The Epidemiology of Leprosy

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1. Introduction.

In this paper I attempt to outline in a general way some of the chief facts and theories regarding the epidemiology of leprosy. I am, however, not an epidemiologist, and also knowledge of this aspect of leprosy is not very extensive or accurate.

Epidemiological studies of leprosy are handicapped because statistics collected by health authorities, census officers, etc. are so inaccurate as often to be very misleading, and also because we have no test which indicates with any accuracy susceptibility or immunity to leprosy. The leprosy worker has to collect his own information and statistics by direct observation of the disease in the peoples affected.

The epidemiological study of any disease involves a study of its history. Leprosy is a disease which is constantly referred to in literature from the most ancient times to the present day, and from a study of these records classical scholars have tried to piece together a history of the disease.

The following is an outline of this history based largely on that given in Rogers and Muir's Leprosy.

2. Outline of the history of leprosy.

This outline is of doubtful accuracy. The disease possibly originated in Africa. In pre-historic times it spread to India and Egypt and a very early reference to it is in the Vedas dated about 1400 B.C. In the Eber's papyrus about 1550 B.C. there are descriptions of skin diseases under the names "Uchedu," "Chon's swellings," and "Anut of Chon's swellings," the symptoms of which correspond very closely to those of leprosy. There is a questionable reference to it in the Egyptian records of 1350 B.C. in the reign of Ramesis II. Munro reads the records as indicating the presence of leprosy in Negro slaves brought from the Sudan.

Whether this reading is accurate or not, there is no doubt that in ancient as well as modern times invasions and the slave trade have been important factors in the spread of leprosy from one country to another.

Thus in very ancient times leprosy was common in Central Africa, India and possibly Egypt and in these areas it is common to this day.

From India leprosy spread eastwards. The oldest
Chinese medical writings give no definite indication of its presence, but in the writings of about 100 to 200 B.C. there are definite references to leprosy.

From Egypt the disease spread round the eastern Mediterranean. The Jewish writings of the Bible contain many references to a disease "Zaraath," which is described in some places in the Bible as being highly contagious, producing patches white as snow, and being fairly readily curable. Other references in the Bible indicate the incurability of leprosy. Possibly under the one term "Zaraath" are included many skin diseases, such as psoriasis and leukoderma, as well as leprosy.

In 150 B.C. when the Jewish writings were translated into Greek, the word "Zaraath" was translated into the Greek term "lepra" which is used in the Hippocratic writings for a scaly disease. It is probable that in the Hippocratic era there was no true leprosy in Greece for Hippocrates described no such disease, but Aristotle about 345 B.C. has references to the disease which may, therefore, have been found only in Asia Minor. Factors which may well have contributed to the spread of leprosy were the conquest of Egypt by Cambyses, 525 B.C., the conquests of Darius in the same century and later in 480 B.C. the conquests of Xerxes. According to Herodotus, Xerxes led 6,000,000 people from Asia into Europe, many thousands of whom remained in Europe. When true leprosy appeared in Greece the term "lepra" was not applied to it but the term "elephantiasis" was used. The use of the word "lepra" for what we now know as leprosy probably originated in a mistake in translation. In early medical writings in Arabic the term "djudaum" is used for leprosy, and when these writings were translated into Greek by Constantine of Carthage in the tenth century, the word "lepra" was wrongly used, but the name has stuck. The Greek term for leprosy, elephantiasis, is still used in some medical writings and in Calcutta when the diagnosis has to be written without the patients or others knowing, the initials E.G. (Elephantiasis Graecorum) are often used.

Leprosy was unrecorded in Roman writings until the time of Pompey, when it is recorded as having occurred in the soldiers returning from the East in 62 B.C. In Roman history from then onwards leprosy is often referred to. The Romans introduced leprosy into other parts of Europe. Galen wrote of it in Germany in A.D. 180 and Virchow reports that by A.D. 600 there were hundreds of leper houses in Italy and Germany. In the fifth and sixth centuries Spain
was infected by Roman troops. After the fall of Rome, the conquest of Alaric and others probably helped to spread the disease. From Spain leprosy spread to France. (The Saracens invaded France from Spain early in the eighth century.) In 1757 laws were passed prohibiting the marriage of lepers and decreeing divorce of lepers.

Leprosy was probably introduced into England by the Romans. Sir G. Newman records that the first leper house in England was founded in Nottingham in the seventh century. The following are the dates of the establishment of the first leper houses: — Ireland 869, Wales 950, Scotland 1177 or 1300 (Newman). Meanwhile Norway was infected in 1266 and Shetlands, Faroe, Iceland, Greenland, Holland, Denmark, Sweden, Russia, the Baltic countries had also been infected.

Leprosy reached its height in Western Europe about 1200 though it was very common from A.D. 1000 to 1400 (2,000 leper houses in France alone). The influence of the Crusades on the incidence and the spread of leprosy has been discussed, but Newman thought that they only affected leprosy by impoverishing Western Europe.

So far the story has been one of the introduction of leprosy into previously unaffected countries, and of its spread in such countries. In the thirteenth century, however, leprosy began to decline in Western Europe and by the seventeenth century it had more or less died out in this area excepting in a few persistent foci. The "epidemic" in Europe had lasted about one thousand years.

The reasons for the dying out of leprosy in Western Europe have been discussed at great length by various writers, but no really satisfactory explanation has been given. Some students of the subject have considered that the isolation carried out in "leper houses" was responsible (there were thousands of such institutions in medieval Europe). Climate and meteorological changes have recently been quoted as important factors. Improved social and hygienic conditions and diet have been cited. A few writers have suggested that the Western European races gradually became immune because of the gradual dying out of the stock which was susceptible to leprosy. Other writers have considered that the tremendous mortality associated with the great plagues of the Middle Ages was an important factor. Some of these factors are discussed later. Other writers think that the reasons for the decline of leprosy in Europe are undetermined and possibly undeterminable, and quote similar phenomena seen in other diseases, such as tuberculosis and plague,
such diseases dying out gradually or suddenly with no apparent cause, sometimes when conditions appear to be most favourable for their spread. One striking example of this is the steady decline in tuberculosis in England which began about 1800 just when the industrial revolution created conditions apparently favourable to its spread. This decline started long before the establishment of any organized anti-tuberculosis work.

I think we must admit that we have no satisfactory explanation of the decline of leprosy in Europe.

While leprosy was declining in Europe it was being conveyed to other previously uninfected countries: to North America and the West Indies by immigrants from Europe and by slaves from Africa, and to South America by immigrants from Spain. In South America leprosy is still widespread, but in North America there are only a few foci of leprosy.

Even within the last half-century leprosy has been introduced into previously uninfected countries, chiefly Pacific Islands. The story of leprosy in Nauru is well known to students of leprosy. Leprosy was introduced and for some years spread very little. After the influenza epidemic of 1918 however (in which an incidence of 100 per cent and a mortality rate of 30 per cent were seen) leprosy spread widely and in a few years about 20 per cent of the population was affected, the disease, however, being in a comparatively mild form. The epidemic was short-lived and is now on the decline.

The history of leprosy is, on the whole, a history of endemicity of the disease in some parts of the world for thousands of years with its introduction from time to time into other parts where it may be seen in the form of very long period epidemics, dying out in time for no apparent reason.

3. Transmission of leprosy.

This matter can only be discussed very briefly here. The only mode of transmission of leprosy about which there is any certainty is transmission by direct contact with infectious cases. Even so we do not know the exact way in which the bacilli get into the body, but most workers consider that it is probably through abraded skin or through mucous membrane.

Transmission by contact with infected articles may be seen occasionally. It is extremely doubtful if the disease can be conveyed by infected air, water or food. Insect transmission appears on the whole to be improbable.
4. The present distribution of leprosy in the world.

Leprosy is now regarded as a tropical disease and it is found chiefly in tropical countries but it still exists to a considerable extent in some non-tropical countries. It will be clear from the history of leprosy which I have given, it used to be very prevalent in many non-tropical countries. The present distribution of leprosy in the world is shown roughly in the accompanying map I. Light shading indicates presence of leprosy but in small amount, less than 1 per mille. The more heavy shading indicates a moderate incidence of leprosy, probably between 1 and 2.5 per mille. The black portion indicates an incidence of leprosy of more than 2.5 per mille. In the black areas the incidence is usually from 5 to 10 per mille but in some areas, such as portions of Central Africa, the incidence may rise as high as 50 per mille. The map shows that the chief foci of leprosy are Africa, India, China and South America, that the heavily affected areas are nearly all in the tropics but that leprosy is found sometimes a long way from the tropics and even inside the Arctic circle, for example, Greenland, Iceland, Norway, the Baltic countries and Canada.

5. Distribution of leprosy in India.

This is shown roughly in the accompanying map II. It will be seen that the areas with a high incidence are in the east and south, that central and western India and the Himalayan areas show a moderate incidence while the north-west of India is relatively free from leprosy. The most heavily affected areas are probably West Bengal, South Bihar, Orissa and Madras.

6. Incidence.

Until about 10 years ago the only information regarding incidence of leprosy in India as a whole was that available in the decennial census, which in 1921 reported a total number of 102,000 cases of leprosy, giving an incidence of .35 per mille. The highest incidence reported in any area was about 1 per mille. During the last 10 years much leprosy survey work has been done in various parts of India and it is found that the census figure for many areas needs to be multiplied by a factor which varies between 3 and 20 and averages about 8. The census return for 1931 shows a figure of 147,911, an increase on the 1921 figure of 45,398. This increase possibly does not represent a real increase of leprosy but only an increase in the accuracy of the return. On the basis of our survey findings we have concluded that
the number of cases of leprosy in India is probably not less than one million. We find, in the heavily infected parts, large areas where the incidence may be 2 per cent of the population. We find small areas where the incidence is 5 or 7 per cent. We find villages in which the incidence is 15 or 20 per cent. It is on the basis of these survey figures that the accompanying map II has been made.

7. Types of leprosy.

These high figures should however be explained a little. In survey work in India we find that on the whole there are about two relatively mild cases of leprosy to every severe case. In some of these mild cases, the disease is of little clinical or public health importance and in quoting these high figures these facts should be borne in mind.

8. The age distribution of cases of leprosy.

The age distribution of cases of leprosy in India reported in the 1921 census is shown in the accompanying graph 1. The highest number of cases is found in the middle age periods. This graph is, however, of little value in indicating the age in which the disease is contracted because leprosy is a very chronic, often non-fatal disease, which exists for many years, and a middle aged or old person suffering from leprosy may often have contracted the disease quite early in life. An indication of the age at which the disease is contracted is given in graph 2 which gives the age at which the first symptoms were noticed, in 400 unselected cases of leprosy of all ages. It will be seen that in most of the cases the symptoms were recognized by the patient before the age of 30. Slight symptoms of leprosy may be present for years before they are recognized, and also the latent period of leprosy is long, averaging possibly three or four years, so that when these facts are taken into consideration, this graph indicates that the disease is probably contracted in the great majority of cases either in childhood, adolescence or early in adult life. The chances of the disease being contracted after the age of thirty are remote, though such cases are seen rarely. The difference in susceptibility at different ages is shown by a study of leprosy in families. Adults exposed to infection show an incidence of about 5 per cent, while children similarly exposed to infection show an incidence of about 50 per cent or more. Another finding which is made from a study of leprosy at different age periods is that leprosy contracted early in life is far more likely to take a serious form than when contracted later in life.

It is a curious and interesting fact that in every country where leprosy is common the number of males suffering from the disease is much greater than the number of females, the proportion averaging about 2 to 1. It may be thought that these returns are due to the difficulty of examining women properly, particularly in such countries as India, but I think there is no doubt that this is not so. Similar returns are made where there is no difficulty whatever in examining women. For example, in New Guinea, where males and females always wear the minimum of clothing and where the whole population is periodically examined naked for leprosy, the incidence in males is twice as high as in females. In the same area it is found that the disease also tends to take a milder form in females than in males. Similar findings have been reported in many other countries. It therefore appears to be quite definite that males suffer from leprosy more commonly and probably more severely than do females.

It is interesting to study the sex incidence at different age periods. This has been done in various centres. It is found that in the early years of life the incidence in the two sexes is about the same and in some countries it has been found that after the age of puberty the incidence in females may actually exceed the incidence in males. Shortly after puberty, however, the incidence in females reaches a peak and then tends to fall quickly, while at the same period the incidence in males continues to rise for several years and then falls but much more slowly. A graph of the sex incidence of leprosy at different age periods has the form shown (see graph 3).

It is very difficult to explain the difference in incidence and severity of leprosy in the two sexes. We might for a moment consider the question of the sister disease, tuberculosis. I have reproduced here a graph 4 made by McNalty indicating the mortality rate from tuberculosis, of the two sexes at different age periods in England. (McNalty considers that the mortality rate is the most reliable index of the incidence of tuberculosis in the two sexes.) In general outline (if we ignore the early peak due to infant mortality from tuberculosis) the curves are somewhat similar to those of leprosy, the incidence of tuberculosis in females is lower than that of males at all ages except the age of adolescence and the mortality rate is also lower. Now in tuberculosis this difference in sex incidence is probably due chiefly, if not entirely, to environmental factors, namely, greater exposure to infection and to factors which predispose to tuberculosis. This is caused chiefly by industrial conditions in England.
Under other conditions the incidence of tuberculosis in the two sexes may be approximately equal; for example, in America, Robinson and Wilson investigated the incidence of tuberculosis in 20,000 industrial workers, 14,000 men and 6,000 women, and found that the incidence in women was slightly higher than in men. In India it is often said that tuberculosis is commoner in females than in males because of the unhealthy conditions of seclusion in which many women live. Therefore the reason for the difference in the sex incidence of tuberculosis is probably environmental. In leprosy, however, it is difficult or impossible to explain the lower incidence in women on this basis. Even in countries where men and women are equally exposed to infection the incidence in women is much lower.

It is a well-recognized fact that women are less susceptible to some diseases than men, but in no disease does this appear to be so marked as in leprosy. An interesting discussion on the sex incidence of the disease in general is given by Stallybrass in his book on epidemiology. I have made the following summary of this discussion:

There are differences, sometimes quite marked, in the incidence and mortality of infectious diseases in the two sexes. Some diseases (e.g., whooping cough and acute rheumatism) are reported as being more common in females, while others (e.g., pneumonia and the middle-age form of tuberculosis) are more common in males. It is also noticeable that the sex incidence of disease often varies with age.

Nevertheless, at all age periods males show a greater incidence of infectious disease and a higher total mortality rate. This difference may be due to greater exposure or to greater susceptibility. Greater exposure may possibly explain the greater mortality in males in adult life, but it is difficult to see how it can explain greater mortality in childhood. Greater susceptibility in males may possibly be caused by sex differences; males are bigger and have a more developed musculature, which may throw a greater strain on the circulatory and excretory systems and so reduce chances of recovery when attacked. Again it is difficult to understand how this can explain the greater mortality in males in childhood.

The sex factor may be physiological rather than anatomical. The sex hormones are connected with differences in endocrine activity, as shown by the greater activity of the thyroid in women. The endocrine system is intimately connected with destruction of bacteria and their toxins. It is possible that differences in endocrine function in the two
sexes may have an influence on the susceptibility to infectious diseases.

In addition to anatomical and physiological differences in the sexes there is the cytological difference. Each cell in the female contains a group of chromomeress different from the corresponding group present in the male, and it is this group that determines sex and sex differences.

Whether these anatomical, physiological, and cytological differences between the sexes have any effect on the sex incidence of disease is uncertain.

To summarize the findings regarding the incidence of leprosy in India, we find that the incidence probably averages about 3 per mille but in some areas it is as high as 20 per mille, that about two-thirds of cases are relatively mild, that the disease is usually contracted in childhood or adolescence and that males suffer from the disease more commonly than females and tend to show a severer form of the disease.


It has been suggested that the subsidence of leprosy in certain countries is due to the development of racial immunity due to a gradual dying out of the stock of the race which was susceptible to leprosy. This has been quoted as a possible reason why leprosy died out in Western Europe. Western European races have, however, at the present day no marked racial immunity to leprosy, since cases of leprosy occurring in such persons who go to countries where leprosy is endemic are not uncommon, and this is found in spite of the fact that such persons do not usually go to such countries until the age of the greatest susceptibility has passed. In India, for example, leprosy in Englishmen coming to the country in adult life is not so rare as one might imagine, and when such persons do develop leprosy the disease on the whole appears to take a rather severe form which would not indicate the presence of any marked immunity. It would appear that all races are susceptible to leprosy but there may be minor degrees of natural immunity. It is certainly true that leprosy shows itself rather differently in people of different races. In the Far East, the Philippines, Japan and Siam, for example, the disease appears on the whole to be more severe than it is in India, and also certain clinical manifestations of the disease which may be attributed to the relatively high resistance of Indians to leprosy are much more rarely seen in these Far Eastern countries. These differences may possibly be attributed to climatic and other factors but in countries such as Malaya or the West Indies
where the population consists of different races—Chinese, Indian and Malayan, or Indian and African—leprosy appears to show itself in forms varying somewhat according to the race of the affected person. Here climatic and other differences are largely eliminated. It does appear, therefore, that there are some grounds for believing that there are minor degrees of racial immunity to leprosy. Regarding the nature of this immunity I cannot say anything here. The question has been raised as to whether racial immunity to leprosy may partly explain the distribution of leprosy in India. Do the races of the north-west suffer much less from leprosy because they have more racial immunity than the people of the south and east? This is a question which it is impossible to answer. However we find that leprosy is quite common among Punjabis and others who have migrated to endemic areas such as Bengal, and when it occurs it may take a severe form.

11. Climate and leprosy.

Climate affects leprosy in two ways. First of all it may affect the transmission of the disease and secondly it may influence the course of the disease. It is certainly noticeable that leprosy is most common in hot humid climates and it is possible that the humidity is of more importance than the temperature. A study of the maps of India and of the world which I have shown indicates how in dry parts, even in the tropics, leprosy is usually uncommon, whereas in moist parts of tropical and non-tropical countries leprosy may be more common. The reasons for this are not clear. One curious thing is that in some countries, where there is a considerable number of imported cases of leprosy, many of them infectious and few of them isolated, examples of such cases infecting others are extremely rare. For example, in England there are probably about 100 cases of leprosy, many of them infectious and many not isolated, but contact cases are extremely rare. The same thing is found in New York city. Climate may also have an effect on the disease and the experience of some physicians who have dealt with leprosy in tropical and in cold countries indicates that a cold country is not favourable for the treatment of leprosy. Thus the climate of countries such as England apparently does not favour transmission of the disease, but it does favour the development of the disease when it has risen.

12. Diet.

There has, for centuries, been a common idea throughout
the world that leprosy is influenced by diet. One of the common ideas in most countries, particularly in India, is that leprosy is connected with the eating of fish. Sir Jonathan Hutchinson took up this idea and gave it as an explanation as to why leprosy was so common in Europe in the Middle Ages when the eating of dried fish was very common, and why a high incidence of leprosy persisted so long among the fisher folk of Norway. Later he modified the idea and said that leprosy was due to eating decomposed fish and other bad food. This idea has now practically no adherents, for leprosy has been found to be common among people who never eat fish. It is however, noticeable that leprosy does appear to be commoner among those peoples whose diet is ill-balanced. In China, for instance, the disease is much more common in parts where rice forms a staple part of the diet, and where protein, fat and vitamins are little taken. The same is true of India, leprosy being common among the rice-eating people of Bengal, Bihar, Orissa and Madras and less common among the people whose staple diet consists of wheat, jowar and other grains richer in protein. Leprosy is also less common among those peoples in India who take milk or milk products. Similar findings have been made with regard to leprosy and diet in Africa. These things may however be pure coincidence. The difference in the incidence of leprosy in the different parts of India may be explainable on the grounds of racial, climatic and other differences and not of difference of diet. There is no doubt that a good diet is an important thing in the prevention of leprosy and in the treatment of leprosy alone, dietetic lines has, on the whole, given disappointing results.

13. Social and hygienic conditions.

There is considerable evidence to show that the incidence of leprosy is markedly affected by social and hygienic conditions and that leprosy tends to die out when conditions are good. The improvement in social and hygienic conditions and diet is given by some as one of the reasons why leprosy died out in Western Europe, but the evidence to show that such very marked improvement occurred between the thirteenth and sixteenth centuries, the period during which leprosy died out, is not very strong.

It is interesting to compare the incidence of leprosy in people in the same country in different stages of civilization. In India, for example, leprosy up to the present has not been common among the aboriginals and semi-aboriginals who live on the whole a healthy outdoor life in very small settlements.
and who, although they are extremely poor, not being vegetarians, often take a much more balanced diet than many other people of India. Also when cases of leprosy occur among them they are commonly ostracized and may be driven out of the community. Thus under primitive conditions in India leprosy is not common. The peoples in India who suffer most from leprosy are the outcastes, the low-caste Hindus and the poorer Mohammedans. I need not here discuss the social and economic conditions which are usually found in the poorer parts of an Indian village, and there seems to be no doubt that these conditions, combined with a poor diet and chronic ill nourishment, together with the effects of such infections as malaria, chronic dysentery and hookworm, have an influence on the incidence of leprosy. We should not, however, imagine that leprosy in India is confined to the lower castes. In the higher castes and among people who have better social and hygienic conditions and who live in better houses and take better diet, the disease is certainly less common, but the disease is actually found in all classes of society in India, from the very highest to the lowest. Cases of leprous servants infecting others living under good sodal conditions are far from rare, and it appears that even under the best conditions in India the presence of infectious cases is a very definite menace to healthy people, particularly young people and children.

If we try to correlate the incidence of leprosy in different parts of India with the general, social and economic conditions of the people we find it is not always easy. One of the poorer parts of India is undoubtedly West Bengal and here the incidence of leprosy is high, but it is certainly no higher than it is in some parts of Madras where on the whole the social and economic condition of the people is very much better.

The social and economic conditions of the people are of course intimately connected with diet, and many people do not take a better diet because they cannot afford it. However, a more wise use of the limited economic resources of a family would often improve the diet considerably, and a reduced consumption of carbohydrates and increased consumption of protein, fat and vitamin might render the people less susceptible to infectious diseases such as leprosy.

I think there is no doubt that in India there are two special factors which have an important bearing on the prevalence of leprosy. The first of these is religious sentiment which regards leprosy not as an infectious disease but as a visitation of the gods, a man’s fate which cannot be avoided.
Religious sentiment also encourages the giving of alms to beggars, particularly lepers, as a religious duty. This fact encourages the wanderings of enormous numbers of lepers all over India, particularly to centres of religious pilgrimage, and because of this sentiment it is impossible to prevent lepers travelling on trains and public vehicles all over India, usually without payment.

The second factor is the "joint-family system" under which a father and mother and all the married sons and their families and all unmarried children share one household. If any member of the family gets leprosy in an infectious form, all the other members of the large joint family are exposed to the infection in the joint-family house, and numbers of them, chiefly children, frequently contract the disease in this way from relatives.

14. Leprosy in rural and urban areas.

Leprosy has up to the present been chiefly a rural problem since most of the people in India are agricultural workers living in villages. During recent years the opening up of previously secluded areas by the development of roads and railways, and the establishment of large industrial areas with large numbers of workers recruited from rural areas, have introduced a new aspect of the problem. Healthy workers from rural areas are migrating to industrial centres with their families, there getting infected with leprosy and later returning and spreading the disease in previously unaffected villages.

Conclusion.

These then are some facts and some theories regarding the epidemiology of leprosy in the world and in India. There are great gaps in our knowledge and the need for further study is obvious. There are three points which are clear and although they are elementary they need emphasis. Firstly, leprosy is an infectious disease though not highly infectious, and the conditions found in most parts of India are favourable to transmission. Secondly, while most adults are partly or completely immune to leprosy children are usually susceptible. Thirdly, the most important thing in the control of leprosy is the prevention of contact between infectious cases and children.