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Health Hazards in 19th Century India: Malaria and Cholera in Semi-Arid Tropics

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Abstract

When we examine the population statistics for British India from the beginning of the 1870s, we see that the population growth rate remained at a very low level until the 1920s. There was a sudden change in the population growth rate in the 1920s. Thereafter it increased to a level of more than 1%. The low population growth rate in the first period was determined by the high mortality rate resulting from numerous famines and epidemics (smallpox, cholera, plague, malaria, influenza etc.).

From the middle of the 19th century most parts of Asia faced globalization in terms of trade growth. We will find that globalization itself led to serious health hazards in some parts of Asia during the 19th century. Both India and China faced serious health hazards in this period. But we can infer that there was a great difference in the scale of damage between India and China. Although we cannot obtain good mortality data on China, particularly inland China, for this period, we can estimate that various epidemics brought about lower mortality in China than in India.

In this paper we will clarify ecological conditions which intensified the human damage of epidemics in India. We take up two epidemics, cholera and malaria which devastated India most seriously during the period. We will particularly focus on climatic conditions, that is to say, high temperature and semi-aridity. These conditions led to very serious types of cholera and malaria epidemics.

1. Introduction

From the middle of 19th century most parts of Asia faced globalization in terms of trade growth. The globalization led to serious health hazards in some parts of Asia during the 19th century. In particular, various epidemics inflicted severe damage in South Asia. From the early 1870s to the late 1910s, South Asia experienced many famines and epidemics, leading to huge death tolls and checking population growth.

Similarly, East Asian countries that opened themselves to the outer world faced epidemic diseases such as cholera. The trade growth had a close connection with the spread of epidemic disease. East Asia, too, faced serious health hazards in this period. There was a great difference in the scale of damage between South Asia and East Asia. Although mortality data on China is not available, particularly inland China, for this period, we can estimate that various epidemics brought about lower mortality in China than in British India. Vital statistics are not available at all, but we may speculate that the level of cholera mortality in China was much lower than in India in the second half of 19th century.[1] According to some studies on the epidemic history of Southeast Asia,[2] during the period in question some areas were ravaged by epidemic diseases like cholera and malaria. The Philippines experienced the highest crisis mortality in late 19th century.[3] In general, Southeast Asia recorded high population growth even in this period.[4] Although high fertility must have been a factor, it seems that the level of mortality was not as high as in South Asia. This is a just rough speculation, but this kind of comparison should be attempted more rigorously in the future.

If we compare India with China, it is certain that there was a big difference of railway development between these countries in the second

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[1] Wataru Iijima compiled the mortality data of treaty ports in China recorded by maritime customs. He pointed out that the frequency and mortality level of various epidemics in modern China were not as high as usually assumed. W. Iijima, ***Pesuto to Kindai-Chugoku (Plague and Modern China)***, Tokyo, 2000, p. 333.

[2] P N.G. Owen 'Toward a History of Health in Southeast Asia', in N.G. Owen (ed.), ***Death and Disease in Southeast Asia: Explorations in Social, Medical and Demographic History***, Singapore, 1987, pp. 8-16.

[3] .C. Smith, 'Crisis Mortality in the Nineteenth Century Philippines: Data from Parish Records', ***Journal of Asian Studies***, vol.38, no.1, pp. 62-72.

[4] A. Reid, 'South-East Asian Population History and the Colonial Impact', in Ts'ui-jung Liu, James Lee, et. al., ***Asian Population History***, Oxford, 2001.

half of 19th century. The total length of railway network recorded 41,221 km in British India in 1903, while in China it was only 3,330 km in 1902. Railway development must have promoted diffusion of epidemics such as cholera or plague in India. But there is another factor for explaining the different mortality levels of epidemics.

Ecological conditions must have disadvantaged South Asia. The semi-arid climate of India might have raised the level of mortality in epidemic diseases. In the case of cholera, for example, the scarcity of water aggravates the problem of water contamination. Ira Klein rightly pointed out a very important factor which amplified the death toll in cholera epidemics in the central India during the late 19th century. He emphasized the arid ecology and lack of water in explaining the huge mortality caused by cholera in the central part of India. When the railways were being built there, many construction laborers came into the area. Then once railway network was built, many passengers passed through central India.[5] The scarcity of water became acute, making the conditions related to the fecal-oral route of cholera infection much worse. This causal relationship was stronger in the South Asian climatic conditions than in any other regions. Recently Tirthankar Roy claimed that we should take into account factors of resource constraint such as water scarcity more seriously in order to explain the low level of well-being of the people during the late British period. It was also pointed out that this factor was neglected by the commonly accepted interpretation of economic development during the British period.[6] In the same way we need to introduce the factor of resource constraint into epidemiological causation in explaining the high level of mortality in South Asian epidemics.

In this paper we will take up malaria and cholera in order to examine above-mentioned factors. In South Asia, the worst cause of death during 19th century was fever[7] and the second one was cholera. We will analyze and categorize types of malaria and cholera, from the viewpoint of epidemiology.

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[5] I Klein, 'Imperialism, Ecology and Disease: Cholera in India, 1850-1950', *The Indian Economic and Social History Review*, vol. 31, no. 4, 1994, pp. 505-510.

[6] T. Roy, *Rethinking Economic Change in India: Labour and Livelihood*. London, 2005.

[7] 'Fever' was the very wide category which included many diseases. Malaria was included in this category. But there is no doubt that malaria dominated other disease in this category.

2. Malaria in 19th Century India

Malaria is usually assumed to be an endemic disease. It is said that malaria is closely connected with locality.[8] But, if we look at demographic history of 19th century India, it is clear that epidemic malaria is more important than endemic one.

(1) Famine and Epidemic Malaria

In the United Provinces between 1873 and 1948, severe mortality crises occurred seven times, in 1879, 1894, 1897, 1905, 1908, 1911 and 1918. In all cases, more deaths were due to disease than starvation. Such diseases as cholera, smallpox, diarrhea, dysentery and malaria, which spread after famine produced large-scale mortality. The most serious epidemic was malaria. 'Epidemic malaria' was the worst factor in raising the mortality rate.[9]

Epidemic malaria most often followed famines. When the nutritional deficiencies among the rural poor after famine coincided with proliferation of *Anopheles* – vector of malarial parasite – caused by heavy rain, *Plasmodium falciparum* took many lives.[10] Other recent studies have shown that such famine-malaria nexuses were also seen in the other provinces.[11]

In early 20th century a British malariologist, S.R. Christophers for the first time consciously analyzed the relationship between famine and epidemic malaria in his report on the 1908 Punjab malaria epidemic.[12] But his study has been neglected until recently. With detailed examination of this epidemic, Christophers wrote a report in 1911, in which he asserted the importance of conditions of human bodies, although the main factor was anopheles, as flooding caused by excessive rain left the area waterlogged.

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[8] Until 19th century malaria was thought to be a 'disease of locality'. As is well-known, it was the disease most aptly applied by miasmatic theory of disease.

[9] K. Wakimura, 'Famines, Epidemics and Mortality in Northern India, 1870-1921', in P. Robb, K. Sugihara and H. Yanagisawa (eds.), *Local Agrarian Societies in Colonial India: Japanese Perspectives*, Surrey, 1996.

[10] S. Zurbrigg, 'Hunger and Epidemic Malaria in Punjab, 1868-1940', *Economic and Political Weekly*, January 25, 1992; A. Maharatna, *The Demography of Famines: An Indian Historical Perspective*, New Delhi, 1996, p.81.

[11] T. Dyson, 'On the Demography of South Asian Famines, Part I', *Population Studies*, vol.45, no.1, 1991; do., 'On the Demography of South Asian Famines, Part II', *Population Studies*, vol.45, no.2, 1991; E. Whitcombe, 'Famine Mortality', *Economic and Political Weekly*, June 5, 1993; O. Saito, 'Kikin to Sibou to Jinkouhendou' [Famines, Mortality and Population Change], in H. Yanagisawa (ed.), *Gendai Minami Ajia 4, Kaihatsu to Kankyo* [Contemporary South Asia 4, Development and Environment], University of Tokyo Press, 2002.

[12] S.R. Christophers, *Malaria in the Punjab*, Calcutta, 1911.

He claimed that the most important factor causing malaria epidemics was economic condition of people. To begin with, he examined the relationship between mortality rates and social status, and then studied the relationship between the prevalence of epidemic malaria and scarcity of food. Concerning the latter relationship, he pointed out that epidemic malaria had often broken out in the year following a famine. Epidemic malaria did not occur during the year of the famine, because there was little rainfall. If excessive rainfall occurred during the year just after the famine, the anopheles factor would become tied up with the human factor, which is the deterioration of nutritional conditions.

> There seems very little doubt that the two factors, rainfall and scarcity, are the determining causes of the epidemic malaria seen in the Punjab. Broadly speaking until plague appeared malaria must have been the main agent which brought to a head in actual mortality the effects produced by the great economic stresses. Just as in famines malaria cannot act until nature is about to bring them to an end, so there can be little doubt that the effects of scarcity are to a large extent held over until the appearance of the first heavy monsoon. Then though the effect of the rain is to reap a harvest of deaths the period of stress is brought to an end.[13]

As already shown, recent studies have also emphasized the close causal relationship between famine or malnutrition and epidemic malaria. For example, Zurbirgg applied regression analysis to the correlation between malaria mortality index and the food price index.[14] She pointed out that 'acute hunger' or 'frank starvation' was important in explaining the causation of epidemic malaria. 'Acute hunger' or 'frank starvation' means that nutritional intake decreases drastically. Her subsequent finding is that the number of casualties in epidemic malaria was determined not by the incidence of infection but by the case mortality. Why did the victims of epidemic malaria diminish after the 1908 epidemic? Her answer was: 'What appears to have changed after 1908 is not so much the incidence of infection —numbers of persons infected during the post-monsoon period—but lethality

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[13] ***Ibid.***, p. 112.
[14] Zurbrigg, 'Hunger and Epidemic Malaria in Punjab, 1868-1940'.

of malaria infection, the proportion of infected people dying of disease'.[15] Stark malnutrition determined the lethality of malaria infection. After the 1908 malaria epidemic, 'the frequency and prevalence of overt starvation clearly declined'[16] in Punjab and elsewhere. This is not an issue of exposure to the pathogens, but an issue of case mortality, though information on the latter is usually absent. The reason for increase of case mortality must have been the reduced nutritional intake.

On the other hand, why did the damage caused by epidemic malaria decline after 1908? Sumit Guha presented almost the same explanation as Zurbrigg's. He referred to S.R. Sen's comparison of agricultural stability between two periods.

> Comparing the period 1900-1 to 1923-24 with 1924-25 to 1950-1, he [S.R. Sen] observed that while foodgrain output was rising in the first period yet the divergence between peaks and troughs of output was also increasing. On the other hand, the second quarter-century saw stagnation in output accompanied by a convergence between peaks and troughs, so that agriculture was stagnant but stable.[17]

He attributed the decline in mortality after the 1920s to the stability of agricultural production. Although he does not mention epidemic malaria, he seems to pay great attention to the famine-induced epidemic malaria.

Famine in this subcontinent was caused by drought, mostly failure of south-west monsoon. Drought-prone areas were usually located in semi-arid zone in the subcontinent. As cultivation of crops was relatively active, population density was not so low in this zone. Failure of monsoon affected rather numerous populations. In combination with famines, epidemic malaria killed many people. However, we need to consider not only nutritional factor but also 'immunity factor'.

(2) Malaria and Semi-Arid Tropics

It is possible that low incidence of malaria due to dry climate reduced malaria immunity among the population living in semi-arid zone. Sometimes fulminant (explosive) epidemics of malaria devastated numerous people with low immunity when they are under the condition of nutritional deficiency.

We need to examine types of malaria more closely. According to Ian Stone,

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[15] *Ibid.*, p. 15.
[16] *Ibid.*, p. 16.
[17] S. Guha, *Health and Population in South Asia: From Earliest Times to the Present*, London, 2001, p. 84.

who emphasized the immunity factor, there were two types of malaria in the western United Provinces during this period; endemic 'benign malaria' and epidemic 'malignant malaria'. The endemic 'benign malaria' was due to *Plasmodium vivax*, which often caused relapses but also maintained the immunity of the infected persons. When a relapse occurred, the infected person suffered from high fever, but did not become seriously ill, except for small children and infants, whose severe conditions could not be treated. On the other hand, the 'malignant malaria', which was due to *Plasmodium falciparum*, became epidemic malaria in this region. People who contracted 'malignant malaria' did not suffer relapses, and so did not maintain their immunity. Therefore the epidemic 'malignant malaria' periodically devastated the region, killing many people. The immunity factor explains these periodic outbreaks of malaria epidemic.[18]

Also we need to look at 'vector' factor. Stone gave us very useful information concerning the ecology of anopheles. In the western United Provinces the main malaria carrier, *Anopheles culifacies*, was so zoophilous and short-lived that the probability of transmission was relatively low. Only when the numbers of this species of anopheles increased significantly, the possibility of transmission increased. Heavy rain increased the breeding places of anopheles and created atmospheric humidity in the monsoon season.[19]

Therefore, we need to situate these types of malaria in the wider geographical configuration. Following the study of Christophers and Sinton, A. Learmonth once pointed out that there was 'the 40-inch (1,016 mm) isohyet as a crucial divide, a line on the map familiar to geographers, as roughly dividing humid (rice-eating) India from arid and semi-arid (wheat and millet-eating) India: here it is similarly taken as the malariological divide between humid and endemic India as against arid and semi-arid epidemic India'.[20] According to Learmonth, furthermore, the epidemic area can be divided into two parts. One was 'area(s) liable to fulminant epidemicity (diluvial) malaria'.[21] Another was the drier area where modest epidemic

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[18] I. Stone, *Canal Irrigation in British India: Perspectives on Technological Change in a Peasant Economy*, Cambridge, 1984. Stone recognized that in any case incidences of both endemic malaria and epidemic malaria were intensified by the waterlogging. Also he pointed out malaria was intensified by canal irrigation, though he claimed that the total economic benefit of canal irrigation substantially exceeded the environmental costs.

[19] *Ibid*.

[20] A. Learmonth, *Disease Ecology*, Oxford, 1988, pp. 205-207.

[21] *Ibid*., p. 206.

malaria occurred. The western United Provinces and the northern and southeastern Punjab are located in the former area. If we look at a map indicating average annual precipitation, we find this area inside of the 20-40 inches (508-1,016 mm) precipitation region. On the other hand, the western Punjab is located in the latter area, where average annual precipitation is less than 20 inches (508 mm). We may assume that fulminant epidemic malaria often occurred in the western United Provinces but rather rarely in the western Punjab. To summarize the above-mentioned discussion, we can conclude that semi-arid climate promoted occurrences of very dangerous epidemic malaria during this period.

Next, we take up a case of epidemic malaria even in endemic area of malaria. As Learmonth pointed out, Bengal can be included into endemic area of malaria. But, since the middle of the 19th century intense epidemic malaria called 'Burdwan Fever' struck the central and western Bengal.[22] Series of malaria epidemics substantially influenced the population change in the second half of 19th century Bengal. C.A. Bentley proved a vicious circle between disadvantaged economic conditions and epidemic malaria in this area. He explained this situation in terms of 'agricultural deterioration'.

> The epidemic malaria of the Punjab is regarded therefore as arising from the conjunction of conditions favouring an increase of anopheles mosquitoes and the consequent spread of malarial infection with a period of serious scarcity of food among certain classes of the population. And in Bengal epidemic malaria can likewise be shown to be due to the action of the same factors. But unlike the Punjab, which is naturally a dry and comparatively well drained country in which abnormally heavy rainfall encourages the multiplication of anopheles, scanty rain and diminished flooding favours the increase of these mosquitoes in Bengal. And allowing for this difference, the epidemic malaria of the latter province is seen to be due to the operation of causes fundamentally similar to those responsible for its occurrence in the Punjab,

[22] Concerning 'Burdwan Fever,' see the following studies. B. Chaudhuri, 'Agricultural Production in Bengal, 1850-1900: Coexistence of Decline and Growth', ***Bengal Past and Present***, Vol. 88, Part 2, No. 166, July-Dec., 1969; I. Klein, 'Malaria and Mortality in Bengal, 1840-1921', ***Indian Economic and Social History Review***, Vol. 10, No. 2, 1972; S. Bose, ***Peasant Labour and Colonial Capital: Rural Bengal since 1770***, Cambridge, 1993; K. Wakimura, 'Anopheles Factor and Human Factor: Malaria Control under the Colonial Rule, India and Taiwan', in M. Hasan and N. Nakazato (eds.), ***The Unfinished Agenda: Nation-Building in South Asia***, New Delhi, 2001.

> viz., an increase of facilities for the spread of malarial infection on the one hand together with abnormal economic stress on the other. In the delta tracts of Bengal short rainfall and scanty inundation favour anopheles mosquitoes, and lead at the same time to agricultural deterioration and poor harvests, the immediate result of this combination of factors being a great intensification of malarial infection, which manifests itself either in the form of acute epidemic outbreaks of the disease or by the more gradual depopulation of the areas affected.[23]

What caused such an 'agricultural deterioration' in the central and the western Bengal? According to Bentley, river inundations had given deltaic Bengal a highly fertile soil; but when railways or roads were constructed, the embankments disturbed river inundation, preventing proper silt accumulation. From the middle of the 19th century to the early 20th century, the 'proportion of current fallow and cultivable waste to net cropped area' had increased in the central and the western Bengal, and the 'percentage by which the outturn of principal food crops fell short of the normal' had also risen.[24] By contrast, in eastern Bengal agricultural growth was very prominent.

During the same period, as already mentioned, population growth was stagnant in the central and the western India. Sometimes population declined due to both malarial impact and out-migration. Sugata Bose wrote in his study:

> Population density in Burdwan fell back from over 700 to under 550 per square during the 1860s and 1870s. The population of Hooghly was said to have been halved between the late 1850s and the late 1870s. In the malaria-infected parts of Midnapur population declined by nearly a third in the latter half of nineteenth century. Local investigations in selected villages of Nadia, Jessore, Burdwan, Birbhum and Hooghly confirmed the impression of large-scale depopulation in epidemic years.[25]

This was a consequence of the vicious cycle between 'agricultural deterioration' and epidemic malaria.

There was another important factor behind outbreaks of the 'Burdwan Fever'. Obstructions to inundation considerably affected the 'vector' factor as well as the nutritional factor. Usually flooding constrained the

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[23] C.A. Bentley, *Report on Malaria in Bengal, Part 1*, Calcutta, 1916, p. 73.
[24] C.A. Bentley, *Malaria and Agriculture in Bengal: How to Reduce Malaria in Bengal by Irrigation*. Calcutta, 1995.
[25] S. Bose, *Peasant Labour and Colonial Capital: Rural Bengal since 1770*, Cambridge, p. 25.

breeding of anopheles. On the contrary inadequate inundation promoted proliferation of anopheles larvae.

> The inundation of the country during the monsoon is unfavourable to the multiplication of anopheles mosquitoes, in the first place, because flooding reduces the dangerous 'water-edge' which affords safe cover for mosquito larvae; in the second place, because owing to the large surface exposed to the rays of the sun the temperature of the water tends to rise so as to be exceedingly unfavourable to the life of anopheles larvae; and in the third place, because the physical and possibly the chemical character of river water is inimical to anopheles larvae.[26]

The main vector mosquito ***Anopheles philippinesis*** preferred 'still water for egg-laying and larval production, not heavily polluted, with rather a low water-table and a moderate (but not light-excluding) growth of aquatic plants'.[27] Interruptions in flooding facilitated the reproduction of anopheles, leading to the prevalence of malaria. It was concluded that hindrances to inundation affected both anopheles and human factors, resulting in 'Burdwan Fever'. This environmental change was brought about by the construction of railways and roads.

This environmental change means a change from the area of endemic malaria to the area of epidemic malaria. Although climatic conditions did not change, the ecological change promoted epidemicity of malaria.

3. Cholera in 19th Century India

Cholera is a typical epidemic disease. The cholera pandemics occurred six times. They were highly infectious and transmissible. But the cholera pandemics always originated from Bengal where it was endemic one. Therefore we need to think about both epidemicity and endemicity of cholera.

(1) Cholera, Railway and Pilgrimage

In India and its surrounding areas, we find certain diffusion routes of cholera epidemics in 19th century. The epicenter was always Bengal. But

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[26] Bentley, ***Malaria and Agriculture in Bengal***, pp. 48-49.
[27] Learmonth, ***Disease Ecology***, p. 5.

there were three routes of spreading from Bengal. Firstly, cholera usually moved to the north-west direction along the Ganges River. It reached Punjab, then sometimes spreading to the Central Asia or Persia, finally Russia. Secondly, usually on the way along the Ganges, cholera epidemics went ahead from the middle of the Ganges valley to the central India, and it reached the western India or the southern India. From the western India cholera strode over the Arabian Sea to the Arabian Peninsula or Persia by shipping. Thirdly, cholera jumped out from Bengal across the Bay of Bengal toward the east or south direction by sea. It often reached the southern India or the western India, even south-east Asia.

Compared between two diseases, cholera may be characterized as 'disease of traffic'. It spread out usually through traffic routes. Especially sea route was important for pandemic cholera. But in 19th century land route was also important. For example, cholera was sometimes transmitted only through land route to Europe in 19th century. Cholera coming from hot and humid locality such as Bengal, travelled down through vast arid and semi-arid zone in Eurasian continent to Europe. How could cholera carry on such a long journey.

Before trying to answer this question, we will look at new development of transportation networks. In the second half of 19th century the situation concerning cholera changed very substantially. Mortality data became available only after the late 1860s. Based on these vital statistics, David Arnold showed the change in fatalities caused by cholera in British India. According to his table, we find that cholera deaths continuously increased from 146,998 in the 1865-1870 period to 444,923 in the 1891-1900 period. As far as the period from 1874 to 1968 is concerned, the average annual cholera mortality rate was highest in the 1874-1899 period. Although we do not have any data on mortality before the late 1860s, we can assume that cholera mortality increased during the second half of the 19th century. The development of the railway accelerated throughout this period. The route miles soared from 838 in 1860 to 23,672 in 1900. The numbers of passengers substantially increased from 19 million in 1871 to 183 million in 1901. There is no doubt that cholera moved more speedily.

We also have to pay attention to the increased number of pilgrims resulting from the development of the railways. As is generally known, pilgrimage was one of the most important factors in the propagation of

cholera throughout India.[28] Here I quote just one passage from the preceding study. 'Even more dangerous from an epidemiological viewpoint than the annual pilgrim traffic were the Kumbh melas held at Allahabad and Hardwar every twelve years and the intervening Ardh Kumbh melas. As many as three million pilgrims at a time participated in these festivals, living in crowded and insanitary lodgings or encampments, bathing *en masse* in the sacred Ganges and sipping its holy water --- conditions that were almost ideal for cholera transmission.'[29]

Introduction of steamship in the Indian Ocean during the 1830s-1850s period also greatly influenced. Opening Suez Canal was important too. Both growth of trade and increase of Muslim pilgrimage to Mecca (Haji) promoted diffusion of cholera.

(2) Cholera and Semi-Arid Tropics

Cholera is assumed to be contagious. But this statement is not exact in the sense that cholera is transmitted directly from person to person. Water is the medium which carries *Vibrio cholerae* from person to person. The most common way is fecal-oral route which means that a person drinks water contaminated with cholera-infected human waste. In this regard, role of water in cholera transmission is almost same as role of *Anopheles* in malaria transmission. In the following discussion we focus on the water factor.

The damage caused by cholera epidemics was different from one place to another, especially depending on the availability of water. We will take up at a case in the Central Provinces. The Central Provinces were located in the center of Indian sub-continent. 'Central India became a transport hub, the terminus of the great trunk lines linking coastal ports. Specifically, it became the juncture for the Great Indian Peninsula and East Indian Railways, joining Bombay and Calcutta, and for lines from Delhi to Madras.'[30] Even before introduction of railway, the central India was the region through which cholera went southward or westward.

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[28] I.J. Kerr, 'Reworking a Popular Religious Practice: The Effects of Railways on Pilgrimage in 19th and 20th Century South Asia', in I.J. Kerr (ed.), *Railways in Modern India*, New Delhi, 2001.
[29] D. Arnold, 'Cholera Mortality in British India, 1817-1947', in T. Dyson (ed.), *India's Historical Demography: Studies in Famine, Disease and Society*, London, 1989, p. 272.
[30] Klein, *op. cit.*, p. 506.

There were some places where the mortality rate of cholera was very high. On the other hand there were other places where mortality rate was low. This difference was determined almost by availability of water. Look at the following observations. These citations are taken from the report written by S.C. Townsend, who was the Sanitary Commissioner of the Central Provinces in the 1860s and 1870s. He investigated the 1868 cholera epidemic in the Central Provinces.

> We find that the highest rate of mortality occurred in the trap formation; that the disease also visited with great severity villages along the banks of the Nerbudda, the Hirun, and the Pureyat rivers, that traverse the wide alluvial tract of the Jubbulopore district; and that, on the other hand, the proportionate number of villages attacked, and the ratio of mortality, were comparatively low in the metamorphic tracts of Kuttunghee, in the Seonee district, and the tracts of the same formations and of the sandstones that are found in the district of Jubbulpor.[31]

> Cholera fell with the greatest severity in the villages in the trap formation, where the water-supply is derived either from shallow surface wells, sunk in porous material, and in situations where the water is especially liable to be contaminated with sewage matter, or from streams which in the hot weather contain water that is nearly stagnant. Again, the disease prevailed severely in the alluvial plain of the Jubbulpore district, where a large proportion of the villages are dependent for their water-supply on rivers which had been polluted by dead bodies thrown into them, on smaller streams the banks of which are habitually fouled, or on tanks that receive the surface drainage of the village area.[32]

Cholera deaths were concentrated in the villages that were situated on 'the tops of rocky ridges', 'high open plateaus' or 'hard impermeable rock' in

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[31] S.C. Townsend, ***Report on the Cholera Epidemic of 1868,*** 1869, p. 70. At that time miasamatic causation theory of cholera was still dominant in Indian Medical Service (See M. Harrison, 'Cholera Theory and Sanitary Policy' in M. Harrison, ***Public Health in British India: Anglo-indian Preventive medicine, 1859-1914***, Cambridge, 1994). But Townsend was a proponent of waterborne causation theory of cholera.

[32] ***Ibid.,*** p. 72.

the trap formation. Water was not easily available in those villages because sub-soil water was non-existent and only available surface water was often contaminated with sewage. Furthermore, the villages in the alluvial part of this region were also severely affected by cholera. Water resources from rivers, small streams or tanks were often polluted. On the other hand, the mortality rate of cholera was low in the metamorphic formation.

> In the trap formation the villages are situated on the tops of rocky ridges, or on high open plateaus; they are almost invariably built on hard impermeable rock, bare of soil, and where sub-soil water is non-existent; in fact, drier, healthy sites could scarcely be found anywhere. It would appear then from the extreme severity with which cholera prevailed in villages under these conditions that elevation and dryness of site are no protection against the invasion of the disease.[33]

Scarcity of water is a key factor in the intensification of cholera epidemics. Usually cholera epidemics reached their climaxes 'in May and the early part of June', the hot and dry season just before monsoon rain. This was a season of cholera epidemics. But in years when plenty of rain fell in the early months 'the progress of the epidemic was slow, and it was confined within a comparatively narrow area'.

> In former epidemics cholera has commonly made its appearance early in the year, in March or April, and has reached its maximum of diffusion in May and the early part of June, when, in consequence of the rapid drainage that is characteristic of the water system of the country, the water-supply is scanty.[34]

> [I]n 1867 the rain-fall throughout the country had been excessive, and that this excess was most marked in the district of Jubbulpore and in the adjoining districts of Dumoh, Nursingpore, and Mundla; and not only this, but in the early months of 1868, the rain-fall had been above the average. In the hot weather of 1868, therefore, the water-supply of the country must have been more plentiful than usual. In the presence of this more abundant supply of water the progress of the epidemic was slow, and it was confined within a comparatively narrow area.[35]

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[33] ***Ibid.,*** p. 70-71.
[34] ***Ibid.,*** pp. 71.
[35] ***Ibid.,*** pp. 71-2.

Condition of water was the most important factor. Needless to say, cholera broke out in urban environments as seriously as in the rural areas. Condition of water determined cholera infection. The introduction of filtered water reduced the risk of cholera infection. The case of Calcutta shows that cholera mortality drastically decreased after 1870 when waterworks were installed.

> In the 29 years 1841-69, prior to the introduction of good filtered water, the average death-rate was 4,575 annually. For the 14 years 1870-83 the average is 1,432, demonstrating how enormous the benefit of good water is in diminishing mortality from this disease, and how urgently its complete and sufficient distribution is required throughout every corner of the Town. The great fall in mortality was coincident with the very month on which the water was freely given.[36]

On the other hand, the slum area (bustee) where the poor classes lived, however, was 'insufficiently provided with hydrants' of filtered water.

> It has been pointed out elsewhere in this report that two-thirds of the total number of cholera cases in the Town occur in bustees, and it is quite possible that the explanation of this fact may be partly due to the want of pure water, in the absence of which recourse is had to foul tanks and wells for even domestic purposes.[37]

Cholera epidemics occurred explosively in the semi-arid zone where annual rainfall was 20-40 inches (508-1,016 mm). Again, 'nutritional factor' is important. Food shortage possibly affected lethality of cholera. Famine was often followed by cholera epidemics. Of course, as we have seen, in the time of famine scarcity of water naturally became acute. Furthermore deficiency of nutrition made resistance of human bodies weaker.

4. Endemicity, Epidemicity and Immunity

In the beginning of previous chapter, we referred to endemicity of

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[36] *Report of the Commission Appointed under Section 28 of Act IV (B.C.) of 1876 to Enquire into Certain Matters Connected with Sanitation of the Town of Calcutta*, Calcutta, 1885, p. 10.
[37] *Ibid.*, p. 10

cholera. We now closely consider this aspect. Endemic area of cholera was limited in terms of space. It consisted of Bengal, Orissa, Bihar, and some parts of Madras Presidency (deltaic area). Commonalities among these areas are as follows.[38]

- Being located generally around big rivers
- High population density
- Lowlands (height of not more than 150 meters)
- Absolute humidity

Why was cholera endemic in these areas? How was fecal-oral route of cholera infection related to these ecological conditions? In these areas it was difficult to dig deep wells because the land was waterlogged. Therefore people had to get drinking water from surface water such as tanks or rivers. There was much possibility that people drank water contaminated with ***Vibrio cholerae*** in these areas. There always remained moderate type of cholera. As there was a herd immunity in these areas, only factors like food shortages or influx of non-immune laboures caused epidemic manifestations.[39]

On the contrary, in epidemic areas of cholera in India, mostly semi-arid zone cholera was usually brought in from outside. The type of cholera was apt to be fulminant (explosive) in these areas. Epidemics occurred mostly in the period just before south-west monsoon when water is most scarce and in the early monsoon period when human wastes on the surface flowed into tanks or rivers and contaminated water.

In the case of malaria too, to distinguish endemicity and epidemicity is effective. In endemic areas of malaria there was a herd immunity. For example, Duars was a portion of the 'terai' land that stretched along the eastern Himalayas. This was a 'hyper-endemic' area where people living there had often suffered from malaria from infancy and had acquired certain degrees of immunity. Symptom of malaria was relatively moderate there. The problem in this area was not malaria among the indigenous people who had lived there for generations, but rather severe infection among the immigrants to the tea plantations. Intense malaria epidemics sometimes attacked this area, a problem that resulted from the continuous entry of non-immune immigrants

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[38] R. Politzer, C***holera***, Geneva, 1959. p. 825.
[39] ***Ibid.***, p. 825.

into the tea estates and their poor living conditions.[40]

Epidemic malaria was intermittent, but lethal at the time of famine. This contrast between epidemic and endemic malaria can be mainly explained by 'immunity factor'.

5. Concluding Remarks

As is generally known, Alfred W. Crosby introduced 'immunity factor' into the narrative on the global history of disease. In the 16th century devastating epidemics like smallpox, measles, and influenza assaulted indigenous peoples in the new continent [Americas] after the start of Spanish colonization. The most important factor is that the new world became connected with the old world for the first time, and became integrated into the disease pool of Eurasia.[41] It was pointed out that 'virgin soil epidemics' occurred.[42] The population of the American continent declined substantially. For example, it is estimated that the population in central Mexico decreased from about 6 million in 1548 to around 1 million in 1608.[43] The globalization caused the most tragic result in the global history of disease.

On the other hand, during the same period areas around the Indian Ocean where the Portuguese conducted commercial activities did not meet any epidemiological disasters. Rather the Portuguese suffered from the local diseases encountered on the East African coast.[44] 'Immunity factor' advantaged South Asia during this period.

After three hundreds years later, a similar nexus between globalization and epidemic diseases occurred in Asia. From the middle of the 19th century to the interwar period international trade between Asia and Europe increased significantly, and Intra-Asian trade picked up as well. Further, the movement of labor from India and China to Southeast Asia accelerated following the growth of trade. During this period some Asian regions faced very severe 'health crises'. These were particularly rampant from the 1870s to the 1910s in South Asia. Although the story was quite different from

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[40] S.R. Christophers and C.A. Bentley, ***Malaria in the Duars***, Simla, 1911.
[41] A.W. Crosby, ***The Columbian Exchange: Biological and Cultural Consequences of 1492***, Westport, Connecticut, 1972.
[42] A.W. Crosby, ***Germs, Seeds and Animals: Studies in Ecological History***, Armonk, 1994, pp. 97-109.
[43] M. Livi-Bacci, ***A Concise History of World Population***, Massachusetts, 2001, p. 46.
[44] M. Pearson, ***Port Cities and Intruders: The Swahili Coast, India, and Portugal in the early Modern Era***, Baltimore, p. 139.

that three hundreds years ago, ‘immunity factor’ proved to be important in the South Asian case.