167 cases and 5,250 deaths were reported. Even before the introduction of El tor cholera in 1964, there was a steady decline in the incidence and case fatality rate in India. The annual death rate from cholera (per thousand population) dropped from 1.58 in 1900-24 to 0.17 in 1948-63 and further to 0.0017 in 1964-68 (Sengupta and Kapoor, 1969). Epidemics also became less frequent. The case fatality rate from cholera declined from 49.34% in 1950 to 35.64% in 1963. The arrival of the El tor strain was marked by a sharp and steady reduction in the CFR (Sengupta and Kapoor 1969).

Graph 3.4 represents incidence rate, death rate and case fatality rate of cholera in India during 1964 to 1998. Data on cases and deaths is available for this period (GoI, 2001). Data on population and decadal growth rates were available for the census years 1961, 1971, 1981,

1991and 2001 (Census, 2001). Population figures for the intervening years was calculated by applying the decadal growth rate to the population of the corresponding census year. Incidence rate and death rate have been expressed per 100,000 population and case fatality rate (CFR) has been expressed as a percentage.

Graph 3.4 : Incidence Rate, Death Rate and Case Fatality Rate of Cholera in India, 1964-98



Incidence of cholera cases had doubled from 1962 to 1963 and during 1964 it remained at that level. *Vibrio cholera* El tor was identified in India for the first time in 1964. Incidence rate progressively declined during the 1960s. There was a rise in incidence rate between 1971-73. This coincided with the phase when arrival of larger numbers of Bangladeshi migrants (following Liberation War of Bangladesh) and cholera spread to several Indian states. Thereafter incidence rate declined to about 1 per 100,000 cases with minor fluctuations across years. Death rate declined sharply with El tor replacing the classical biotype across the country. Though incidence rate increased during 1971-73, death rate

continued to decline even during those years. There has thus been a steady systematic decline in the death rate. 20,000 cholera deaths were reported in the country in 1964 and only 10 cholera deaths were reported in 1994 (GoI, 1998) – death rate reduced from 3.620275 per 100,000 to 0.001036 per 100,000 during this period.

Case fatality rate declined because of the intrinsic milder nature of the El tor strain as compared to the classical strain. Before the arrival of the El tor strain there was already a sharp decline in the case fatality rate due to classical cholera between 1956 (43.06%) and 1963 (35.15%). Despite the introduction of the new strain (El tor), the CFR continued to decline to 19.49% in 1969. Though cases declined in 1970-71 there were relatively more deaths and the CFR increased marginally. Thereafter it was a steady declining phase.

The reduction in CFR started from the mid-1950s. From 1920, for three-and-a-half decades, CFR remained in the range of 80%-90%. In the next one-and-a-half decade, the CFR reduced by 70%, when the WHO introduced oral rehydration therapy (ORT). In another one-and-a-half decades, following the introduction of ORS there was a slower decline by nearly 20%. As with many other diseases the peak reduction had been attained before the discovery/application of the specific therapeutic agent.

Seasonal Trends

In the endemic states, cholera does not show a stable endemicity. It demonstrates seasonal fluctuations and is also prone to focal epidemics. The seasonal incidence varies between different regions of the country and is subject to change in the same endemic pocket. In some states, the peak incidence is in August. In West Bengal (and Bangladesh) there has been a shift in peak incidence from summer and early winter to autumn. Compared to the classical biotype, El tor cholera has a higher infection-to-case-ratio (i.e. inapparent infections and mild cases) and is therefore prone to endemicity (Park 2000).

The Seventh Pandemic spread to all the continents; trade, commerce and faster modes of travel facilitating the spread. Countries experiencing war, civilian strife and famine were pictures of familiar vulnerability. Spreading across the Indian states, *Vibrio cholerae* El tor established itself in new areas. Notwithstanding the introduction of a new strain, incidence rate and death rate reduced consistently. The new endemic foci were predominantly urban areas; a repetition of the endemic pattern in industrial towns of North America and Europe a hundred years earlier. The vulnerability of poorer communities living without basic services was exposed. Even where piped water supplies had been established, there were problems of maintenance and contamination leading to focal outbreaks. Water storage, food safety and personal hygiene issues gained importance.

O139 BENGAL

Two letters were published in the March 13, 1993 issue of The Lancet regarding cholera outbreaks in India and Bangladesh with microbiological features that suggested the involvement of a hitherto unknown strain.

The first one was from Ramamurthy *et al* of the National Institute of Cholera and Enteric Diseases (NICED), Calcutta. They referred to a cholera like outbreak in Madras, Tamil Nadu that began on October 1992 and continued till the end of November 1992. The NICED examined 49 representative strains of that outbreak. In January 1993, 28 and 48 more strains of *V.cholerae* were received from Madurai and Vellore respectively for microbiological characterisation. By November 1992, more than 95% isolates were of non-O1 type and like the strains from South India were 'untypable'. Serological tests on 124 strains revealed that they were neither agglutinable with the O1 antiserum nor with any of the monoclonal antibodies against factors A, B and C of *V.cholerae* O1. The strains were categorised as *V.cholerae* non-O1. Except for one, all other strains from Madras were not agglutinable against the 138 antisera available at the National Institute of Health, Japan indicating the possibility of a new serotype.

These non-O1 strains had the unusual trait of producing cholera toxin (CT). Most of the strains were resistant to cotrimoxazole (98%), streptomycin (92%) and furazolidone (86%) but were sensitive to other commonly used antibiotics including tetracycline. The similarity in biochemical, serological and toxigenic status of the strains of *V.cholerae* non-O1, the association with an outbreak in Madras and isolation of similar strains from widely separated regions of India was interpreted to be of epidemiological significance and indicated the clonal spread of a 'novel' non-O1 serotype.

In the other letter published in The Lancet, Albert et al (1993) reported from the International Centre for Diarrhoeal Diseases Research, Bangladesh (ICDDR,B), Dhaka of an outbreak of acute watery diarrhoea in mid-January 1993 that resembled cholera. The outbreak occurred in southern Bangladesh and adults were the most affected group. By the middle of February, about 10,000 people had been affected and the estimated deaths were to the tune of 500. 67% of the rectal swabs cultured from the field outbreak had yielded non-O1 *V. cholerae.* 70% of the swabs cultured from the admitted patients at the ICDDR,B also yielded *V. cholerae* non-O1. The non-O1 strains could not be identified with any of the known types and the pandemic potential of this 'novel strain' was recognised. In Calcutta, there was no increase in the number of admissions of cholera cases at the Infectious Diseases Hospital but Ramamurthy et al reported that isolation of *V. cholerae* non-O1 had registered an unusual increase.

It was the unusual feature of the outbreaks of cholera that alerted investigators in Vellore, Madras, Madurai and Calcutta. Prior to this non-O1 vibrios had never been associated with any clinical disease, or more importantly, epidemic spread. Microbiological investigations of these outbreaks revealed that these isolates produced copious amounts of the cholera toxin (CT), though they were non-O1. Investigators at Kyoto University, Japan had previously developed grouping sera for all recognised non-O1 *V.cholerae* and they had been enumerated from 02 to 138. The new CT producing non-O1 *V.cholerae* did not group with any of these 137 serogroups; hence it was designated O139.

The O139 serogroup organisms were indistinguishable from other serogroups of *V.cholerae* in terms of morphology, cultural characteristics and biochemical reactions. It was resistant to Mukherjee's IV and V phages that were specific for *V.cholerae*. It did not agglutinate with O1 antiserum. But all strains of O139 that had been tested, produced CT. The amount of toxin produced by the Madras strain was 10.4-80 ng/ml, which corresponded to the level produced by the wild strains of *V.cholerae*. In the south Indian studies, *V.cholerae* had been found to be sensitive to tetracycline but resistant to cotrimoxazole and furazolidone. Using the O139 antiserum, 518 strains received from different parts of India during November 1992 were examined (Table 3.3).

Table 3.3 : Reported serogroups of *V.cholerae* in some Indian states

State	No. of strains	O 139	O 1	% of Non-O139 isolation
Andhra Pradesh	3	3	0	100
Karnataka	4	4	0	100
Maharashtra	16	16	0	100
Tamil Nadu	298	271	13	91
West Bengal	197	186	0	94
Total	518	480	13	93

Source : Adapted from Sachdeva, 1995

93% of the 518 samples tested were of the O139 type. Serogroup O139 dominated in all the states from where samples were received. There were early indications that the diseases was spreading across different states of India (Sachdeva, 1995) .

The non-O139/non-O1 strains did not produce any cholera toxin. At that stage, the authors were of the opinion that 'the O139 serogroup was spreading rapidly and was in the process of replacing El tor *V. Cholera*.' (Sachdeva, 1995). The rapidity of the spread of the O139 strain was reminiscent of the spread of the El tor in 1964. By 1965, about 89% of the *V. cholerae* isolates from different parts of the country were of the El tor type. In Calcutta, the O139 variety had completely displaced the El tor serogroup by May 1993.

Mapping the Spread of O139

Jesudasan and Jacob John (1994) traced the time sequence of the appearance and spread of the O139 serogroup. The earliest detection of the strain was from Vellore, Tamil Nadu in September, 1992 from an outbreak. In the following month, the strain was isolated from an outbreak in Madras. In Calcutta, the strain was identified in November 1992. In December 1992, there were reports of the new strain from southern Bangladesh and by February-March 1993, cases were reported from central and northern Bangladesh. Outbreaks were reported from Bangkok, Thailand in April 1993.

Investigators in Tamil Nadu (Vellore, Madurai and Madras), Karnataka (Mysore and Amaravati), Andhra Pradesh (Vishakapatnam) and Maharashtra (Nagpur) had sent strains of non-O1 *V. cholerae* isolated from patients with cholera during October 1992, to the National Institute of Cholera and Enteric Diseases (NICED), Calcutta for characterisation. In all these batches of samples, *V.cholerae* O139 was identified. The authors hypothesised that this clone had first appeared in the southern Indian peninsula (Vellore, Madras, Madurai or Mysore) and then spread both inland to Nagpur, Wardha, Rohtak and Ludhiana and along the coast of the Bay of Bengal to Vishakapatnam, Calcutta and Bangladesh. The infection also spread to Bangalore, Pune, Jodhpur, Jabalpur and Delhi. Within 6 months *V.cholerae* O139 had spread from South India to Bangkok providing evidence of the epidemiological propensity of the strain to survive and spread, in a context of ever increasing human movement across countries for trade and other purposes. This prompted different authors to forecast the rise of the O139 strain as the beginning of the 'Eighth Pandemic'.

Jalgaonkar and Fule (1994) studied the *Vibrio cholerae* O139 outbreak in Yavatmal (Maharashtra) in 1993 that marked the first reported incidence of the new infection from Western India. This outbreak occurred between March and July 1993. 34 strains of *V. cholerae* were isolated from 154 patients admitted at Shri Vasantrao Naik Govt. Medical College and Hospital, Yavatmal, Maharashtra with cholera. Of these 34 strains, 26 were non-O1 and 8 were O1 El tor vibrios. Incidentally, in July 1992, 7 strains of El tor vibrio, Ogawa, belonging to T2 phage were isolated from that region. The O139 isolates were found to be

sensitive to gentamicin (95.65%), tetracycline (75%), ampicillin (54.16%), cotrimoxazole (50%), chloramphenicol (40.74%) and streptomycin (11.23%).

Narang et al (1994) at the M. G. Institute of Medical Sciences, Sevagram. Wardha, Maharashtra isolated 44 strains of *V.cholerae* O139 between April and August 1993. All the strains were sensitive to tetracycline, erythromycin, gentamicin, ciprofloxacin and norfloxacin but were resistant to cotrimoxazole and polymyxin B (50 IU). The authors also reported an increase in the incidence of *V.cholerae* since 1990 when 15 strains were isolated as against 4 in 1989. The isolation was 36 in 1991 and 39 in 1992. In 1993, 54 strains were isolated of which 44 were O139 and 10 El tor Ogawa. It is important to note that no further strains of O139 were isolated after August 1993. The authors had therefore concluded that it would be difficult to predict whether the new strain would acquire epidemic proportions while other researchers were predicting the advent of the Eighth Pandemic.

At the same time as the Maharashtra outbreaks, O139 was reported from Haryana. Sabherwal and Sikka (1994) cultured 116 stool samples at Rohtak Medical College. These samples were collected from patients belonging to Rohtak and the neighbouring areas. 27.27% of the strains belonged to the O139 serogroup.

Almost at the same time Prabhakar et al (1994) studied stool samples at the Christian Medical College, Ludhiana, Punjab. These samples were collected from patients admitted to the Hospital between May to August, 1993. 35 out of the 41 strains of *V.cholerae* isolated were of the O139 type. Clinically, all the O139 cases suffered from diarrhoea and vomiting but without any cellular exudate in stools. 24 were adults and 11 were children. Tetracycline emerged as a drug of choice in Ludhiana as sensitivity was 100%.

Though the initial indications of epidemic spread of a new strain was obtained from different parts of Tamil Nadu (and investigated by the NICED, Calcutta), the worst affected contagious area was districts of southern Bangladesh and 24 Parganas (South) District of West Bengal. Between November 1992 and May 1993, 52,000 persons were affected in 24 Parganas (South) District. Over 11,000 were admitted in various health centres and hospitals

of the district; 688 died. During this period there were an estimated 100,000 cases and 10,000 deaths in southern Bangladesh (Park, 2000). Most of the cases in both countries were adults, pointing to the invasion of a new organism in a population without pre-existing immunity. Watery diarrhoea and severe dehydration was recorded in most of the cases. *V. cholerae* non-O1 had been known to be ubiquitous in tropical aquatic environs and 24 Parganas (South) district in India shares the same estuarine eco-system with Bangladesh. In addition there are regular population movements and trading (particularly fishing and forest based activities) between the two countries (Dasgupta, 1995 a).

The Infectious Diseases Hospital, Calcutta admitted large numbers of cases from the Calcutta suburbs and 24 Parganas (South) District. Table 3.4 reports the details of the acute diarrhoeal cases admitted in the hospital in 1993, including the O139 cases (Bhattacharya et al 1994).

Table 3.4 : Acute Diarrhoea Cases at the Infectious Diseases Hospital, Calcutta, 1993

Month	A. D. Cases <10 yrs age			A. D. Cases > 10 yrs age			Total Diarrhoea Cases		
	M	F	CFR	M	F	CFR	A. D.	O 139	Age > 10y
	No.	No.	%	No.	No.	%	No.	(%)	(%)
Jan	392	251	15.55	399	306	65.24	1348	35.0	52.3
Feb	369	244	16.31	506	480	36.51	1599	28.0	61.7
Mar	526	386	20.83	2613	1854	36.48	5379	44.9	83.0
Apr	517	406	43.33	5311	3161	26.91	9395	54.1	90.2
May	513	366	45.51	2718	1891	22.99	5488	54.2	84.0
Jun	425	337	18.37	998	914	17.78	2674	55.1	71.5
Jul	393	245	23.51	1288	1082	20.67	3008	53.4	78.8
Aug	363	302	24.06	1323	1421	20.40	3409	60.8	80.5
Sep	529	412	20.19	1897	1605	21.41	4443	57.3	78.8
Oct	549	367	22.92	2112	1331	20.33	4359	51.0	79.0
Nov	287	224	11.74	917	762	13.69	2190	41.2	76.6
Dec	260	159	7.15	501	310	32.06	1230	11.1	66.0
Total	5123	3699	24.14	20583	15117	25.54	44522	50.43	80.2

Note : A. D. : Acute Diarrhoea M : Male, F : Female, CFR : Case Fatality per 1000 cases
Source : Bhattacharya, 1994

50% of the admissions for acute diarrhoea cases i.e. 22,453 cases of *Vibrio cholerae* O139, were reported from the Infectious Diseases Hospital, Calcutta during 1993. Cases were reported throughout the year with peak during August, September and October. 80.2% of the

acute diarrhoea cases were aged more than 10 years, suggestive of a new strain: case fatality rate among this group was as high as 65.24% in January but later declined. The data reported shows that number of male admissions were consistently higher than females, across months and age groups; the study does not elaborate on the factors that could explain this difference.

In an in-depth study of 113 patients of O139 cholera at the Infectious Diseases Hospital, Calcutta, a distinct clinical entity was described that was characterised by painless watery diarrhoea, vomiting, dehydration and absence of fever (Bhattacharya et al 1994). The illness was generally a severe life-threatening infection with dehydration (85%). None had blood in the stool but abdominal cramps were recorded in 44.3% of cases. Thus, the disease was clinically indistinguishable from a typical severe O1 cholera, with the exception of cramps.

The National Institute of Communicable Diseases, Delhi (Sachdeva et al, 1994) reported a large series both from different states where NICD had investigated cholera outbreaks and a summary is presented in Table 3.5 :

Table 3.5 : *V.cholerae* O139 isolated at NICD from different states, 1993

State	Samples	Isolation (%) of	
	No.	*V.cholerae*	O 139
Andhra Pradesh	9	0.0	66.7
Assam	4	0.0	0.0
Bihar	17	0.0	5.9
Delhi	2850	24.4	29.2
Gujarat	5	0.0	0.0
Haryana	122	10.7	27.9
Karnataka	89	1.1	52.8
Madhya Pradesh	37	0.0	35.1
Maharashtra	44	0.0	15.9
Rajasthan	28	3.6	39.3
Tamil Nadu	2	0.0	50.0
Uttar Pradesh	66	0.0	62.1
Total	3273	21.7	30.3

Source : National Institute of Communicable Diseases

Out of 423 samples received from different states of the country, other than Delhi, 38.1% were confirmed to be *V.cholerae* O139. 29.2% was the isolation rate for *V.cholerae* O139

from Delhi. Isolation rates from south Indian states and Uttar Pradesh were higher, in the range of 50-60%. The number of total samples tested in Delhi is high as it includes the routine testing of all diarrhoea cases admitted at the Infectious Diseases Hospital, Delhi whereas the samples from other states were only those of suspected clinical cases of O139 cholera.

Till 1993, non-O1 vibrios had been associated with sporadic cases or relatively confined outbreaks of diarrhoea (Ramamurthy et al, 1993; Karnel, 1971). Incidence of non-O1 *V.cholerae* was reported to be 5-10% of the hospitalised cases in Calcutta (Ramamurthy, 1992) and 1-3% in Bangladesh (Albert et al, 1993). The isolation rate of *V.cholerae* O1 was 12.2%-40.9% between 1983 and 1992. Although during 1993 there was a sudden spurt in the isolation rate of *V.cholerae* non-O1 (O139), the isolation rate of *V.cholerae* O1 remained similar to the previous years. The isolation rate of *V. cholerae* O139 was twice as high as that of *V.cholerae* O1 from April to June in 1993 and thereafter the isolation rate of *V.cholerae* O139 declined and isolation rate of *V.cholerae* O1 increased.

Resistance to cotrimoxazole was as high as 94.5% in northern India. Strains in Maharashtra were also resistant to cotrimoxazole but not to that extent. Resistance to cotrimoxazole and furazolidone was also reported from Bangladesh and southern and eastern states of India (Sachdeva, 1995).

While some strains of non-O1 vibrios cause diarrhoea mediated by NAG-ST, only some rare strains have been known to produce CT. Although it is clear that the O serogroup specificity and CT production are unrelated properties, for all practical purposes agglutination with O1 or O139 antisera is sufficient to call a given strain of *V.cholerae* as choleragenic. The O1 variety does not have invasive properties and it does not cause bacteraemic illness. In contrast, non-O1 organisms occasionally cause invasive illness. In cholera endemic areas many adults are immune, at least partially, to the O1 serogroup; thus children are more affected than adults. In the outbreaks due to O139 vibrios, a higher than expected proportion of adults were affected indicating that all communities were epidemiologically naive to this clone.

O139 outbreaks, despite spreading rapidly, did not culminate in the 'eighth pandemic' of cholera. It did not eliminate the El tor strain as was hypothesised by some experts. Even at the stage when it was spreading rapidly, Dasgupta (1995 b) demonstrated in a cholera outbreak in Garhi Village, Delhi that it was possible for El tor and O139 to co-exist. By the beginning of 1994, El tor had resumed its dominance in Bangladesh and this trend continued in 1995. Mahalanabis (1994) studied 502 cases of O139 cholera patients and 63 cases of O1 cholera in Dhaka, by systematic sampling of 1854 cases and reported that there were differences in vulnerability factors between the two variants of cholera. He observed that only 1.6% of O1 cases had in-house potable drinking water compared to 7.2% of O139 cases. 30.7% of O139 cases had access to sanitary latrine while only 9.1% of O1 patients had access to this facility. In India, O139 has continued to flare up later at different points of time but no specific risk factors could be identified. *Vibrio cholerae* El tor soon regained its position as the dominant strain.

Cholera never lost the status of being a political disease; *Haija* still retains a certain political connotation in Delhi. The chapter traces the response of the colonial regime to different facets of the disease in order to gain a holistic understanding of various forces; beyond the paradigm set by traditional bio-medical epidemiological models. History of cholera in India corresponds to the history of pandemics. Poverty, lack of social security, failure of the state to provide equitable basic services (and protect socio-economically and politically weaker communities) and poor public health engineering standards stand out as vulnerability factors down the ages. Though the decline of cases and deaths may seem impressive, the emergence of endemic urban pockets of cholera is both a matter of shame and concern. Among the endemic states, Delhi stands out prominently. While civil society grapples with social, economic and political priorities of provision of basic essential infrastructure, the pathogen also throws up new challenges to expose the gaps. *Vibrio cholerae* O139 Bengal was able to raise the possibility of an eighth pandemic because of these intrinsic weaknesses. The following chapter will examine the time trends of Delhi in the backdrop of the 1988 epidemic that was remarkable not so much for of its magnitude but because it highlighted the plight of poor communities and faulty services in the capital of the country.