THE BACKGROUND AND PREDISPOSING CAUSES OF LEPROSY IN POONA DISTRICT, BRITISH INDIA

T H E S I S

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by

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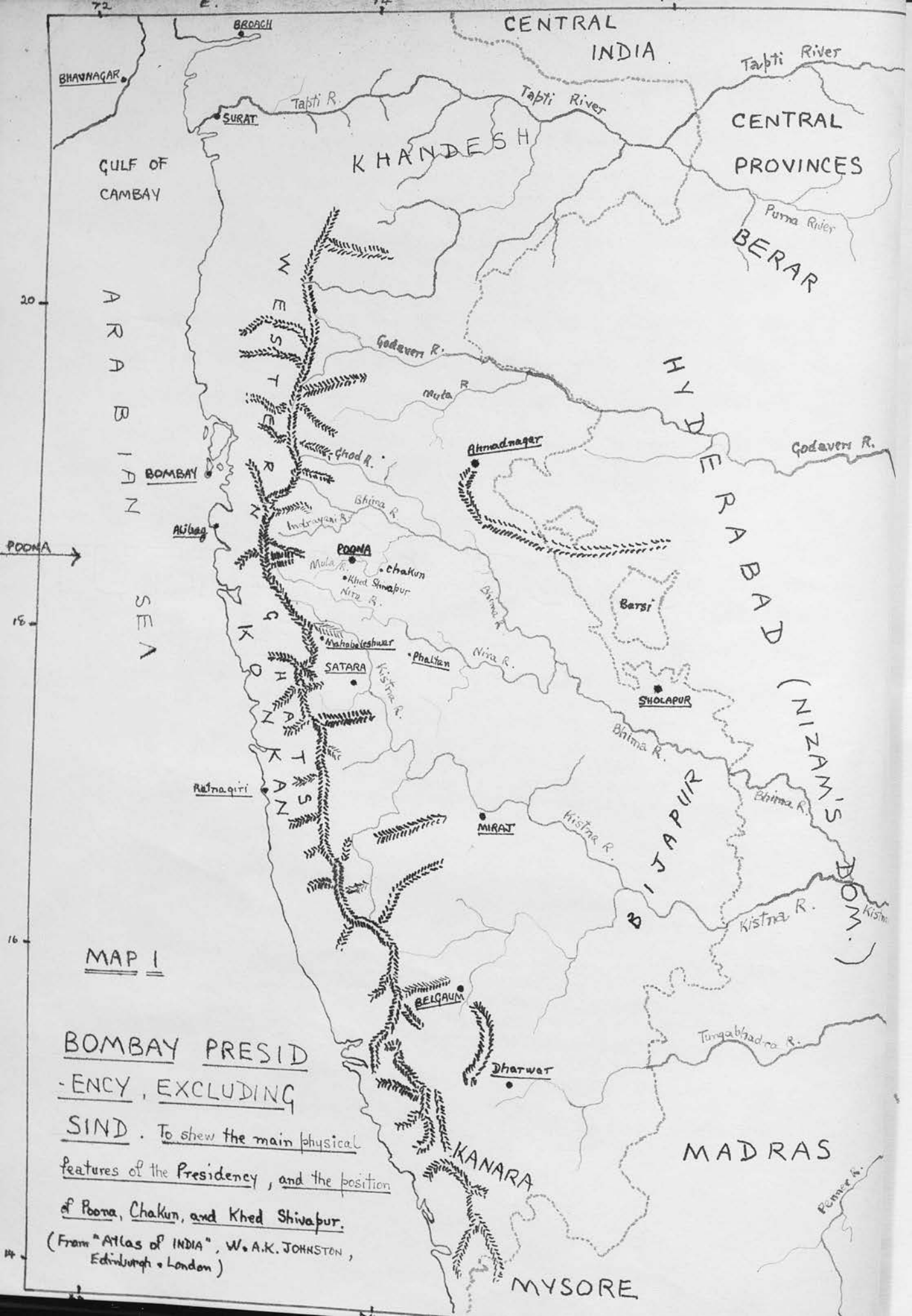
1. INTRODUCTION.

The interest of the author in leprosy was first aroused in his first voyage to India in 1928, when he had the great good fortune to have as a fellow-traveller, Dr. E. Muir, Leprosy Research Scholar to the Government of India. In various conversations, Dr. Muir described the position as regards leprosy in India, and gave an estimate of the number of lepers in India as close on one million, the Census figures in all parts being far exceeded by the figures obtained by expert surveys. MUIR and SANTRA, 1932, (1) have expressed this opinion later, that "the probable incidence of leprosy in India is not less than between 500,000 and 1 million." These truly staggering figures give good cause for the belief that a study of leprosy in any part of India is well worth while.

After study of the vernacular of the Maratha country, and after experience at the large American hospital at Miraj, where there is also a Leper Hospital with the largest "cure" rate of any in India, the author took up his work in Poona. (See Map 1). This work consisted of medical charge of the N.M. Wadia Hospital of the Church of Scotland Mission in Poona, and Medical Superintendent of the Khondwa Leper Asylum near Poona. The N.M. Wadia Hospital is a general hospital of between 1,500 to 2,000 inpatients per annum, and 15,000 to 20,000 outpatients per annum. It



MAP I

BOMBAY PRESIDENCY, EXCLUDING SIND. To shew the main physical features of the Presidency, and the position of Poona, Chakun, and Khed Shivapur.

(From "Atlas of INDIA", W. A. K. JOHNSTON, Edinburgh & London)

has also two village dispensaries which are visited weekly - one at Khed Shivapur at 15 miles distance, and the other at Chakun, 21 miles away. (See Map 1). The hospital is well-equipped, and has a good laboratory, and its wealth and variety of clinical cases provide a valuable aid to the study of the background of leprosy in the District. The Leper Asylum at Khondwa is under the direction of the Government of Bombay in part, and the Mission to Lepers in part and is occupied chiefly by 'statutory lepers,' that is, leper vagrants who have been gathered in by the police, or pauper lepers who have been declared as such before a magistrate. It will be understood that the majority of the inmates are in an advanced stage of leprosy, and not likely to afford a high discharge rate. Nevertheless, they presented good material for prolonged investigation in leprosy. The asylum has a resident Indian medical officer, a lay superintendent, and contains on the average from 120 to 140 lepers.

Another aspect of leper work was soon revealed in the N.M. Wadia Hospital itself. Every year there were a number of lepers discovered in the Outpatient Department, and even among the patients admitted to hospital. In the latter case, patients admitted for other complaints were found during the completer physical examination in hospital to be lepers. In the year 1929, there were 5 such: in 1930, four: in 1931, seven: in 1932, three: in 1933, three. All these cases were found to be bacteriologically positive

by nasal or skin examination, but with only slight external manifestations of leprosy. Of more obvious lepers, a greater number annually was detected in the Outpatient Department, and in the village dispensaries at Khed Shivapur and Chakun. These facts led in 1930 to the establishment at the hospital of an Outpatient Clinic for lepers, and treatment centres were opened in the two village dispensaries. At the hospital, starting with 2 patients, the number grew until in 1933 some 70 lepers were attending, mostly of the early stage of the disease. This clinic served to balance the less hopeful 'refuge work' type of case at Khondwa. An outpatient leprosy clinic has its disadvantages, in that the lepers are not segregated, and in that they have to meet in the grounds of a 'clean' hospital in the midst of a crowded city, but it was considered justifiable because of the inadequacy of Khondwa Asylum to contain them, the great opportunities of study of early lepers, the opportunities for propaganda among the more intelligent type of lepers about personal and public hygiene in leprosy, and the greater incidence of arrest of the disease.

During the years 1929 to 1933, the lepers met with in the Khondwa Asylum, in the clinics at the hospital, in the village dispensaries, and in various villages of Poona District, have been studied. Understanding has been sought as to how and why these lepers got the disease, and the facts of epidemiology, etiology, and predisposing diseases and causes have been given the chief attention.

GEOGRAPHICAL FEATURES.

Poona District[2] lies in the Central Division of the Bombay Presidency of India (see Map 2), between 17 deg.54 min. and 19 deg. 24 min. N., and 73 deg. 19 min. and 75 deg.10 min. E., with an area of 5,349 square miles. It is bounded on the north by the District of Ahmadnagar, on the east by Ahmadnagar and Sholapur, on the south by the Nira river, separating it from Satara and Phaltan, on the west by Kolaba District. Poona District lies entirely in the Deccan.[3] The Deccan commences south of the Tapti river, (see Map 1), a great three-cornered upheaval plateau, sloping gently from the Western Ghats at 3,000 feet to the Eastern Ghats. It consists of wide rust-coloured plains streaked and dotted with hills, with intervening bare patches of stunted forests. Black soil, known locally as 'black cotton soil,' prevails near the rivers, and as there is no more productive soil in India, numerous villages of an agricultural people are to be found near them. The highlands are drained by the Godaveri, Kistna, Penner, and Cauvery rivers, with their countless tributaries. Fringing this plateau on either side is a narrow strip of coastal land comprising the lowlands of Bombay on the west, and those of Madras on the east. The Western Ghats have their seaward slopes clothed with dense jungle, and the lowland coastal strip is known as the Konkan, Kanara and Malabar. Towards the west of Poona District itself, the country is undulating and intersected by many spurs of the Western Ghats, which

break off in a south-easterly direction, becoming lower as they pass eastwards, and in the end flattening to the general level of the plain. On the extreme western border the land is so rugged and cut up by valleys, that on the slopes and sides of the hills a system of spade tillage is necessary. Many rivers rise in the western hills, and flow eastwards, until they join the Bhima river, which passes through the District from northwest to southeast. The main tributaries are the Vel, Ghod, Bhama, Indrayani, Mula, and Nira. The water of the rivers is considered good for all purposes by the people. Poona city and cantonments are well supplied with water from six artificial lakes, and two canals, but there are defects leading to much seepage water, and consequently hyperendemicity of malaria in many parts of the town. The rivers are well stocked with fish, there being about 30 kinds obtainable in Poona markets.

The height of the Poona plateau is 1,800 feet[4]. This fact, and its freedom from alluvial deposits, and the prevalence of westerly breezes, and its dry invigorating air, make Poona popular with Europeans. The air is lighter, the heat less oppressive and the cold more bracing than in other parts of the Presidency, except Mahabaleshwar (4,500 feet). The cold season is from November to February, the hot season from March to June, and the wet or monsoon season from June to October. Cool land winds

prevail during the cold season, with sea breezes mostly after sunset. Hot winds are characteristic of the hot season, but they are over by the middle of May, and occasional thunderstorms, with heavy rain or hail, may temporarily cool the air. The temperature falls to 48 deg. F. in November in the night and early morning, but in the middle of the day may rise to 90 deg. F., even in the cold weather. The temperature is highest in May, but is rarely over 107 deg. F., with a night temperature of 95 to 100 deg. F. The Southwest Monsoon begins about the middle of June and lasts to the end of September, and it is only during the monsoon that the relative humidity is over 80%. For the rest of the year, Poona is very dry.

The rainfall (Map 2) varies considerably in different parts of the District. In the western parts, where it abuts on the Ghats, in a limited area the rainfall is heavy and regular. In the central belt it is moderate, and in the east it is very irregular. At Lonauli on the Ghats it averages over 185 inches annually. In Poona City, 32 inches is the average, while further east it does not exceed 20 inches in places. ROGERS, 1932, [5] has published maps of the leprosy ratios per mille in different countries of the world, and of the annual rainfalls in the same parts, which show a remarkable relationship between high leprosy incidence and high rainfall, especially in tropical climates. Whether this holds for Bombay Presidency and Poona District

will be considered later.

With much of its rainfall cut off ~~its rainfall cut off~~ by the western hills, large tracts of land in the east of Poona District have a very uncertain water supply.(6) See Map 2 and Fig 2. During the last 500 years there is either traditional or historical mention of at least 25 famines. The fact that the dry parts of Poona District are facultative famine areas tends to negative the expectation of low leprosy incidence associated with low rainfall. E. MUIR and I. SANTRA, 1932,(7) in their survey of India found leprosy incidence high in famine areas, especially those with a porous quickly drying soil such as laterite or black cotton soil. As the surface water is low they depend for their crops upon water supplied by the monsoons, any failure or irregularity of which is apt to produce famine conditions. MUIR and SANTRA instance Western Bengal, and the same applies to much of Poona District.

As regards the general healthiness of Poona, owing to its comparatively agreeable climate, it has long been regarded as a health station. In the past 20 years this has begun to be doubted, (8) and with reason, for there is a high incidence of malaria, of bacillary and also amoebic dysentery , and sprue is not uncommon. "Poona tummy," which is a Flexner type dysentery, afflicts practically every newcomer to Poona. Poona is moreover one of the plague spots of India, and in 1933 there was a bad outbreak

of plague.

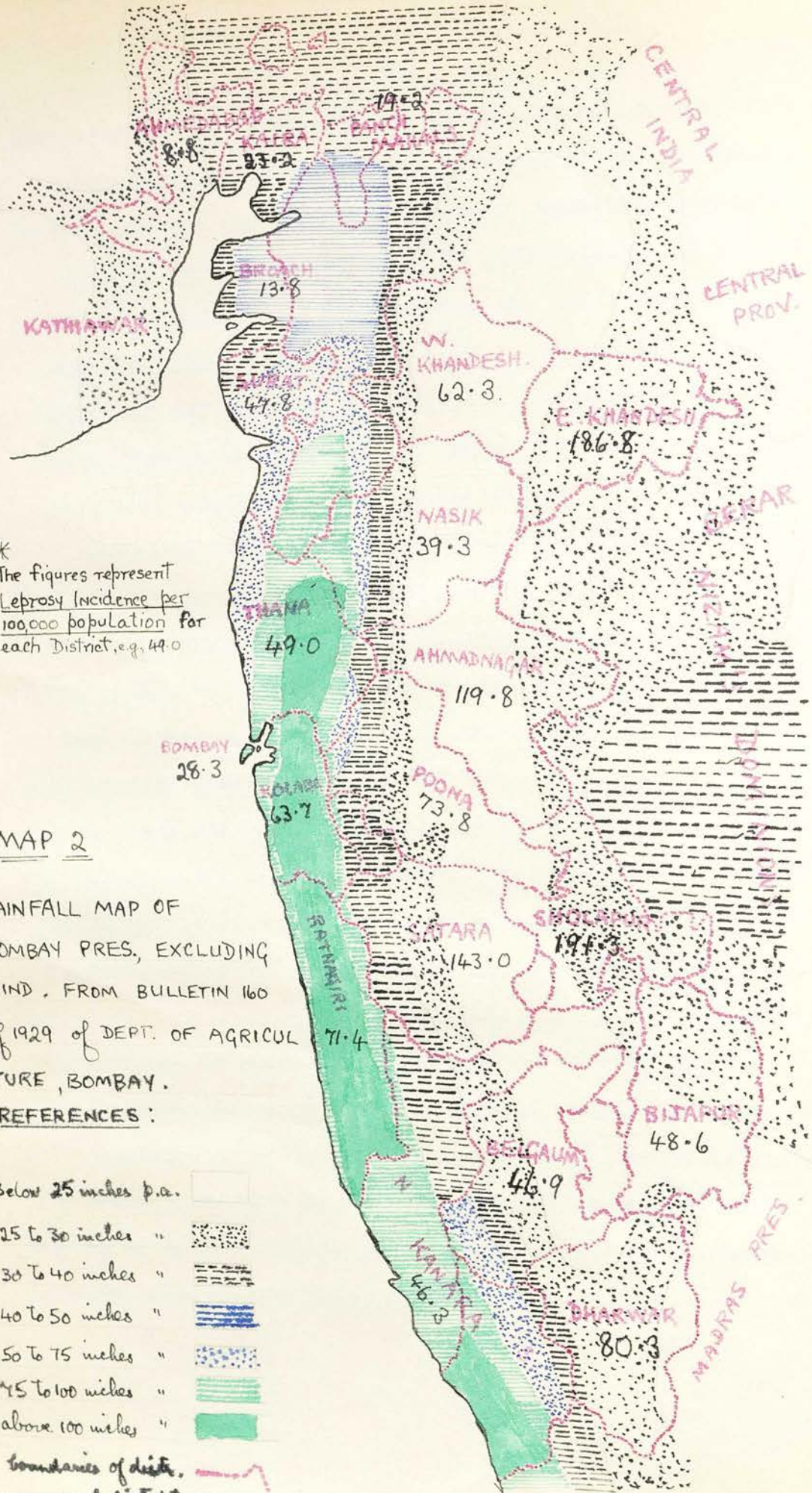
ETHNOGRAPHY. (9)

The peoples of India are distinguished provisionally as belonging to three main classes, Aryan, Dravidian and Mongoloid. Seven main physical types compose these three, - the Dravidian, the Turko-Iranian, the Indo-Aryan, the Scytho-Dravidian, the Aryo-Dravidian, the Mongolo-Dravidian, and the Mongoloid. Of these, only three need concern us. The Dravidians may be taken as the earliest inhabitants of India of which we have any knowledge. It is the oldest and most primitive of the types. The Dravidian has a black skin, squat figure, long head, and rather flat nose. The type which is found all through and predominates in Poons District is the Sycho-Dravidian. The Maratha Brahmins, the Maratha Kunbis or agricultural caste, the Ramoshis or watchmen caste, the Dhangars or shepherds, and the Mahars and Mangs (outcastes), are all varying admixtures of the Sycho-Dravidian, the lower castes and outcastes being more Dravidian. The Scythian element is supposed to have come from Scythian invaders from Central Asia. At one end of the scale there are the fair and intellectual Brahmins, some even grey-eyed, and at the lower end of the scale the black and stunted Mangs and Mahars. The Aryo-Dravidian or Hindustani type is also found to some extent in the District, mainly as traders and shopkeepers, e.g., the Marwadis.

This type really belongs to the United Provinces, Rajputana, and Bihar, and is probably the result of admixture, in varying proportions, of Dravidians and Indo-Aryans. According to I. SANTRA, 1933, (10) the responsibility of each racial element for the spread of leprosy to the other is difficult to assess. He points out that while all the Indo-Aryan tongues have a word for leprosy, the Dravidian have none, thus suggesting that leprosy was brought to the Dravidians. In this connection it is noteworthy that CILENTO and NORTH (11) state that leprosy in Australia, after introduction by the Chinese, spread first among the aborigines, including the 'myalls' or wild aborigines. The aborigines are comparable to the Dravidians of India, and it is possible that something like what happened to them happened in India. The inhabitants of Poona District are by a large majority Scytho-Dravidians, and the Kunbi or Maratha farmer forms the great bulk of the population. There are of course small numbers of Marawadis, Europeans, Parsis, South Indians and Mahommedans of very mixed origin.

CASTE.

A caste may be defined as a collection of families or groups of families, bearing a common name, which usually denotes, or is associated with, a specific occupation; claiming common descent from a mythical ancestor, human or divine. A member of the large circle denoted by the common name of the caste is invariably prohibited from marrying



* The figures represent Leprosy Incidence per 100,000 population for each District, e.g. 49·0

MAP 2

RAINFALL MAP OF BOMBAY PRES., EXCLUDING SIND. FROM BULLETIN 160 of 1929 of DEPT. OF AGRICULTURE, BOMBAY.

REFERENCES:

Below 25 inches p.a.
25 to 30 inches "
30 to 40 inches "
40 to 50 inches "
50 to 75 inches "
75 to 100 inches "
above 100 inches "
boundaries of dists.
names of districts POONA

outside that circle, but within the circle there are often a number of smaller circles to each of which the same prohibition applies.

INCIDENCE OF LEPROSY IN POONA DISTRICT AND SURROUNDINGS.

CHOKSY, 1930,[12] reports on the incidence of leprosy in the Bombay Presidency, from data gathered by the Government of Bombay in a survey by revenue and police officials. Because it was by a lay agency, it takes note of visible cases only. The Central Division of the Presidency, in which Poona District lies, was by far the most heavily infected.

TABLE I.

Rate of incidence per 100,000 in the Central Division.

Sholapur District	191.3.
Satara District	143.0.
Poona District	73.8.
Nasik District	39.3.
E. Khandesh District	186.8.
Ahmadnagar District	119.8.
W. Khandesh District	62.3.

In Poona District, with a population of 1,009,033., 254. villages were found affected, and 754 lepers found. In Poona City itself, 150 lepers were found. In the Presidency as a whole, the number of lepers in a population of 17,962,965. was 11,568., equivalent to the ratio of 64.3. per 100,000 of population. CHOKSY suggests that we should take for our guidance the experience of the prevalence of leprosy in other parts of India, where it has been calculated that for every visible leper, there exist

at least 4 to 5 lepers. The leper population of the Presidency must then be somewhere between 50,000 and 60,000. and the probable ratio between 257 and 321 per 100,000. In the same way, there must be in Poona District 3725 lepers, a ratio of 369.0. per 100,000. In Poona itself there must be about 1,000 lepers, in a population of 400,000, a ratio of 250 per 100,000.

Leprosy is widely distributed in villages and hamlets. Of 4,112. villages, in the Presidency, only 271. reported no lepers in their midst.

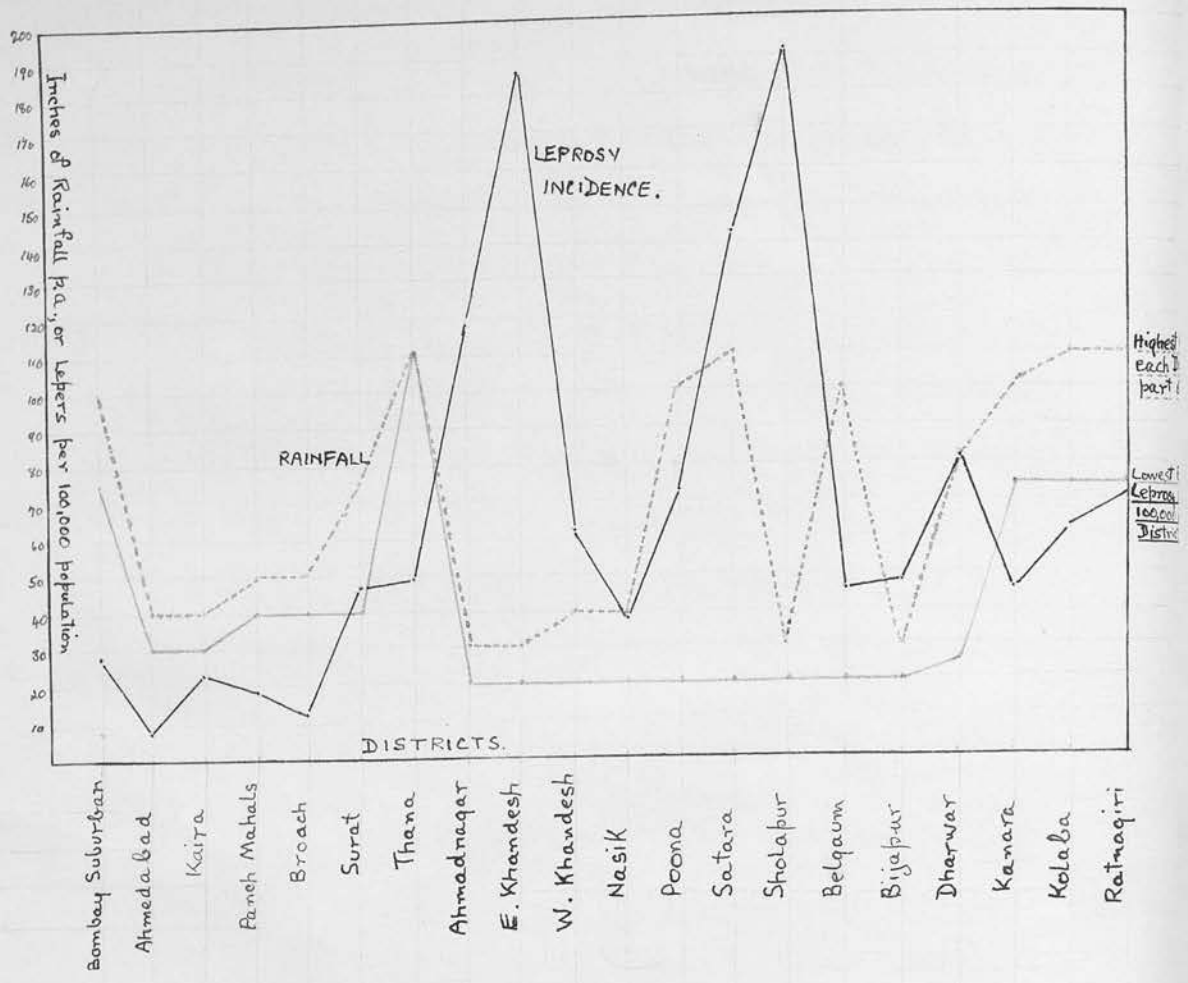
RELATION TO RAINFALL.

In view of the opinion of ROGERS (5) quoted above, that there is a remarkable relationshop between high rainfall and high leprosy incidence, let us compare the figures for the Bombay Presidency. The figures for leprosy incidence are those given by CHOSKY, (12) and the figures for annual rainfall are by PATIL, 1929. (13) See Map 2.

TABLE II.

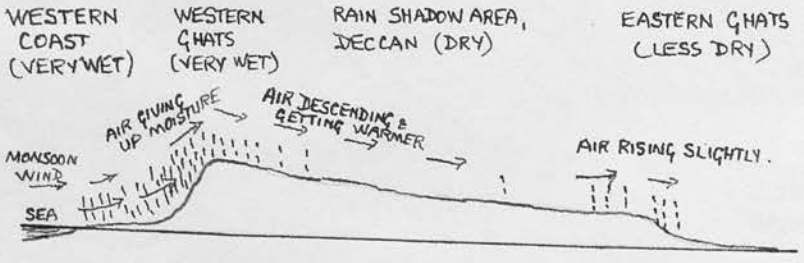
District.	Ratio of lepers per 100,000.	Rainfall per annum.
Bombay Suburban	28.3.	75 to 100 inches.
Ahmedabad	8.8.	30 to 40 inches.
Kaira	23.2.	30 to 40 inches.
Panch Mahals	19.2.	40 to 50 inches.
Surat	47.8.	40 to 75.inches..
Thana	49.0.	above 100 inches.
Ahmadanagar	119.8.	below 25 to 30 inches.
E. Khandesh	186.8.	below 25 to 30 inches.
W. Khandesh	62.3.	below 25 to 40 inches.
Nasik	39.3.	below 25 to 40 inches.
Poona	73.8.	below 25 mostly, but up to 100 in parts.

FIGURE 1.



Graph to shew degree of correspondence between Rainfall and Leprosy Incidence in Districts of Bombay Presidency.

FIGURE 2



Section across the Deccan Plateau, illustrating rainfall conditions.
(From "Asia", by L. Dudley Stamp, 1929. p. 318.
Methuen & Co., 36 Essex Strt., W.C., London.)

TABLE II. (cont.)

District.	Ratio of lepers per 100,000.	Rainfall per annum.
Satara	143.0.	below 25 in half, but up to 100 in the west.
Sholapur.	191.3.	below 25 to 30. inches.
Belgaum	46.9.	below 25 inches, but up to 100 in parts.
Bijapur	48.6.	below 25 to 30 inches.
Dharwar	80.3.	25 to 30 inches.
Kanara	46.3.	75 to 100 inches.
Kolaba	63.7.	75 to above 100 inches.
Ratnagiri	71.4.	75 to above 100 inches.

A glance at Map 2. will show that there is not a very close correspondence in Bombay Presidency between leprosy incidence and high rainfall, as Thana, Kolaba, Ratnagiri and Kanara, which have a high annual rainfall, have a lower leprosy incidence than Ahmadnagar, Satara, Poona, Sholapur and E. Khandesh, which have a comparatively low annual rainfall. In addition, the coastal climate of Thana, Kolaba, etc., is far hotter and more humid than the districts in the Deccan proper. The graph (Figure 1.) shows clearly the degree of relationship between the rainfall and leprosy rate in each District.

LEPROSY IN INDIA.

Leprosy is endemic also in all the provinces of India. (I. SANTRA, 1933)[10]. In recent years census figures, and expert surveys yielding greatly enhanced figures, have left no doubt as to the widespread endemicity of leprosy in India. Largely owing to more accurate enumeration, during the last decade the number of lepers according to the

census has increased by 44.2%. against an increase in population of 10.6%. (10). In some provinces where expert surveys have been carried out, the increase is very high, as, for example, an increase of 85%. in the province of Bihar and Orissa. (10). In addition to the expert enumeration, an increase in leprosy seems to have followed modern rapid communications, and industrialization of many parts. The 1921 Census figures for leprosy in India give an incidence of 100,000. (ROGERS, 1928) (14) , but these do not include early cases, and we have already seen that E. MUIR and I. SANTRA, 1932, (1) estimate between 500,000. and 1 million. That they have reason for such an estimate is shown by their survey of the Satara District. The previous figure for this District was 143.0. per 100,000. (12). E. MUIR and I. SANTRA (1) in 1932 visited 70. villages, examined 108,027. persons, and found 1,138 cases, which is 325. per 100,000. A few samples of the results of the like expert surveys, which have been carried out in India, may be tabulated as follows:-

TABLE III.

Place & date.	Leprosy Rate per 100,000 pop.	Authors.
Satara 1932.	325.0.	E. MUIR and I. SANTRA (1)
Cooch Behar State, 1930.	240.0.	B. N. GHOSH. (15)
Howrah Dist. 1931.	82.0.	B. N. GHOSH. (16)
Cuttack Dist. 1932.	768.5.	U. GUPTA. (17)
Kangra Tehsil, 1932.	238.3.	S. S. JAIKARIA (18)
Salbani, Midnapore Dist., 1932.	1,395.4.	K. R. CHATTERJI. (19)

MAP 3. WORLD DISTRIBUTION OF LEPROSY (AFTER ROGERS, Trans. Roy. Soc. Trop. Med. & Hyg., 1923, Vol. ~~XVI~~ P. 454: 2nd Map.)

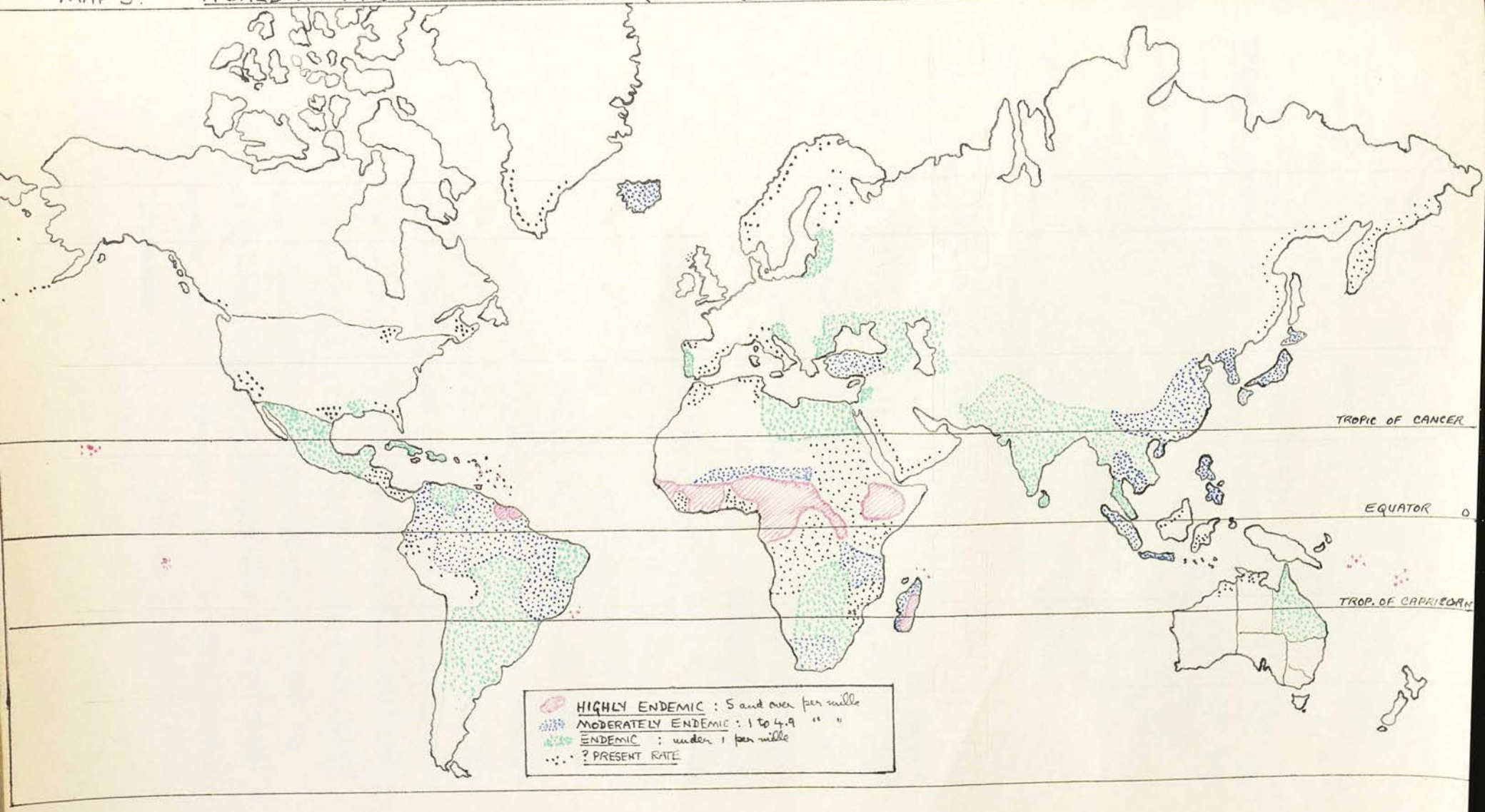


TABLE III. (cont.).

Place & date.	Leprosy rate per 100,000 pop.	Authors.
Calcutta, coolie labourers, 1930.	94.0.	E. MUIR (20)
Villupuram, S.Arcot 1929.	999.6.	I. SANTRA (21)
Hyderabad, Nizam's Dominions, 1933.	500 to 1,000.	J. LOWE. (22)

These figures may be paralleled by many throughout India. Madras Presidency has the highest number of lepers among the provinces of India (I. SANTRA) (21), and Ceylon must be included with India as a leper country, for COCHRANE, 1933, (23) estimates the number of lepers there as about 3,000.

WORLD DISTRIBUTION OF LEPROSY.

This is best considered by reference to Map 3. ROGERS, 1932, (24) gives the world distribution of leprosy in rates per mille. The highest rate per mille in the world is 200, that of a 150-miles' strip of the eastern border of the Belgian Congo. Other high rates are French Guiana (11.0) and Dutch Guiana (25), in South America: a large belt of tropical Africa, including French Guinea (5), French Ivory Coast (60.7), North Nigeria (5.2), the Kameruns (20.0) French Equatorial Africa (13.0) Abyssinia (20.0), Belgian Congo (200), E. Madagascar (14.0). In Oceana, there are New Caledonia (26.0), Loyalty Is. (36.0) Marquesas Is. (66.7) and Hawaii (11.8). In Europe there is little leprosy, though it is common in Iceland, and found in Spain, Italy, Balkans, Turkey, Crete, and Cyprus. A much lower

incidence is found in Norway, Sweden, Greece and some of the Mediterranean Islands. It is rare in France, Germany, and it is almost extinct in Denmark, Belgium, Holland, Austria-Hungary and the British Isles.

In Asia it is found practically in all parts except the more northerly.

In Australasia, it is found in Australia principally in the eastern state of Queensland, but there are some lepers also in New South Wales and Victoria. In New Zealand, it is known among the Maoris. The Pacific Islands are heavily infected, as Tahiti, Sandwich Islands, New Caledonia and Fiji.

Leprosy is spread sporadically over the United States, but is rare in Canada, Incidence is well-marked in Mexico and Central America and in the West Indies. In South America, it is common, Colombia, Venezuela, the Guineas and Brazil.

Africa appears to be very widely affected, and the greatest area of high incidence is in Africa.

As ROGERS points out, and as is shown by his map, the Tropics bear the heaviest and widest incidence of leprosy, especially the wetter tropical countries, whereas the dry tropical and the temperate zones have a much lower incidence. As far as India is concerned, he states that "the high rate in the Bombay Deccan and Berar is the only important departure from the general relationship between rainfall and leprosy." (25)

NUMBER OF LEPERS IN THE WORLD.

The estimate of ROGERS, 1924, [26] is of considerable interest, for after quoting HEISER'S figure of 2,000,000, he advances 3,000,000 as a more correct figure, giving the following recorded data:-

> In Europe 7,000, with about 1,000 each in South Russia, Baltic Provinces and Crete, and about 500 each in Turkey, Rumania, Spain and Portugal.
> In Asia 1,250,000, including 500,000 to 1,000,000 in China with its dense population, 102,000 in both Japan and India, 15,000 in both Indo-China and Siam, 5,000 in both the Philippines and in the E. Indies, and smaller numbers in Malaya, Ceylon, Palestine, etc.
> In Africa 500,000, nearly all in tropical Central Africa, with Egypt 6,500, Madagascar 4,000, South Africa 2,500. In the western hemisphere 30,000, including Brazil 15,000, Guiana 3,000. Cuba 1,500, the British West Indies 1,000, Colombia 6,500, Venezuela 750, etc., while Oceana has about 5,000.

Considering that the recorded figures total 1,792,000, and relate only to typical advanced cases, ROGERS thinks that 3,000,000 is far from being an improbable figure for the whole world, constituting leprosy a problem of unsurpassed difficulty and importance. The estimate for the British Empire is 156,000 of which two thirds are in India, if one takes only the recorded cases, but the probability of all cases is at least 1,000,000. As knowledge of leprosy has become spread, so has the recognition of the disease in the earlier stages, and the leprosy incidence figures tend to rise in every country where there is a forward movement in leprosy matters. Accordingly, the greater figures do not necessarily represent a true increase of the disease. They rather indicate that the world is

awake to the leprosy problem at last. Much of this alertness is due to the modern methods of segregation and treatment, and their more hopeful results than at any time before: amd as a symptom of this alertness may be mentioned the formation in 1931, of the INTERNATIONAL LEPROSY ASSOCIATION,[27] to co-ordinate and assist anti-leprosy efforts by persons of all nationalities.

HISTORICAL.

The Rig Veda, one of the ancient sacred writing of Aryan India, of date about B.C. 1400, describes a condition akin to leprosy, and this has caused some to consider India as the original home of leprosy.[28] On the other hand, the "Papyrus Ebers," an Egyptian medical treatise of date 1300 B.C., has also a description of a disease similar to leprosy, and so by many Egypt is regarded as the primary focus. The other great ancient literature which refers to leprosy is the book of Leviticus, in the Old Testament. It is true that the leprosy described in the thirteenth chapter under the Hebrew name 'zaarath' is suspected of being psoriasis (GARRISON),[29] and it is very likely that the ancients confused leprosy with may other diseases having ulceration and nodules, but other evidence in the Scriptures indicates that true leprosy was known and was prevalent among the Hebrews. In spite of the confusion of diseases, and the association of leprosy with gonorrhoea and leucorrhoea in the thirteenth to fifteenth chapters of Leviticus, the directions laid down are very remarkable. Rules in regard to segregation, the directions as to scraping the walls of the infected house, or even destroying it altogether, also incineration of garments and other fomites, are suggestive of an acute perception of some contagious disease. These precepts were taken over by posterity as of value against leprosy, and in the Middle Ages

were still in force.[30] The following is an outline of the historical course of leprosy in the world, (ROGERS, 1922).[31]

Leprosy was present in India and Egypt, about 1400 to 1350 B.C.

in China	about 200 B.C.
in Greece, (Aristotle)	345 B.C.
in Germany (Galen)	180 A.D.

Leprosy spread over Europe in the eighth to the tenth century, and became a scourge in Europe in the twelfth to the thirteenth centuries. It declined in the fourteenth, due to measures of segregation and social improvement, and remains endemic to-day in outlying parts of Europe where segregation is either not much used, or has only comparatively lately been carried out. Such countries are Norway, Spain, Portugal, Greece, Turkey and Russia.

The spread of leprosy to the West Indies, the northern parts of South America, and Mexico, took place at the end of the fourteenth century, by means of the invading Portuguese. Later, leprosy was brought by negro slaves from Africa, and Chinese and Indian immigrants.

The disastrous spread to Oceana in epidemic form occurred in the second half of the nineteenth century, to the Sandwich Islands in 1853, to New Caledonia in 1865, by Chinese immigrant infection and to the Marquesas in 1878.

It is believed that leprosy was introduced into Europe from Egypt, in the first ventury B.C., by the returning legions of the Roman Commander Pompey.[32] It is

possible that the introduction may have taken place earlier, by the invasion of Europe through Greece about 350 B.C., by the armies of Darius, the Persian.[33] As a result of the Crusades, the disease was spread widely over Europe by the returning Crusaders, and in the fourteenth century leprosy became so prevalent that in Europe there were approximately 20,000 leper asylums necessary, and 2,000 in France alone. As a result of strict methods of dealing with lepers, the disease began to decrease in the fourteenth century and by the fifteenth had practically left Europe as a whole. In England and Scotland there were some 220 of these 'leprosoria,' or 'leprodochia,' as they were called.[34] The building of these leprosoria was a great social and hygienic movement to cope with leprosy, and though in them not much attention was paid to treatment, and by them only nursing and segregation were afforded the inmates, there can be no doubt that they were a potent factor in the eventual stamping out of the disease. Leper hospitals had existed at an earlier date, as they are mentioned by Gregory of Tours, about 560 A.D., but their number was rapidly added to after the thirteenth century, when their advantages for the purpose of segregation became recognised. The more primitive and alternative method was to have the leper wandering abroad as a human outcaste, after a medical inspection called on the Continent 'Lepraschau,' condemning him to a civil death; forcing him to live in huts

in the open field, and to give warning of his approach by horn or bell.[34]

The whole matter of medieval leprosy is of great modern interest, because of the apparently victorious outcome of the medieval battle against leprosy. GARRISON[35] states that leprosy in France, for example, had so completely died out by the end of the sixteenth century, that Louis XIV. was able to abolish the leper houses and to devote their endowments to charity and to general hospital construction. Whether the segregation of lepers in these asylums was the sole reason for the medieval success in eradicating the disease is a different matter. GARRISON[36] describes the incidence of epidemic disease in the Middle Ages as being extremely heavy, and mentions as the real predisposing causes the crowded state and grossly bad sanitation of the walled towns, the squalor, misrule and immorality occasioned by the many wars, and by the fact that Europe was overrun by wandering soldiers, students and other vagabonds, while the general superstition, ignorance and uncleanliness of the masses has probably not been exceeded before or since. The gradual improvement in these evil conditions as the Middle Ages wore out, may have been just as potent a factor as segregation in stamping out leprosy. One social custom which was abolished about 1520, is an example of an improvement which would be of great value in lessening the incidence of leprosy. This was the medieval custom of

public baths,[37] many of which were frequented by men and women alike, all of whom sat and bathed together in one huge common vat or tank. It was only with the advent of leprosy, plague and syphilis, that this custom was abolished by special laws. On the other hand, ROGERS[38] partly ascribes the remarkable decline of leprosy in the latter part of the fourteenth century to the Black Death of 1349, which supposed to have carried off nearly half the population of Europe, and he compares this with the reductions of leprosy in Iceland and in India in recent times, due to epidemic diseases and famines falling most heavily on uncared-for lepers.

LEPROSY IN SCOTLAND.

According to P. RATTRAY,[39] the death of King Robert the Bruce, from leprosy, is suggested by an entry in the Chronicon de Lanercost, and if he was a leper, infection from Ireland is suggested, and his hard and strenuous life as a predisposing factor. In feudal Scotland, about 1200 A.D., great was the poverty of the diet of the people. There was a marked scarcity of fresh vegetables, and if meat and fish were eaten cured, as they often were, they were insufficiently cured, owing to the high price of salt. It is interesting to note how these conditions resemble present-day Indian ones. Scotland and India are comparable in that endemic leprosy has been found among a people among whom poverty was wide-spread and the common

diet very defective. Even in sparsely populated Scotland leprosy found a ready home, and though it was more common amongst the 'villeins,' the king apparently was not to escape. The earliest mention of leper houses was in 1100, there being 17 of them. The Highlands contained none, and this fact is taken as evidence that the Highlands escaped leprosy, which is not surprising considering the long-continued difficulties of communications with those regions. RATTRAY indicates that the Old Laws of Scotland reveal that strict segregation as understood now-a-days never entered the minds of those desiring to separate the lepers from the healthy: for example, lepers were allowed to enter towns on Mondays, Wednesdays and Fridays, from 10 to 2 o'clock. Leprosy lingered on in the Shetlands to the eighteenth century, but died out in the rest of the country at the same time as in the remainder of Europe.

LEPROSY IN NORWAY.

This country is of especial interest and importance, in that after the great diminution of leprosy, in common with other parts of Europe, in the fifteenth and sixteenth centuries, the disease did not die out altogether, probably owing to ineffective segregation, and further there was an alarming recrudescence of the disease about 1856, (H. P. LIE) [40] At this time, there must have been at least 2,858 lepers in a population of 1,500,000. The story of its rise and fall up to modern times is instructive.

Leprosy is first mentioned in Norway about 1,000 A.D., and was probsbly introduced by the Norwegian Vikings, who had brought it back from their voyages to England, Scotland and Ireland. As may be expected from such an origin, the main seat of the disease has always been along the coastal districts in southwest Norway, though it spread over the whole country. In the thirteenth century, leprosy reached its height, and hospitals were built. The most important were two hospitals in Bergen: of these, one later fell into oblivion in history, and the other was turned into a general hospital in 1545, though it still retained accommodation for a few lepers. The disease was from about that time thought to be conquered, but instead, leprosy very slowly increased in the seventeenth and eighteenth centuries, with a rapid increase in the nineteenth. From 1839 onwards the subject of leprosy aroused great attention, and in 1847, DANIELSON and BOECK published their famous work "Om Spedalskhed." This work was a secure basis for a clinical, administrative and legislative campaign. New leper hospitals were built, and stricter isolation, though not always necessarily segregation, was obtained. Thus if a leper did not wish to go to hospital, he was allowed not to do so, but if he did not obey instructions given him about isolation, he could be removed to a leper hospital by the police. Likewise, it was made a punishable offence to neglect or

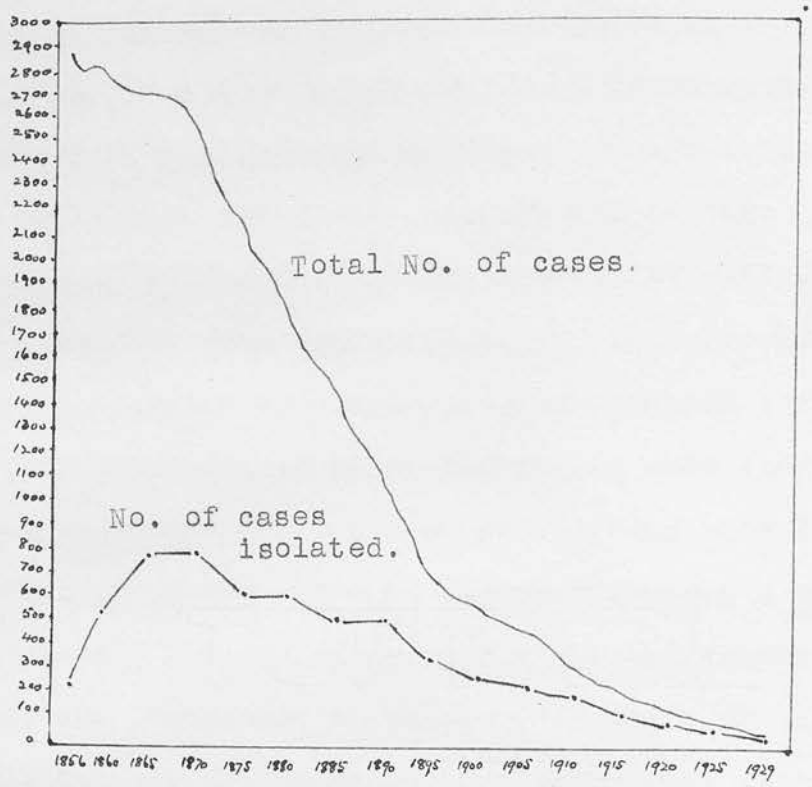


FIGURE 3. The Course of Leprosy in Norway, from 1856 to 1929.

(After H.P.LIE, "Leprosy Review," Vol. 1, No. 2, April, 1930: p.14.)

obstruct disinfection of the house and fomites of a leper. By such measures of control, a very satisfactory reduction of the incidence of leprosy since 1856 has been obtained in Norway, as the graph given by H. P. LIE shows.[41] This graph is reproduced in Figure 3. As the position now is, there is good hope that Norway is within measurable distance of the disappearance of leprosy, and the lesson for other countries is that the Norwegian workers insist on strict isolation and control of all lepers if such a result is to be obtained and maintained. BURNET,[42] who visited Norway, reported, "Norway is the classic example of the progressive eradication of leprosy through the systematic isolation of lepers."

THE BACKGROUND OF GENERAL DISEASE.

In order to obtain a picture of the background of general diseases which the people of Poona District present as regards leprosy, an analysis was made of 5,000. cases treated in the N.M. Wadia Hospital, or met with in its clinics. The purely surgical cases were put aside, that is, cases of the type of burns, fractures, dislocations and operative procedures for conditions in which no obvious root disease could be discriminated. If, however, in a surgical case the cause was clearly revealed as one of the root diseases, it was classified as such, and not rejected as a case for inclusion; for example, a case operated on for acute intestinal obstruction in which the obstruction

was found to be masses of Ascaris lumbricoides, would be included as a helminthic disease, or an operation for liver abscess would be included as dysentery if its amoebic causation had been established. At the same time, this method has not been pushed to its limits, as, for example, would be done if cases of vesical calculus were put among the deficiency diseases, on the ground that there were some who consider them as such.

As the hospital is a general one, all castes and races are met with, including Europeans, and country people as well as city. For this reason, a consideration of its cases gives a very fair idea of type of disease which is common in the District. Any hospital in India, where clinical material is enormous, tends to have a selective incidence of certain diseases according to the demands of local circumstances, or the special provision made to deal with them. This fact should be borne in mind when considering the great incidence of tuberculosis and of veneral diseases in the following table, for there exist extremely few facilities for the treatment of these diseases in the District, and as the hospital has made an attempt to cope with the problem, it tends to get far more of such diseases than it should, if public health provisions were more adequate.

Of the 5,000 cases taken, separating first 1,057 cases of minor interest for our purpose, which include medical cases of general type, the remainder may be grouped as follows:-

(In Figure 4. the occurrence of some of the diseases is represented graphically).

TABLE IV.

453.cases, or 90.6% belong to the MALARIA GROUP.
597.cases, or 11.94%. belong to the TUBERCULOSIS GROUP.
348.cases, or 6.96%. belong to the DYSENTERY GROUP.
552.cases, or 11.04%. belong to the VENERAL DISEASES GROUP.
24.cases,or 0.48%. of TETANUS.
13.cases or 0.26%. of LEISHMANIASIS.
12.cases,or 0.24%. of DENGUE or PHLEBOTOMUS FEVERS.
6.cases,or 0.12%. of RAT-BITE FEVER.
57.cases,or 1.14%. of PLAGUE.
101.cases,or 2.2%. of ENTERIC.
150.cases,or 3.0%. of DRACONTIASIS.
200.cases,or 4.0%. of BERIBERI.
24.cases,or 0.48%. of PELLAGRA.
26.cases,or 0.52%. of SCURVY.
12.cases,or 0.24%. of CHOLERA.
25.cases,or 0.5%. of SPRUE.
51.cases,or 1.2%. of INFANTILE BILIARY CIRRHOSIS.
2.cases,or 0.04%. of ULCERATING GRANULOMA OF THE PUDENDA.
49.cases,or 0.98%. of FILARIASIS.
51.cases,or 1.2%. of VARIOUS POISONINGS.
250.cases,or 5.0%. of HELMINTHIASIS.
11.cases,or 0.22%. of SNAKE BITE.
249.cases,or 4.98%. of MYIASIS.
211.cases,or 4.22%. of SKIN DISEASES.
46.cases,or 0.92%. of ULCUS TROPICUM.
29.cases,or 0.58%. of MYCETOMA.
10.cases,or 0.2%. of CLIMATIC BUBO.
13.cases,or 0.26%. of RHINOSPORIDIOSIS.
150.cases,or 3.0%. of EYE DISEASES.
48.cases,or 0.96%. of DIABETES.
152.cases,or 3.04%. of CIRRHOSIS OF THE LIVER with ASCITES.

The prominence of tuberculosis is partially explained by the selective factor exerted by the hospital, as previously mentioned, but there remains no doubt that it a very common disease, and perhaps the commonest. Pulmonary, glandular, abdominal, cutaneous, bone and joint tuberculosis occur just as in Europe, and they occur in a people who have less resistance. In passing, it may be mentioned

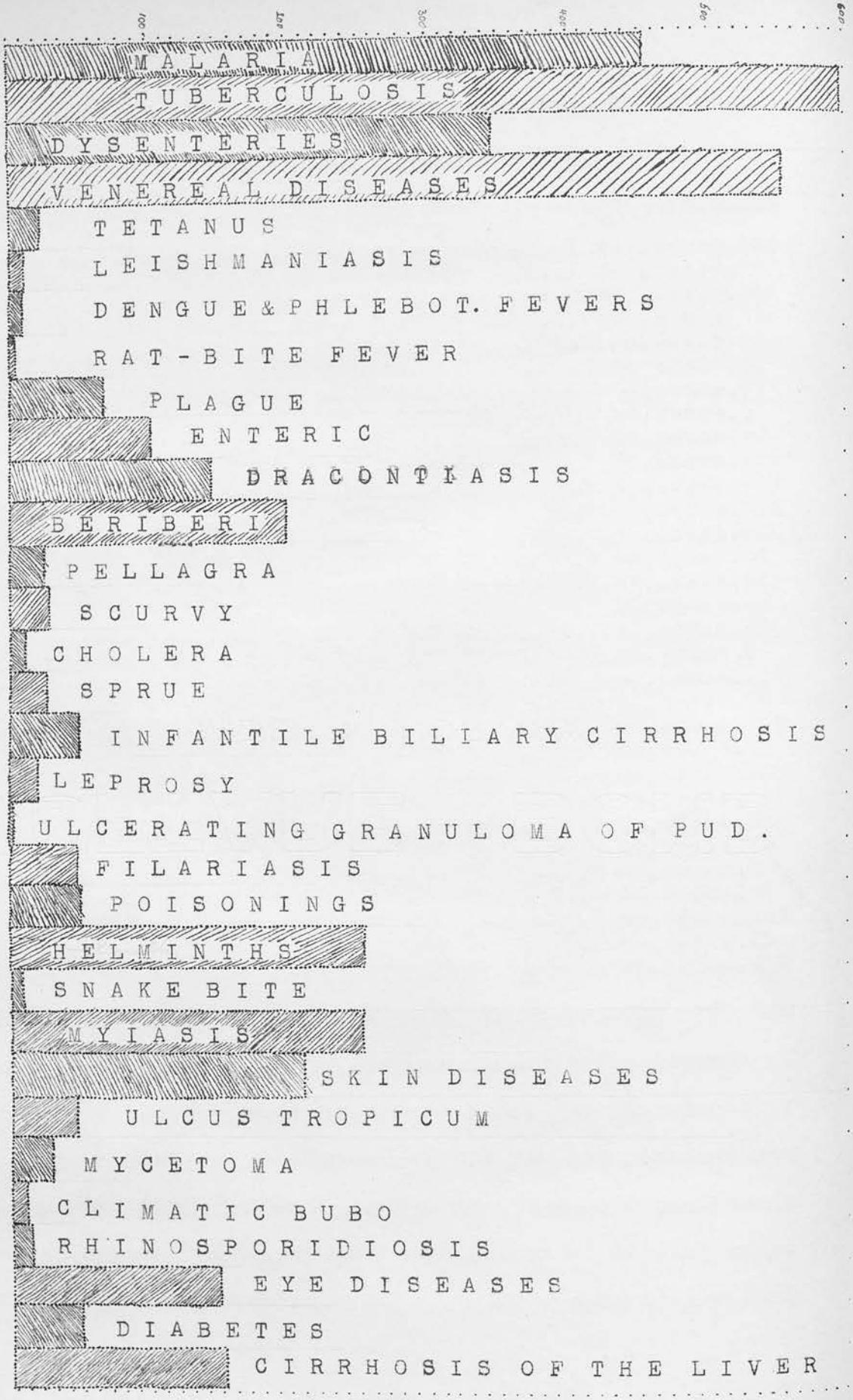


Figure 4 The comparative incidence of certain diseases in a series of 5,000 cases met with at Poona.

that ROGERS in his Croonian Lectures (43) draws attention to an inverse ratio between leprosy prevalence and tuberculisation, so that there is a low degree of tuberculisation of the people in just those tropical areas where exceptionally high leprosy rates per mille have been recorded: intermediate leprosy rates are seen in such long tubercular infected countries as India and China. He describes the tuberculisation rate in India as 'fairly high.'

The veneral diseases column in Figure 4. will be noticed to be the next in height. This rate also is partly selective, for a special clinic is conducted for veneral diseases, and attracts the greatest proportion of these cases in Poona. Here again, however, these diseases appear to be far more common than in Europe. In the year 1931, a serological test for syphilis, the Kahn Test, was carries out on all the inmates of the hospital on two separate occasions, giving a total of 180 cases. The percentage of Kahn Tests that were positive 'two plus' and above was 15. Gonorrhoea is almost as common as syphilis, but as it is more often treated by country medicine, it does not appear more than as one quarter of the syphilis total in our cases. Chancroid is not so commonly seen as the two other main veneral diseases. There are undoubtedly a vast number of cases of untreated congenital and acquired syphilis in the population.

Malaria comes next in the list, and here there is

little doubt that we are dealing with an extremely common disease. The cases seen were chiefly benign tertian, and malarial cachexia, though the District is liable to localised epidemics of virulent malignant tertian. Blackwater fever is rare, though our series contained one case, in a European, who had lived in the malignant tertian District of Nasrapur for over 20 years. There are well-known malarious districts and areas where almost every patient who comes under view, for whatever purpose, has a grossly enlarged spleen. Such an area is that of Yerandavna, in Poona itself, less than two miles from the hospital. Another is the Kharakwasla Lake area, about 11 miles from Poona. Malaria, then, may be described as more than moderately endemic.

The dysenteries are what makes Poona notorious. The idea that they are a perquisite of the European, and do not afflict the native much, is mistaken. It is true that the European is pecularly liable, but there is a vast reservoir of dysentery in the native population. The latter may have some sort of a natural resistance, with the result that there is a greater amount of sub-clinical dysentery than amongst Europeans, but in an Indian hospital one can see cases every whit as acute and as serious as among the foreigners. Bacillary dysentery predominates, and is in Poona chiefly caused by Bacillus Dysenteriae, Flexner, (and Sonne occasionally), in the milder types and Shiga in the

epidemic or severe cases, The great prevalence of intestinal troubles in Poona, particularly 'Poona-itis,' or 'Poona tummy,' has been shown by MANIFOLD and de MONTE, 1928, to be due to infection with the Flexner bacillus.[44] (Manifold J.A. & de Monte A.J., 1928. Report on the investigation of dysentery and diarrhoea in Poona. Indian Journ. Med. Res. Vol. XV. No.3. P. 60.) Amoebic dysentery and amoebic liver abscess are not so common as bacillary cases in hospital practice, but are numerous enough. In our series of 348 cases,

TABLE V.

B.dysenteriae was found in 168, or 48.2%.

Exudate typical of bacillary dysentery, but B. dysenteriae not isolated, in 90 cases, or 25.8%.

Entamoeba histolytica found in 40 cases, or 11.4%.

Amoebic liver abscess, 32 cases, or 9.1%.

Indefinite exudate, in 18 cases, or 5.1%.

In this series, bacillary dysentery accounted for 74%. and amoebic for 20.5%.

Helminths play a large part in the general morbidity. To place them in order of importance, ascariasis is first, then ancylostomiasis, taeniasis (usually Taenia saginata, but also Taenia solium , while hydatid disease of lung, liver and peritoneum is by no means uncommon), Enterobius vermicularis, and a very few of Trichiuris trichiura, Trichinella spiralis, and Hymenolepis nana.

Dracontiasis is of much importance, and is separated

here from filariasis, which is not more than moderately endemic. Guinea worm is widespread and troublesome and is one of the commonest sights at certain seasons in all outpatient dispensaries. Massive incidence is common in villages. Just as whole villages may be found the victims of hookworm or round worm, so one has visited villages where every inhabitant suffered or had suffered from guinea worm. "Worms" of all sorts bulk largely in the life of the people. As regards filariasis, in the 49 cases, a few examples each of lymph scrotum, varicose glands, chyluria (2), chylocele, chylous ascites (1), elephantiasis, orchitis, funiculitis and synovitis, occur.

Myiasis calls for remark. It is a striking and nauseating feature of great frequency. Perhaps more than anything this fact gives an indication of the standard of living among the people, for dirt and carelessness in personal hygiene are the prime factors in high incidence. Nasopharyngeal, ocular, aural, vaginal and wound myiasis, due mostly to Chrysomyia bezziana, and intestinal myiasis due to Musca vicina, make up the series.

In the skin diseases,scabies, and all sorts of ringworm, are ubiquitous, and lead the lists. A streptococcal impetigo which resembles scabies is the great dispensary skin trouble, but true scabies is also much seen. Eye diseases reveal also a low standard of living, with the neglected trachomatous lesions, glaucoma, corneal ulcers,

conjunctivitis, keratomalacia, and the like which show plainly how little care is taken of the eyes. Dietary errors and deficiency diseases are shown to be frequent, as the columns in the diagram depict, those which refer to beriberi, pellagra, scurvy and diabetes, and perhaps infantile biliary cirrhosis may be included under this head. Rickets is not shown, nor osteomalacia, but they also are common in Poona.

Finally, as it is of social importance, attention is drawn to the 51 cases of various types of poisonings. Alcoholism is a habit of many of the lower castes, and calls for medical care in many cases, either as acute alcoholic or chronic alcoholic poisoning. Opium is a very widely used drug, for babies, and as a habit in adults, and Ganja or Cannibis indica is smoked. Then there were one or two cases of poisoning in which ground glass, datura, or white oleander was suspected. To treat an overdose of opium in a baby is quite a usual emergency and babies suffering from chronic opium poisoning are all too often seen.

The incidence of 152 cases of cirrhosis of the liver refers to a group of diseases difficult to classify according to causes, as alcohol, syphilis and unknown toxins may play a part. It is likely also that some of the cases with ascites may not be cirrhosis of the liver at all, but the Post-dysenteric ascites described by MANSON-BAHR (45) quoting MEGAW. (Manson-Bahr -"Manson's Tropical Diseases,

1929." P.388. - Cassell & Co., London.)

GENERAL CONDITIONS OF LIFE.

The unit of any District in India is the village, and in Poona District the land is fairly thickly dotted with them. To the traveller by motor the tawny brown hills seem to contain hardly any villages, because they are uniform in colour with the landscape, and are very irregularly placed. Only to the patient traveller by foot or by pony, or bullock cart, will most of these villages be accessible. If the maps published under the direction of the Surveyor-General of India, 1928, Nos.47 F/N.E. and 47 F/S.E. be consulted, (COL. E. A. TANDY, R.E.)[46], a good idea of the number of villages will be given. Thus, in a five minute square, about 16-20 villages will be counted, and in almost every one minute square, 2 villages as the maximum and one village at least. A square of one minute represents roughly one of 1900 yards.

As an introduction to the subject of local conditions, the words of the famous ABBE J. A. DUBOIS[47] may be quoted:-

> "The climate, which is the chief cause of the degeneration of the human race in these countries, exercises a no less fatal influence in the animal and vegetable kingdoms. Green stuff, roots and fruits are for the most part insipid and tasteless, and do not possess half the nutritive value of those grown in Europe. A very few may be cited as exceptions to this rule. The vegetable products of India are

"pungent enough to destroy the membrane of one's throat. Again, the indigenous flowers, with two or three exceptions, have no scent. Lastly, the trees and shrubs to be found in forests or in uncultivated places are generally covered with thorns and prickles. The elephant and tiger are strong and vigourous enough, but all other animals, whether wild or domesticated, share in the universal debilitation. What we call butcher's meat has very little succulence in it, and there is nothing in the flavour of game that would tempt the least fastidious European palate. Vainly would one search for a good hare or partridge. One is inclined to think that nature here has reduced the nutritive value of all animals and vegetables in proportion to the weaknes of human beings whose food they are to be."

VILLAGE LIFE IN POONA DISTRICT.

As the village is the important unit in social life, and the opportunities for visiting different villages were frequent, the author has pursued many enquiries into life in them. The people have been at all times friendly and informative, and as their own vernacular, namely Marathi, was used always in intercourse with them, the truth was perhaps more available than if 'the language of the Saheb' (Hindustani) had been used. One must at all times take into account the universal tendency for the

Indian to answer as he thinks the questioner will like, and not as the truth is, but a good knowledge of the vernacular and of the people can be used to correct this. The valuable Study of MANN and KANITKAR[48] of the village of Jategaon Budruk, some 20 miles from Poona, has been used as a guide and a check in obtaining information, and in the ensuing account, indebtedness to their work is gratefully acknowledged. The description may be conveniently given in the form of question and answer.

WHO LIVE IN A POONA VILLAGE?

The agricultural caste of Marathas or Kunbis are the strongest element in all village pooulations, and each village is comprised of a social entity organised by means of the caste system. Each caste has its position and its duty to the community, and all the wants of the villagers are attempted to be provided for. In Jategaon, the families were distributed as follows:-

TABLE VI.

Caste.	Number of families.	Total population.
Marathas or Kunbis	113.	553.
Brahmins	2.	6.
Marawadi (moneylender)	1.	10.
Gurav (priest)	1.	6.
Lohars (smiths)	3.	26.
Mahomedans	3.	20.
Nhavi (barber)	4.	20.
Ramoshi (watchman)	3.	8.
Chambar (shoemaker)	3.	18.
Mahars ('untouchables')	14.	69.
	147.	736.

WHAT SORT OF DWELLINGS?

The village is invariably a jumble of decrepit-looking huts of stone, roofed with curved country tiles. There are enclosures for cattle in the same compound walls as the human dwellings, and the rooms are small, low and dark, with an uncovered well as water supply. Granaries are in the house itself, and rats are numerous. Some attempt at an airy part of the house is represented by the 'ota' or veranda, on which the members may sleep on their flimsy bedsteads at night, in the very hot weather. The house is always far too small for its population, and overcrowding is the natural condition of life. There are several public places which are used as sleeping places by the males, such as the village temples, or a 'dharamshala' or rest house. The huts of the low castes and the outcastes are wretched in the extreme, as they are little more than fragile erections of any sort of old material, as sacking, tins and mud, which can be obtained. The outcastes' huts are at some distance apart from the village, but never very far, as they are useful servants to the village community in the matter of disposal of excrement, and of carrion. The disposal of excrement is very unsatisfactory, as it may be thrown into streams or pits or waste ground. The better castes may have some attempt at latrines in or near their houses, or there may be a village latrine, but there is a considerable proportion of the people which con-

siders the whole world as its latrine. The breeding of Muscines is always enormous in the villages.

WHAT ARE THE SOCIAL CUSTOMS OF IMPORTANCE?

The caste system in its ancient rigidity must have served as a means of maintaining a check on the spread of disease, for it restricted intercourse between the higher and the lower members of the community. Many villages preserve the ancient restrictions, but in others the rules of caste are not so well observed, under the pressure of modern influences. One of these is the coming of the motor buses, which now travel crowded and with frequency between most of the villages. In them, folk of all castes may be jammed together, caste forgotten in the necessity of getting and keeping a place in the bus. Another, is the propaganda of Mr. Gandhi for the uplift of the outcaste, with the result that they now demand and sometimes obtain in the villages unheard of privileges, in the way of schools, being allowed to attend public meetings, use public wells, and the like. It must not be imagined that this change is widespread, but it is slowly occurring. From the strictly medical point of view, the break up of caste is not an unmixed blessing, for the Untouchables are an undoubted reservoir of disease. In them, syphilis is most common, and the incidence of leprosy is heaviest. It is remarkable that the Brahmins have such a small incidence of leprosy compared with other castes, and this is probably to be related to their aloofness in social life, and their

greater cleanliness and hygiene. Caste, then, is the first most striking feature of village life, and so far as it is observed with strictness, is a means of restricting the spread of a disease like leprosy.

The joint family system is the rule, and is believed to be one of the principal agencies in the spread of leprosy. (LOWE).[49] Under this system several related families live in one house. One finds commonly a father and mother with several married sons and their families living under one roof. When a son marries he brings his wife to his father's house, and there brings up his family. When a daughter marries she goes to the house of her husband's father. Village houses are frequently crowded with members of three generations of the same family. In the case of a leper in a joint family house, unless there is marked nodulation or trophic lesions, there is usually no attempt at segregation. When marked lesions appear, the leper is usually provided with separate eating and drinking utensils, but contact with healthy members of the joint family continues, and it is only in the case of a few advanced lepers that separate accommodation is occasionally arranged.

A custom of some value is that if leprosy appears in a member of a family before the age of marriage, marriage cannot take place as no one will knowingly marry his son or daughter to a leper. Not only the leper, but other

members of the family, may find marriage difficult or impossible, on the mere suspicion of leprosy perhaps. If leprosy appears in the wife after the marriage but before there are children, a divorce is usually obtained, and the wife returns to her father's house. If the husband develops leprosy, the wife may or may not be able to meet the cost of divorce, and she may run away to her family home. If there are children to the marriage before leprosy appears in one of the parents, the family ties often keep them together. On the whole, little activity is displayed in the face of the development of leprosy in a member of the family. Segregation is all too rare, and when employed is too late. The author has come across many families where by a sort of tacit consent the presence of leprosy in a senior member of the family has been ignored, whether from politeness, inertia, or economic necessity. Knowledge of the contagiousness of leprosy is widespread, but only the advanced cases are considered contagious, and the earlier infectious cases are left to live their life in contact with all, including the very susceptible children.

The custom of child marriage has long been admitted to be a debilitating factor in social life. Almost at puberty the Indian girl may begin childbearing, and with this is bound up the question of widow remarriage. If the husband dies, in the higher castes the widow is forbidden to remarry, and may drift into a life of prostitut-

ism. There is an appalling infant mortality. In a population of 720 in one village, of the deaths recorded 45% were of children below the age of three years. Childbirth is conducted by the village 'Dai,' an ignorant insanitary creature who believes in force as the main means of obstetric delivery. Frequent are the cases of ruptured uterus, inversion, peritonitis, and uterine inertia which come to hospital after her forcible ministrations. Medical help is scanty or non-existent in many villages, and the morbidity is heavy in the extreme.

As mentioned, prostitution is given material by the rule against widow remarriage. Also the women of the 'untouchables' may be prostitutes for all castes, even Brahmins, hence the saying 'Beware a black Brahmin and a fair untouchable.' Sexual promiscuousness is to be regarded as normal in the social life and matters are not improved in recent times by the migration and return of labourers to and from Bombay factories, as many a simple villager brings back bad habits from Bombay. The religious festivals are not above reproach, and one popular festival in particular, called Holi, has a bad reputation in this respect. Most of the Maratha villagers are worshippers of Shiva and his 'lingam.'

The custom of 'Yatra' or pilgrimage is deeply rooted among the village people. This is of great importance from the point of view of leprosy. The two chief pilgrimages annually which appeal to the villager in Poona

District are the Alandi and the Pandharpur. It is well known the difficulties with cholera and dysentery and other epidemic diseases these pilgrimages occasion, and after every such pilgrimage one is accustomed in Poona to have a rush of patients as the pilgrims pass through, especially on return. Leprosy also seizes its grim opportunity, and, owing to the fact that priests at the shrines are often lepers, and that many lepers journey to the feet of the god in the hope of getting better of their disease, and stay there if they do not, the famous shrines of the gods are notorious foci of leprosy. Spread of leprosy is only to be expected under such circumstances. The author visited Alandi at the height of the festival, and personally counted 23 lepers in one hour's watching at one spot. The social and religious necessity of men not shaving their own beards, and depending on the services of the village 'nhavi,' or barber, is worthy of note. The barber will cheerfully shave a leper and then a healthy man, or he may himself be a leper in an infectious stage. Such an eventuality has been observed in the villages. Allied to the village barber in potentialities for spreading leprosy is the village 'gawali' or milkman, who is not averse to milking the cow or buffalo for healthy persons, when he himself is an infectious leper. This also has been observed.

The custom of toddy drinking is confined to the Kunbis and lower castes. There is no question that the average

Kunbi will drink toddy when he can get it, and drunkenness is fairly frequent, but poverty is a check on over-indulgence. A 'seer' (roughly a quart) of toddy costs eight annas (about ninepence), which is a large sum for a man who may earn only seven rupees (about ten shillings) per month. Toddy is the fermented sap of the toddy palm, sold under Government licence. It is considered by the people of medicinal value as a diuretic, and useful in gonorrhoea, and perhaps it is a pity it is not given to children instead of the universal opium pellet, for G. W. BRAY[50] in Nauru has shown that partially fermented toddy is efficacious in preventing and curing infantile beriberi. Infantile beriberi is one of the diseases met with in the District. Opium smoking and eating and smoking of Indian hemp, are common among adults.

Connected with the economic position of the individual villager is the custom of being in debt. It is not too much to say that if the villager were not in debt he would feel lost. The moneylender in the village is usually the Marwadi shopkeeper, or he may be another foreigner such as a Pathan. A Marwadi in one village settled in the place with a capital sum of Rupees 500., and after 35 years, he became master of nearly 172 acres of land, and has money out on loan to the extent of over Rupees 50,000. It seems to be the universal practice in the villages to give a bond for twice the amount borrowed from the money-lender. Every cultivator who has

borrowed tells the same story, and on being questioned as to why he borrows the money under such onerous conditions, replies saying that there is no other source from which he can obtain money when he wants it. In cases where the original sum can be restored with interest, the money-lender takes it back without demur, but such cases are very few. The borrower may be ready to pay back a part of the sum after a year or two, and in this event the money-lender will not accept it, and demands the whole sum or nothing, while allowing payment to be further postponed. After a time a suit is filed according to the bond, and the borrower may be obliged to pay, with interest, what often comprises four times the original amount borrowed. The rates of interest vary from 12 to 72 per cent, though the extreme cases are not many.

WHAT IS THE ECONOMIC POSITION OF THE VILLAGER?

The average state is one of great poverty, according to our standards. In a village near Poona, consisting of 147 families, the total annual income was found to be Rupees 24,348. to which falls to be added income from village 'babul' trees and fruit trees of Rupees 615., giving a total available income for the village of Rupees 24,963. This works out at Rupees 167.13.0. per annum per family, and an annual income per head of Rupees 33.12. 0., or about two pounds ten shillings sterling. There were 740 people in the village.

The land is the real and permanent capital in the villages but it tends to slip out of the hands of the debt-laden villager into those of the moneylenders or bigger landowners, and the system of subdivision of smallholdings for the sons is widespread. The land itself is very close to being in an unimproved condition. There are no fences except temporary ones put up to protect special crops. Drains do not exist. Roads and tracks are nothing more. There are a certain number of embankments, a few wells, one or rwo water channels, the village houses, and the agricultural stock of buffaloes and draught cattle, but these form a smaller ratio to the unimproved value of the land than is the case in other parts of India. Drainage takes place with a good deal of erosion, and wastes soil, and the rainfall does not penetrate as it should. Outside the irrigated areas, the larger part of Poona District contains agricultural villages which suffer from a 'gamble in rain.' If a good season does come, in spite of everything, the people do fairly well, according to their own standards. Good seasons, however, only occur two or four times in ten years, or nine times out of the twenty-four. An average year leaves the village under-fed, more in debt than ever, and apparently less capable than ever of obtaining, with the heavy population and the present methods of cultivation, a real economic independence.

The average cost of food for the family has been worked out as follows:-

TABLE VII.

Material	Quantity needed per annum.	Value per annum.
Bajri or jowar grain @ 18 lbs. per rupee	1800 lbs.	100 rupees.
Pulses @ 20 lbs per rupee	600 lbs.	30 "
Rice and wheat @ 15 lbs per rupee	150 lbs.	10 "
Salt		4 "
Chillies, etc.		4 "
Oil		4 "
Gul (sugar)		3 "
Miscellaneous		5 "
		160 "

The total cost per family per month for food is, therefore, Rs.13. 5. 0., and it is calculated that the necessary food expenditure per annum for a man will be about Rs.42., for a woman Rs.33. and Rs.25. for each child. The price of grain dominates the cost of food in the villages, for 70 to 80%. of the annual expenditure on food goes on grain. Very little milk or butter is ever taken, as there are never enough milch cattle for more than one or two families.

As a result of the economic stress in the villages, there has been a migration of many labourers to Bombay and other large centres for work. Those who go do not altogether sever their connection with the village, as they are away for from four to eight months in the year, and return for the rest of the time. Only a few stay away

permanently. The advantage to the village in reducing the pressure on the land is great, but very little direct financial gain accrues to it. The amount of money sent back by post is small, and the workers who return from Bombay do not admit that they have benefitted permanently very much. It is significant that hardly any are able to buy land on their return.

WHAT ARE THE SOILS OF POONA DISTRICT?

This question is of some importance as indirectly affecting leprosy incidence in a village through its direct effect on the economic position of the people. The following facts are derived from D. L. SAHASRABUDDHE, 1929.(51)

There are three separate tracts of which the District is composed, the Mawal tract, the Transition tract, and the Desh. The Mawal tract is the hilly belt in the most western part of the District, which receives heavy rain in the months of June to September. Very little of the rain is retained, as most of it runs down the slopes carrying with it fine particles of soil. The soils on the hill slopes are poor and shallow and grow only inferior millets, and the soils at the foot of the slopes are good enough to grow rice. The Transition tract lies between the hilly western tract and the dry Desh tract on the east. Many of its soils are fertile, and the best garden soils of the District are situated in this tract. Vegetables, grapes, figs, oranges, plantains, are grown with success. The greater part of the tract has black or medium black

soil, also called black cotton soil. As this soil is very typical of Poona District, its analyses will be given:-

TABLE VIII.

Mechanical Analysis.

Clay and finest silt	4.8%.
Fine silt	61.2%.
Medium Silt	10.7%.
Coarse silt	8.4%.
Fine sand	8.1%.
Coarse sand and gravel.	6.7%.

Chemical Analysis.

Loss on ignition	19.2%.
Silica and insoluble silicates ..	63.8%.
Organic matter	2.38%.
Lime (CaO)	1.90%.
Potash (K_2O)	1.60%.
Phosphoric acid (P_2O_5)	0.23%.
Nitrogen	0.06%.

The high percentage of loss by ignition of the dried soil is very charactistic of black soil and is due to the presence of much combined water, and not to a large quantity of organic matter. The organic matter is usually very small. The deficiency in organic matter and nitrogen is the most strongly marked feature of this soil. It is rich otherwise.

The Desh tract has poorer soils of various types of black, red and yellow porous mixtures, and millets are grown. The value of soils in the District depends on their retentiveness and depth. The black soils are the best, because they retain water better than others. The other soils are inferior because they are porous and water

runs through them easily out of the range of the plant roots. The dry Desh tract is under a double disability, namely, low rainfall and porous soils. It is obviously not enough to correlate leprosy incidence with high rainfall. A soil which is of rain not retentive will not produce good crops, and leads to a low economic standard among the people. In addition to considering rainfall, it is necessary to consider the type of soil on which it falls.

The Transition area is on the whole the richest part of Poona District, as far as agricultural land is concerned.

WHAT ARE THE FOOD GRAINS OF POONA DISTRICT?

Since over 70% of the annual expenditure on food goes on grain, some account of the food grains of the District is called for. The following facts are derived from A. H. CHURCH, 1930.(52).

The large group of the minor cereals, which may be designated 'millets,' together constitute a more important crop than rice or wheat, and are grown more extensively. They occupy about 83%. of the food grain area in Bombay province, the chief species are:-

Jowar or Great Millet (Sorghum vulgare).
Bajri or spiked millet (Pennisetum typhoideum).
Ragi (Eleusine coracana).
Italian millet (Setaria italica).
Chena (Panicum miliaceum).

Next in importance to the millets come the group of the larger cereal grains. This includes wheat, rice, barley and maize. The area occupied by these grains is

always small. Thus in Jategaon the figures for a number of years are:-

TABLE IX.

Year.	Percentage of Grain Area occupied.							
1897.	Bajri	44.1.	Jowar	44.7.	Wheat	10.6.	Others	0.6.
1901.	"	76.5.	"	20.7.	"	2.5.	"	0.3.
1910.	"	61.0.	"	33.8.	"	5.2.	"	...
1914.	"	86.9.	"	7.6.	"	5.0.	"	0.5.
1917.	"	45.8.	"	49.3.	"	4.8.	"	0.3.

The small amount of wheat and rice grown and eaten sharply marks off Poona region from the Punjab or from Bengal. These grains are commonly eaten, but they must be imported, and the people tend to use millets as the basic dietry grains. While wheat approaches in composition that of a standard food, rice is very far from doing so, both in regard to albuminoids and mineral matters. Much more pulse must be introduced into a rice diet than into a millet or wheat diet, in order that the proper proportions of proteins and salts may be reached. Millet is by no means a perfect food grain, which should contain less than five parts of starch or its equivalent of oil present to one part of albuminoid. It approaches rice in containing more than ten parts of the former to one of the latter, though not so deficient as rice in potash, phosphoric acid, lime and other mineral matter.

TABLE X.

COMPOSITION OF A MILLET (Kodra Millet, husked).

	In 100 parts.
Water	11.7.
Albuminoids	7.0.
Starch	77.2.
Oil	2.1.
Fibre	0.7.
Ash	1.3.
Postash	0.2.
Phosphoric acid...	0.8.

COMPOSITION OF RICE.

	In 100 parts.
Water	12.8.
Albuminoids... ..	7.3.
Starch	78,3,
Oil	0.6.
Fibre.	0.4.
Ash	0.6.
Potash	0.065.
Phosphoric acid...	0.284.

COMPOSITION OF WHEAT (Average Indian).

	In 100 parts.
Water	12.5.
Albuminoids	13.5.
Starch	68.4.
Oil	1.2.
Fibre	2.7.
Ash	1.7.
Potash	0.51.
Phospohoic acid...	0.76.

The superiority of wheat is evident, but unfortunately it enters little into the general diet. When wheat is sold in Poona District, it is moreover often foreign, and inferior to Indian wheat. No sample analysed of Indian wheat has ever contained less than 10% of albuminoids, whereas a large number of samples of first rate English, Canadian and Australian wheats analysed by A. H. CHURCH gave results

between 8 and 9. Rice is in a similar position. In addition to being an inferior food itself, it is often imported for use inferior to the local product. As supplementary to the millets, the various pulses are more important than wheat and rice. The seeds of the leguminous plants, generally known as pulses, are in common use. Pulses differ from the cereal grains in several particulars. They sometimes contain rather more oil or fat, a constituent which may rise to 17%. (Inga beans), to 18%. (in Soya beans), and to 50%. (in pea nuts). They rarely yield less than 2%. and often as much as 4%. of mineral matter or ash. More important than oil or mineral matter is the nitrogenous matter of pulses. This is often called legumin, or vegetable casein, but in reality it varies in different kinds of pulse, and is a mixture, not a single definite compound. Unfortunately, the digestibility of the albuminoids in pulse as compared with those in the cereal grains is regarded as low. In general, they are not only digested and absorbed at a slower rate, but a larger proportion of the total amount remains unused in its passage through the alimentary tract. The proportion of unused to used albuminoids is highest when pulses form the largest part of the diet; it is much reduced when the pulse eaten is not more than one quarter of the daily food; amd it is still further lowered when the pulse is eaten with milk, butter and eggs, or other easily

digested animal foods. Even under the best circumstances the unused portion amounts to 8%. of the total. Of starch in pulse, from 93 to 96% may be taken up, but the fat or oil is less available, except in the case of soya beans and pea-nuts.

Of the pulses, there is one which is regarded as of value in leprosy. This is the chick-pea or common gram, the young leaves of which fried in oil or ghi are sometimes ordered as the exclusive diet in leprosy by 'vaidyas' or Indian physicians. The question of diet values in relation to the disease will be considered in a later section.

In concluding this description of general conditions of life in Poona District, it may be stated that Poona City, suburbs and population form what is really a large village. Most of what applies to the village in the country can be taken as descriptive of Poona itself. Unlike cosompolitan Bombay, Poona is but an enlarged edition of the unit village. Differences from the 'mofussil' village are in degree rather than in kind. Thus a village makes little or no attempt at segregation of lepers: Poona itself also makes little or no attempt, for it has only succeeded in segregating some 120 lepers out of an estimated leper population of 1,000. A village may have six or seven Brahmins, Poona has enough to form a fair-sized community of them, with proportionate influence.

The houses of a village look and are small, decrepit and overcrowded: Poona houses look the same and there are many more of them. The economic position of the villager depends on the land, and there are few opportunities for other labour inside the village: the Poona inhabitant has more opportunities in the commercial and clerkly branches, but as it is not an industrial city, he also must look to Bombay for further work, even as the villager. The presence of the European civil population and soldiers, and the number and range of its educational establishments, are perhaps the only distinctive features of Poona, as compared with the country village.

The average inhabitant of Poona District, whether in the country village or the town, can only be described as semi-civilized.

A BRIEF DESCRIPTION OF LEPROSY AS A DISEASE.

Although the purpose of this Thesis is the study of leprosy in Poona District in its problems of causation and epidemiology, a short clinical sketch of the disease will be of help in orientation of these problems in one's mind. The authorities for the ensuing account are LT.-COL. GREIG (in his lectures during a course in Tropical Medicine personally attended at Edinburgh), MANSON-BAHR[53] ROGERS & MUIR,[54] STITT,[55] MUIR,[56] and COCHRANE.[57]

Definition. Leprosy is a chronic infectious disease, usually of long incubation and duration, which involves chiefly the skin and mucous membranes on the one hand, and

the peripheral nerves on the other, and is characterised by the formation of new non-inflammatory tissue due to the proliferation of Hansen's bacillus (1871) or Bacillus Leprae.

Cause. There is a general consensus of opinion that human leprosy is due to the organism first described by HANSEN in 1871, now called the Mycobacterium leprae, or Bacillus leprae. This belief depends on the repeated finding of the bacillus by workers all over the world, in greater or smaller numbers in all leprous lesions. Final proof by animal inoculation with the production of lesions in all respects similar in course and in histological appearances to those in man is still lacking. The organism in morphology rather resembles the tubercle bacillus. With the Ziehl-Neelsen stain it is seen as a uniformly stained rod, straight or slightly curved, varying in length from 1.5 to 6.0 microns, and in breadth from 0.2. to 0.45. microns. In highly positive lesions the organisms are present in very great numbers, and they tend to be arranged in bundles of greater or smaller size. In weakly positive lesions this feature is not present. The chief features of the organism are:-

1. It is, in general, acid-fast:
2. It grows on artificial culture media with difficulty or not at all:
3. Its pathogenicity is low:
4. Introduction of it into laboratory animals has not so far resulted in the production of typical leprous lesions:

5. Ordinary methods of examination frequently fail to reveal it in certain types of lesion which are clinically leprotic in nature.

Predisposing Factors. While there are still factors in the etiology of the disease that have not been elicited, the chief factor seems to be close and prolonged contact with lepers who are discharging the bacilli from their bodies. Children and young adults and those whose resistance has been lowered by disease or by unhygienic living or inadequate diet, seem to be in the greatest danger of acquiring the disease. Transmission by water, food and contaminated soil cannot definitely be excluded. There is little direct evidence of insect vectors. Leprosy is not hereditary, for children born of leprous parents, if separated very early, are no more liable to develop the disease than other children. Leprosy is environmental rather than hereditary.

Mode of Infection. This is unknown. There is a general impression that there is entry of the bacillus through a lesion of the skin or mucous membrane. It is supposed that if an individual's resistance is lowered by ill-health, or by any other means, a small invasion of bacilli from any source may be the starting point of the infection. Workers have shown that in some cases leper bacilli can be found in the fluid aspirated from lymph nodes of healthy contacts. It is legitimate to conclude that the organism may lie latent in some tissue space or lymphatic gland, and if at any future date the health of the patient

is lowered, the disease may manifest itself. This is a possible explanation of the long latent period of the disease, which may extend from a period of three months to ten, twenty, thirty or more years. A great deal of evidence exists that leprosy commences as a local lesion at the site of infection.

Pathology and Symptomatology. The essential lesion in leprosy is a non-imflammatory proliferation of tissue, called the leproma or area of leprous infiltration. Secondary lesions develop later in the form of ulcers and trophic sores. The leproma varies in character according to site, whether in skin, nerves or mucosa. It consists of a proliferation of tissue cells, round cells, spindle cells and large vacuolated cells, and bacilli of leprosy are present in numbers and lie in and between these cells. The term 'leper cell' is applied to the large vacuolated oval or irregular cells containing several nuclei and large numbers of bacilli. They are rather like the giant cells in tuberculosis. 'Globi' is a term applied to masses of bacilli which lie between the tissue cells. 'Gloea' is applied to a remnant of a cell which has undergone degeneration by reason of invasion by lepra bacilli.

The leproma is moist, has a pearly-white or slightly yellow tinge, and varies in size from a pin's head to an inch or more in diameter. It differs from tubercular lesions in that it does not readily necrose. The leproma

is most often found in the skin, mucous membranes, submucous tissue and peripheral nerves (a perineuritis).

Clinical types of leprosy are three, - nodular leprosy, of the skin and mucous membranes: nerve leprosy: and mixed cases.

Symptoms of Nodular Leprosy. There may be prodromal symptoms, such as febrile attacks, the nature of which is not recognised. The first sign of importance is a localised thickening of the skin, the leproma, which may or may not exhibit a certain amount of discoloration. The colour is apt to be reddish or slightly fawn-coloured, but there may be quite normal skin colour. The thickening of the skin may be in the form of a definite nodule, or may be diffuse, or in a plaque. The first lesion may be single, or a dozen or two may appear at once. The commonest site of the early leproma is the face, especially the cheeks, eyebrows, chin, alae of nose, lobes of the ears.

The nodule or diffuse thickening may slowly increase in size and become prominent, or may disappear. In a month or two additional lesions will appear. The lepromata show little tendency to ulceration; the most to be seen is a superficial erosion healing with a white scar. The result of successive crops is to produce considerable infiltration of the skin, giving rise on the face to the characteristic leonine facies due to the bosses and folds of the thickened skin. Internal organs usually remain clear, but leprosy bacilli have been found in deposits in the liver

and spleen. Eventually, longstanding lesions of the skin or mucous membranes may undergo extensive necrosis and ulceration.

The most important systemic symptom is fever. It is of two types. Leprous fever is the febrile reaction which invariably ushers in a new crop of skin lesions: it lasts a few days to a few weeks, and then subsides, the ordinary health returning. This goes on for a long time, until the terminal type of fever, with the final decline, sets in, with emaciation, sweating, and general weakening. The course in nodular leprosy is usually long, though rapid cases do occur. Lesions of the mucous membranes are certain sooner or later in nodular leprosy; the mouth and nose are common sites, and ulceration of the larynx leads to the characteristic sign of huskiness of the voice. Conjunctival and eye tisses involvment is frequent, leading often to complete disorganisation of the eyeball, and blindness. The scalp is curiously involved but rarely, but eyebrows are common sites of lepromata, and their hair is lost.

Symptoms of Nerve Leprosy. The first sign is usually shooting pains along the distribution of some of the peripheral nerves, as the developing lepromatous tissue produces irritation of nerve fibres. The shooting pains are usually associated with discoloration of the skin, either increased or decreased pigmentation. As a rule, early alterations of sensation occur, the first change being bouts of sweating and hyperaesthesia in the part involved. Later on,

hyperaesthesia changes to anaesthesia, but that may not be for years. The early signs are those of peripheral neuritis. The destruction of the nerve is a gradual process, and the sensations disappear in the following order:-

(1) Fine temperature sense.
(2) Fine superficial touch sense.
(3) Gross pain and temperature sense.
(4) Deep pressure sense, Joint sense is lost very late in the disease, it at all.

Beading or spindle-shaped thickenings, one or more, are readily palpable on the nerves affected. The commonest pigmentation changes in diminution, causing the well-known vitiliginous or leucodermic areas. These vary in size, from a centimetre across to as big as the palm of the hand. Confusion may be caused with true leucoderma of non-leprous origin, but testing the skin sensations, palpating the nerves, and bacteriological examinations of a skin clipping will serve to differentiate leprosy. Reddish patches of hyperpigmentation may be seen. These patches tend to lessen the excess of pigment in the middle, while spreading at the periphery, which gives a ringworm appearance.

As the condition progresses, nerves become more and more destroyed, and motor changes, muscular weakness, and atrophy appear. Due to atrophy of the thenar, hypothenar and inter-phalangeal muscles, the claw-like hand very typical of leprosy develops. Similar changes in the foot give rise to foot drop and inversion, with a characteristic gait.

Facial muscle palsies and loss of expression, and ptosis are common.

Lastly come trophic changes, with necrosis of bone, ulceration of soft parts, bullae, pigmentation and other skin lesions. Plantar ulcer is very common, and a penetrating painful ulcer also occurs in the palm of the hand. Such ulcers begin on the ball of the toe or the heel, and are extensive and deep. Extensive mutilation results as the disease progresses: joints are involved, fingers lost, hands reduced, feet become stumps, and the like. Spontaneous absorption of bone leads to great shortening of the phalanges.

In mixed leprosy, all types of symptoms occur together.

Course of Leprosy. It has long been known that leprosy tends to be a self-healing disease, and it runs a prescribed course which varies with each individual. The leper usually dies from some intercurrent affection, but sometimes as a result of the cachexia induced by repeated reactions, or rarely during acute lepra-reaction. If the disease continues to its ultimate end, as it usually does, in the untreated individual, it leaves the patient deformed and mutilated, which is the stage formerly described as 'burnt-out,' but now as 'arrested with deformity.' Even after the disease has died out, deformities and ulcers continue to form as the result of nerve destruction. Investigations within the last decade have established the fact

that in the majority of countries leprosy passes through three stages:-

(1) The early non-infectious nerve stage, which not infrequently commences as a localised lesion.
(2) The intermediate contagious skin stage.
(3) The innocuous stage when the disease has died out of the body leaving the subject a mutilated wreck.

CLASSIFICATION OF TYPES OF LEPROSY.

The Internation Conference on Leprosy held at Manila in 1931, has recommended a classification of cases of leprosy. As this classification is coming into universal use, a short description of the main features is necessary.(58) As all diagnosable cases are in a sense 'mixed,' the classification is based primarily on the predominating clinical findings.

A. MAIN TYPES.

All cases that show evidence of actual or previous nerve involvment are designated Neural, or N cases. The nerve changes are not accompanied by leprotic changes in the skin.

All cases showing leprotic lesions in the skin are designated Cutaneous, or C cases. Such cases may or may not show, at any given time, clinical manifestations of nerve involvment.

B. SUB-TYPES (INDICATING DEGREE OF SEVERITY).

Neural - 1 or N.-1. are Slight Neural cases with one or a few small areas of distributed sensation, which may or may not show:-

alteration of circulation,
alteration of pigmentation,
paralyses,
trophic disturbances of minor degree.

Neural - 2. or N-2. are moderately advanced neural cases with extensive or numerous areas of disturbed sensation, not confined to any one part of the body: with paralyses and/or

visible evidence of trophic disturbances,
marked depigmentation,
moderate atrophy,
Keratosis,
bullae, etc.

Neural - 3. or N-3. are advanced neural cases with more or less extensive areas of anaesthesia and marked motor and trophic disturbances: and marked paralyses,

atrophies,
contractures,
trophic ulcers,
mutilations.

Cutaneous - 1. or C-1. are slight cutaneous cases with one to few leprotic macules, or a few small areas of infiltration or nodules,

Cutaneous - 2. or C-2. are moderately advances cutaneous cases with numerous leprotic macules, or fairly numerous or marked areas of infiltration or nodules, frequently with lesions of the mucous membranes.

Cutaneous - 3. or C-3. are advanced cutaneous cases, with numerous or very marked leprotic lesions in various stages of development or ~~tret~~ retrogression, usually with lesions in the mucous membranes.

In all cutaneous types there may be varying degrees of

neural involvment, and such cases may be recorded, as, for example, C-2, N-1.

The Internation Conference on Leprosy also defined certain terms, which, as they are of great use, are given below:-

Leprotic. - This term should be applied to those changes which present clinical or microscopic evidence of processes of a granulomatous nature, apparently caused by the presence of the Mycobacterium leprae in them. In such lesions the organism can usually be demonstrated by the ordinary methods of examination.

Leproma. - This is applied in a general sense to any lesion of a leprotic nature.

Ulcers. - Ulcers in leprosy are of two types. The leprotic ulcer occurs in leprotic tissues and discharges bacilli: the tropic ulcer is a sequela of nerve changes, usually occurs in non-leprotic tissues, and does not discharge bacilli.

Infiltration. - This is a term applied to a diffuse thickening of leprotic nature involving the skin or mucosa which is not of definite nodular, papular, or macular form. The term may also be applied to diffuse leprotic conditions in other organs.

Nodule. - A nodule is a definitely thickened, rounded circumscribed mass of leprotic nature commonly occuring in the skin, subcutaneous tissue, or mucosa.

Papule. - A papule is a small solid elevation of the skin, of leprotic nature, not more than 5 millimetres in

diameter.

Macule. - A macule is a circumscribed area of the skin showing slight elevation or depression, and changes in colour. A macule may be hypopigmented, hyperpigmented, erythematous, circinate, marginate, zonal, raised, atrophic.

PREDISPOSING CAUSES OF LEPROSY.

It is pointed out by E. MUIR (59) that the leprosy germ will not grow in the healthy human body, and the soil must be prepared for its growth by some predisposing cause or other. He, among all the workers on Leprosy, insists most on the great importance of these factors, and he enumerates them as follow:-

Various Debilitating Diseases.

(1) Those which are self-healing, such as enteric, small-pox, cholera, influenza.

(2) Those which have to be removed by special treatment, as syphilis, staphylococcal and streptococcal infections, malaria, dysentery, helminthic infections.

Dietary Defects due to:-

(1) Improper balance of the various constituents of food, lack of vitamins, unsuitable quantities of food, over-indulgence or starvation.

(2) The eating of decomposing food, such as stale rice, salted fish, decomposing meat.

(3) Over-cooked, unsuitably cooked or rich food.

Pernicious Habits, such as:-

(1) Laziness.
(2) Overworking.
(3) Irregular habits.
(4) Sexual excess.
(5) Lack of personal cleanliness.

Certain Physiological Conditions:-

as puberty, pregnancy and lactation.

Climatic Conditions:-

Excessively hot or cold climates are unfavourable, especially if accompanied by high humidity.

Insanitary Surroundings:-

These include over-crowding and lack of sunlight.

The Mental Attitude.

The mental factor is a very important one, and the fear of leprosy not infrequently predisposse to its occurence and leads to its increase.

The above predisposing causes depend on the orthodox view that leprosy is a germ disease, and when this is denied, any one of these factors may become exalted into a prime cause. Of such a nature is the JONATHAN HUTCHINSON fish theory,[60] which dates from pre-bacteriological times. In 1863 he declared leprosy to be a 'fish-eater's gout,' and dependent on excessive consumption of badly-preserved or decomposed fish. He afterwards modified this view, holding that eating any bad or half-cured fish on a single occasion might produce leprosy developing many years later. After he had himself seen that in the Basutos in South Africa leprosy had spread among a people who eat no fish, he once again modified his theory by admitting that in addition to arising from consuming bad fish, leprosy may also sometimes originate by 'commensal communication,' such as by eating food contaminated by a leper.

An interesting modern theory of the same type is that advanced by L. COOK, 1933,[61] that leprosy is a deficiency disease. He states that we have failed to prove Hansen's bacillus is the causative agent of the disease: that we have no positive proof that the disease is contagious; and that the prolonged incubation period is not a feature common to any other disease known to us which is supposed to be caused by infection. In the countries showing the greatest prevalence of the disease, the people live under primitive conditions, with concomitant limited nature of the dietary. In suggesting that leprosy is a deficiency disease, he gives these points:-

(1) Clinically, a peripheral nervous lesion is predominant, and cutaneous lesions are bound up in their origin and nature with nerve lesions, i.e., are of a trophic variety.

(2) Hansen's bacillus may be the result of the disease, not the cause, analagous to Negri bodies in rabies.

(3) It is an imbalance of the metabolism of the tissues due to some defect in diet, which causes the formation of toxic products affecting the peripheral nerves.

Further, he compares pellagra and leprosy, indicating as the common factors in these two diseases:-

(1) There are exacerbations and periods of quiescence:

(2) Skin affections:

(3) Nervous affections, in pellagra chiefly cord and brain, in leprosy, of the peripheral nerves.

(4) There is an acute form in both diseases, namely, pellagra - typhus and lepra fever, respectively.

In Bihar and Orissa, he points out, the diet is the

same for every meal and every day. There is a limited variety, cereals, pulses and even vegetable foods are preserved dry, and there is an absence of fresh foods, such as milk, meat, fruit and vegetables. He suggests an inherent familial susceptibility to nervous affections, and the slow formation of a poison as the result of imbalance of metabolic processes as the explanation of the long incubation period.

Whatever may be the merits of this theory of COOK, the possibility of a connection between pellagra and leprosy would be difficult to deny. J. LOWE, 1931,(62) reported 40 cases of pellagra at the Leper Hospital at Dichapli in India and R. M. WILSON, 1925,(63) and 1933,(63a) reported an outbreak of pellagra in a leper institution in Korea, a country where pellagra is either unknown or very rare. The mystery of the connection between the two diseases is as yet unexplained. LOWE found the diet was much the same in his leper pellagra cases as in non-lepers. The protein and fat were markedly deficient, the protein of poor biological value and the carbohydrate in excess; mineral salts and Vitamin A were deficient.

MUIR and SANTRA, 1932,(64) made a valuable survey of India as regards predisposing factors. 1. They found that famine conditions due to failure of crops led to a high incidence of leprosy, as in Western Bengal. 2. Tracts which a short time previously had been reclaimed from the jungle, leading to the 'jungly' people's admixture with

surrounding more civilized folk, were the seat also of increased leprosy, as in East Bihar. The explanation was that people in such a state of transition were more susceptible to the infection, or in many cases met the danger of leprosy for the first time.

3. People were predisposed to leprosy in areas where there are backwaters connected with the sea or river mouths, with periodical flooding of a low-lying delta, as in Travancore. In these areas filarial infection is very high, and possibly the high incidence of leprosy may be partly dependent on this factor.

4. In mountainous tracts where the people suffer from privation in the winter months, where villages are insanitary and isolated for a large part of the year, visited chiefly by wandering pilgrims, who are often lepers, once leprosy gains entrance it is apt to spread alarmingly. Examples are Nepal and Kashmir.

5. They also found great susceptibility in highly industrialized areas into which labour had been imported from highly endemic areas without due safeguards, as in Assam and in Calcutta.

6. Leprosy in India they found to belong to a certain stage of civilization. Aboriginal tribes in inaccessible districts were free. When the aboriginal begins to hire himself out as a labourer, he is apt to forsake his tribal rules and is exposed to greater infection. The semi-aboriginal is very liable to leprosy. He is also

responsible for spreading the disease to the house of his employer and to the higher class employees, as in the case of the Pariahs and Pillays of Travancore.

7. In villages where caste rules were strictly observed, there was little or no leprosy. There was more lsprosy where there was mixing of the people, and laxity of caste rules.

8. In industrial centres, where labourers, skilled and unskilled, are congregated in a small area, sexual and other promiscuousness is common and predispose to leprosy. Thus in Bengal, in an industrial population of 172,870. examined, 1.3%. were leprous. Sexual promiscuousness has much to do with leprosy, though there is no reason to call leprosy a veneral disease.

9. Dietary errors are thought by MUIR and SANTRA to be a major factor in India. Inadequacy of diet is very widespread. The porosity of soils frequently lead to famine when the rains fail or are irregular. In tracts of such soils (parts of Poona District form such) vegetables also are difficult to grow, and grazing ground is poor. Lack of milk and green vegetables results in a state of health favourable for the growth of the leprosy bacillus in the body. In the Western Ghats region, high rainfall erodes the soil, and the consequent poverty of the crops leads to ill-nourishment. The deficient diet is usually supplemented or made more tasty by the use

of decomposed food, such as stale rice, fish or meat. The food of many town dwellers is deficient in proteins, fats and vitamins, partly due to poverty, ignorance and social or religious prejudices. Highly milled cereals and the adulteration of ghi and oil are replacing the more wholesome food of the villages. (in 1933 in Bombay a series of analyses of ghi sold in the bazar shops were made and over half of these were found to be of samples more than 70%. adulterated with fats worthless as human food). Among Hindus and Buddhists, vegetarianism is the rule, but if milk is not available, the diet is very deficient in animal protein and fat. MUIR and SANTRA think that the scarcity of leprosy in the plains of the Punjab is due, to a certain extent, to the comparatively wholesome diet of the people there. According to the EAST INDIA CENSUS of 1931,(65) Hindus and Buddhists together number over 251 millions, which makes vegetarianism and the deficient diet it leads to a matter of great importance.

Lack of milk, mentioned above, is characteristic of India.(66) The ROYAL COMMISSION ON AGRICULTURE IN INDIA, 1928, draws attention to the fact that, in addition to the miserably poor production of milk, the climate makes the keeping and transport of fresh milk difficult. The greater part of what milk is produced is consumed in the form of ghi, curds and sweetmeats. In all the larger towns, the supply of fresh liquid milk is small; in Bombay, it has been estimated at about seven gallons, and in Calcutta

about eight gallons per head per annum. The price of pure milk is high, and if it could be reduced to half the current rates, there is little doubt that the consumption would be more than doubled. Owing to an increase in the habit of tea drinking in recent years, the demand for milk in urban centres has increased, and there are now considerable imports of condensed milk, especially into Burma. Throughout India generally, the supply of fresh milk in villages is very defective. In the Central Provinces, the supply is estimated at less than three ounces per head daily. In Bombay, it is less than two ounces per head daily. In Bihar and Orissa, the supply is similar, while Madras and the United Provinces are slightly better. All the evidence available points to the conclusion that the consumption of fresh milk in India is very small when compared with such countries as the United States, Denmark, Sweden and Switzerland. "The difficulties in the way of economic milk production and distribution in India are formidable;" such is the conclusion of the ROYAL COMMISSION.

10. MUIR and SANTRA conclude that apparently there is no racial predisposition to leprosy and MUIR in another place[67] describes evidence against any hereditary or racial lowered resistance, and in favour of the great importance of healthy surroundings in governing the incidence of leprosy. In connection with the largest leper colony in India, that at Purulia, there is a village,

Uffmanpur, a few hundred yards distant. This village is inhabited by the descendants of the colony, who were separated as children from their leprous parents. Many of these have grown up, married and borne children. Among these children scarcely any cases of leprosy have occurred, in spite of their close proximity to the leper colony. The population of this village was 194 in 1930: the average birthrate for the last five years is 54. per mille, and the average deathrate is 3.7. per mille. Yet many of the parents and grandparents of the villagers were acute and advanced cases of leprosy.

11. After mentioning as a finding of their survey that "one of the important factors in inducing high incidence of leprosy in certain areas is the low resistance of the populace due to other endemic or epidemic diseases," they mention some of the predisposing diseases. These include malaria, the dysenteries, helminthic infections as ancylostomiasis and filariasis, syphilis, typhoid and influenza.

As well as in Travancore already mentioned, in the highly filarious districts of Orissa and Northern Madras, leprosy is highly endemic.

In the plains of Bengal the most important predisposing cause is malaria. Leprosy when first introduced into a highly malarious area spreads rapidly.

Dracontiasis is another disease which is found to predispose to leprosy. In a Satara village (Satara is the

District adjoining Poona District) they found that the abolition of a step well led to the disappearance of dracontiasis and the diminution of leprosy incidence.

Examination of the inmates of five leprosy institutions showed an average of 90%. infection with hookworm. Treatment for this was in Purulia instituted and regularly carried out, and improved the general condition of all the inmates.

Leprosy is found frequently to follow kala azar in Bengal. MUIR first reported these cases in 1927.[68]

After the influenza epidemic in 1918-1919, many patients dated the beginning of their leprosy from an attack of influenza.

12. Leprosy is chiefly a disease of the lower classes, but it is also found among the wealthy and those tempted to self-indulgence. MUIR and SANTRA record that sudden wealth coming to a village in the Manbhum District, owing to the sudden demand for lac, led to various forms of indulgence, and the leprosy endemic there spread with alarming rapidity.

The experience in the United States is rather different as regards class incidence. R. HOPKINS and O. E. DENNEY, 1929,[69] in their study of 700 cases in the National Leprosarium, found that "The social status of the patients is a cross-section of the normal populace as regards education, wealth, and culture." Again they state that the incidence of leprosy in the white population of Louisiana is computed as twice that in the negro population.

MUIR and SANTRA conclude their survey of India with the statement that leprosy is probably very widespread, but only develops into a clinically or bacteriologically recognisable disease in a fraction of those infected; natural resistance must exist in the remainder. When leprosy does develop, it indicates either hyperinfection, or probably more commonly, lowered resistance. This lowered resistance appears to be due chiefly to various factors which affect the general health, as predisposing ans accompanying diseases, improper diet, unhealthy habits, climate and surroundings.

Sex as a factor in leprosy.

The conclusions come to by J. LOWE, 1932,[70] and 1934,[71] from his study of the subject are:-

1. In practically all countries where leprosy is highly endemic the number of male lepers exceeds the number of female lepers, usually by about two to one.

2. There are differences in the sex incidence at different age periods. In childhood, as reported in some countries, the frequencies are approximately equal. In other countries are male children more affected. Certainly the difference is less marked in childhood than in adult life. Puberty very often causes a relative increase in leprosy rates in females.

3. There is some evidence that sex influences the form of leprosy, men showing on the whole a severer form of the disease and a greater mortality than women.

4. The possible causes of the differences in the sex incidence of leprosy are considered to be environmental and physiological. The chief factor is probably environmental. In many countries men are more exposed to infection and to conditions which predispose to leprosy. Environment does not seem to explain fully the difference in incidence in some countries, and it is possible that physiological differences may be associated with the difference in suseptibility.

I. KERR, 1932,[72] thinks that if women lived under the same conditions as men, they would show an equal or even greater incidence of leprosy. She points to the fact that in Africa, where women live under the same conditions as men, the incidence is about equal. In India, the incidence on women is 28%. of the total. Both these observers have in mind certain predisposing factors pertaining to women, and remark on the advances in leprosy and the higher incidence in women in association with puberty, pregnancy, labour, lactation, menstruation and catamenia. I. KERR has observed that, next to concomitant disease, the menstrual periods are what aggravate leprosy in women and increase its virulence. Of the 100 women in the Dichpali Leper Home, she states that five definitely trace the onset of leprosy as coincident with their early menses, while two of them are particular that the symptoms appeared with their first menstruation. Others assert that fresh lesions and aggravations of old ones took place concomitant

with their early menses. It is possible to watch, she says, the rhythmical advance of leprosy in many women during their menses. In about half the women, leprosy began after confinement. The explanation suggested by I. KERR is that a deficiency of blood calcium is a predisposing cause of leprosy, and in women menstruation and maternity are the most serious causes of calcium deficiency in the tissues. She has noted that syphilis, which hastens the progress of leprosy from its earliest stages, and predisposes to secondary nerve leprosy, also seems to aggravate menstruation more than any other complication in the disease. She has found the Kahn Test for syphilis was positive in over half of the cases who had normal and increased periods, while it was positive in less than a fourth of the women who had amenorrhoea. Her inference is that syphilis has its influence on leprosy in women by reason of its apparent power to deplete the blood calcium.

LOWE also draws attention to similarities in the sex incidence of leprosy and tuberculosis. Though on the whole incidence is heaviest on men, age incidences and sex incidences are similar. After puberty, tuberculosis falls heaviest on females, up to the age of 25, probably connected with puberty and pregnancy, and their consequences in young women.

Leprosy predisposed to by shock.

J. E. BRAUL, 1930 (73) traces the onset of leprosy in

eight cases to shock: as an operation resulting in chloroform trauma of the lungs, a burn of the foot, an induration of the gluteal muscles following an injection; in the other cases, aggravation of existing leprosy took the form of a mixed infection, following an attack of malaria or influenza.

Leprosy predisposed to by overcrowding and caste laxity.

I. SANTRA, 1930,[74] from his study of Puri District in India, gives a few striking figures to show the influence of congestion and laxity in observing caste in the villages.

TABLE XI.

Type of village.	No. of Villages	Total populn.	Average pop in each village.	Total No. of lepers.	Average No. in each village.
All one caste	20.	2,985.	149.	8.	0.4.
Separate castes in separate quarters.	73.	20,575.	280.	176.	2.4.
Many castes with their houses all mixed together.	88.	52,300.	600.	526.	6.0.

It will be seen that in the 'mixed villages' the incidence was very much higher. In addition to overcrowding, SANTRA found that floods and famines had gradually devitalised the people. Under present economic conditions the villager is reduced to a state of poverty. His diet consists mainly of soaked rice and dried fish. This low diet gives him less energy, hence less work and less wages, and

again less food. The joint family system and the inability to buy more land condense families into small houses.

Leprosy predisposed to by oral sepsis.

I. KERR, 1930 (75) examined 140 cases, and found that only two had really clean healthily-kept teeth. All the others were distinctly dirty, while 75%. of them were suffering from pyorrhoea alveolaris, or conditions which would lead inevitably thereto. Inadequate cleaning of the teeth is one cause of this state of affairs. No really effective antiseptics are used by Indians: they use 'babul twig' (of Acacia arabica) which does not enter between the teeth, and food clogs about the interstices and decays. Another cause, and the most serious, is the custom of 'pan' and betel chewing. This habit not only wears down the teeth, but is liable to lead to inflammation of the margins of the gums, which goes on to pyorrhoea. Such suppuration is injurious to health, lowers the resistance of the patient to diseases such as leprosy, and keeps the resistance low. Moreover, the gastritis and chronic indigestion usually found with bad teeth will also lower resistance.

Predisposing Causes in Cooch Behar.

B. N. GHOSH, 1931 (76) found that among the 1200 lepers in this native state in India leprosy was predisposed to mainly a devitalising diet, worms, pyorrhoea,

syphilis and the damp hot climate.

Predisposing causes in Puri District.

U. P. GUPTA, 1931,[77] reports that Puri is one of the most important pilgrim centres of the Hindus. Annually millions of pilgrims assemble there. The congestion is at its height during the 'rathjatra' or car festival. The Hindus believe that leprosy could be cured by paying homage to the god Jaganath, and before the days of the railways, the pilgrim lepers used to travel by road, and take shelter in the villages for a night or more. By this means the villages of Puri and those on the route to it must have become infected, and leprosy has probably been endemic for a long time. Other conditions are suitable, for Puri is an endemic focus of filariasis and malaria is also prevalent in the District. The incidence of diarrhoea and dysentery is unusually high, and there is a high mortality from cholera every year, especially after the car festival. Syphilis and gonorrhoea cases attend dispensaries in large numbers and can be called common diseases in the District. Skin diseases are found amongst nearly 4% of the patients attending the various dispensaries. These factors reduce the vitality of the people of the locality and act as predisposing causes for leprosy. Lastly, the poor quality of the diet of the average Oriya, diet poor in proteins, fats and vitamins, combined with ignorance and lack of proper hygienic knowledge are very marked factors in Puri.

Predisposing causes in Manbhum District.

B. N. BOSE, 1931,[78] found that the villagers, due to their callousness and ignorance of leprosy and of ordinary personal hygiene, associated most indiscriminately with highly infectious lepers. He found infectious lepers sitting in publis meetings and smoking the same pipe or hookah with healthy persons. Isolation is practically unknown, and infectious lepers are found living in the same room with other healthy members of the same family. There is lack of houses and overcrowding. The diet is defective, consisting mainly of rice and maize, and so containing too much carbohydrate and too little protein, fat, vitamins and salts, especially calcium and phosphorus. In the remotest villages, scarcity of good water for bathing or drinking is a contributing factor to the low standard of living. There is an excessive prevalence of malaria, hookworm, and venereal diseases.

Predisposing causes in Cuttack District.

U. GUPTA, 1932,[79] found a low incidence of leprosy among the high castes and a great incidence among the lower castes. This he ascribes to the exclusiveness of the one and the more promiscuous intercourse of the other. A low economic condition was found to be the case in those suffering from leprosy. About 50%. of them had not sufficient income to maintain a reasonable standard of living and diet. The diet is of poor quality, consisting mainly of rice, dal

and vegetables, and poor in proteins, fats and vitamins. The average labourer has no chance of getting milk, and dried fish is eaten in the interior: it is apt to cause chronic indigestion. Diseases like malaria, filariasis, syphilis and intestinal worms are prevalent.

Presisposing causes in Kangra District.

S.S. JAIKARIA, 1932,[80] found general ignorance and lack of proper sanitary knowledge, and defective diet, (deficient in fats, proteins and vitamins). Allied to these factors is the prevalence of bowel complaints such as dysentery, diarrhoea and constipation. High prevalence also obtains of veneral diseases, malaria, early rickets, osteomalacia and goitre.

Predisposing causes in Burdwan District.

B. N. GHOSH, 1932,[81] ascribes importance to poor condition of the soil, and deficient and irregularly distributed rainfall, for their influence on the economic state of the people, which is generally bad. Exceptionally high atmospheric humidity is a factor of importance, likewise certain diseases, as kala azar, intermittent and remittent fevers, intestinal diseases, veneral diseases and defective diet.

A continuous flow of immigration from neighbouring districts of high leprosy endemicity is spreading leprosy in Burdwan. The increasing density of the population, unclean habits and insanitary mode of living of the people, and promiscuousness, ignorance of the people regarding the

infectious nature of the disease, are other predisposing factors.

Predisposing causes in Midnapur District.

K. R. CHATTERJI, 1932,[82] states "Poor soil breeds lepers." Poor soil cannot grow sufficient food and predisposes to leprosy. He points out the striking difference in this respect between the southeast and the northwest halves of the District. In the southeast half, the land is flat alluvial and the soil is fully cultivated and fertile. The people have a good diet and even obtain milk, and leprosy is scarce. The northwest half is laterite soil, porous, and difficult of cultivation. Due to its porous and sloping nature, the rain water runs off, washing away the surface soil, and large areas of land have little productiveness. Cows are ill-fed, milk is scarce, and vegetables almost unknown. The diet of the people in this tract is, therefore, limited to rice and the less digestible forms of dal. Animal food is in the form of badly-preserved fish, and decomposed carcases of cattle and other animals in the case of the low castes. Boiled rice is often taken by preference soaked in water overnight, because of the slight decomposition, and certain castes take rice beer in large quantities. Not only is the diet bad, but there is an added toxic factor in the shape of the decomposition products, in the bad fish, meat and rice often eaten. Another factor is that the people of the poor areas may migrate to industrial areas and con-

tract veneréal disease, or leprosy itself.

CHATTERJI classifies the predisposing factors in the case of 791. lepers as follows:-

BAD DIET	622.
VENEREAL DISEASES.	121
MALARIA, DYSENTERY, PYORRHOEA, TYPHOID.	50.

Predisposing factors in Central Provinces.

D. N. MUKHERJI, 1930[83] found a high incidence of leprosy in the Chhatisgarh Division of the Central Provinces, due to the following factors:-

The climate is hot and moist.
The soil is poor.
The people are in a very low stage of civilisation, though the primitive tribes as yet are free from leprosy.
The people are very dirty in their habits and their houses and surroundings are insanitary.
The diet is defective. They take nothing but rice, and that is stale. Milk, vegetables and other vitamin-containing food is lacking.
The Chambar caste eat carcases of carrion, and in this caste the incidence of leprosy is highest.
The morals of the people are lax. Veneréal diseases are rife. Promiscuousness is common.
Amoebiasis and other intestinal diseases are common as the result of drinking polluted tank water.
Filariasis was found endemic in one thana, and some hookworm.
There is a marked absence of any fear of leprosy.

Predisposing factors in Muzaffapur District.

U. P. GUPTA, 1930,[84] incriminates malaria, kala azar, round worm and hookworm as the chief among the predisposing diseases. He goes so far as to name overwork as a factor. There is a want of nutritious food and as 'khesari' occupies the chief role in the diet, it is suspect as a devitalising agent. 'Khesari' or 'Khasari' is a vetch (Lathyrus sativus and allied species) and is respon-

sible for a very chronic, seldom fatal disease called lathyrism, characterised by ataxy, spastic paraplegia, weakness, muscular pains, incontinence of urine and impotence .[85] It occurs in India where vetches form the main article of diet, as in Muzaffapur. It is curious that here is another disease associated with diet, like pellagra already mentioned, which is suspected of having some relation to leprosy. In all three diseases the nervous system is affected.

Predisposing causes in the Tippera District.

B. N. GHOSH, 1931.[86] established the fact of the high incidence of leprosy in this District. He shows how trade intercommunication with highly endemic centres like Mymensingh, Sylhet and Dacca, has caused a great spread of leprosy in Tippera. No leper in Tippera has as yet been put under treatment, and highly infectious lepers are allowed to have free access everywhere, and even to dine with others, as at marriage ceremonies. Ignorance of the disease is wide and deep. The exceedingly high humidity of the country, the increasing density and overcrowding of the population and lack of knowledge of living, are other important factors.

Predisposing causes in Purushothampur.

K. PAL, 1931.[87]. reports 80%. hookworm infection in 222 patients. Venereal infection is not uncommon, as thousands of coolies migrate to Burma every year, and they lead a lax bachelor life there. As a consequence, both

syphilis and leprosy are common among the coolie classes. In the majority of cases, general debility and overwork due to chronic poverty and life in conditions of stress, on food poor in quality and quantity, are the underlying causes of sustaining leprosy infection.

Predisposing causes in Bhutan.

J. A. MACDONALD SMITH, 1933,(88) has little doubt that over-crowding is the explanation of the great prevalence of leprosy in Bhutan. In that country there is an unfortunate tax on houses, not on the people. Naturally the inhabitants try to do with as few houses as possible, and overcrowding is appalling. The houses are double-storied. In the rooms on the ground floor are housed the cattle, ponies or mules, and part is used as a storeroom. Upstairs, not very high up, are the living rooms. In one room is a Buddhist altar, and it is said that only lamas are allowed to sleep in it, which still further restricts the accommodation. The average house has two other rooms, one a common room where all sleep, and the other a smaller room used as a kitchen. Several related families may live together in the same house, with the result that 13 to 20 people may sleep together in the common room. They sleep in a long row from wall to wall of the room, some in the corners, and a few in the kitchen.

Predisposing causes in Kulu.

S. S. JAIKARIA, 1933,(89) shows that malaria, dysentery, round worms, goitre, syphilis and gonorrhoea are

prevalent predisposing diseases. Ignorance and lack of knowledge of hygiene are the rule. The people shut themselves up in their stone houses in winter, and infectious lepers mix freely with all, sharing the same food, floor, blankets and hookah.

Predisposing causes in Hlegu, Burma.

I. SANTRA, 1933,(90) mentions that some of the patients thought that taking mercurial preparations was a predisposing cause of leprosy. Others blamed irregularities of menstruation. The native doctors of Burma incriminate crabs, prawns and the flesh of 'thamin,' a kind of spotted deer. That close intermarriage may have some bearing on leprosy is suggested by the history of the royal family, the first leper in which was the eldest of a group of brothers and sisters who married each other according to the custom of the royal family.

Excessive courtesy in Burma must be ranked as a predisposing cause, for though most of the people know that a leper may communicate his disease, they are too courteous to prevent a leper coming into their houses. In Burma the village headman has by law some powers of segregation of lepers, but they are seldom effectively used, and even so the segregation is apt to be stimulated by the appearance of the leper and not his real infectivity.

Comment by HAYASHI on Syphilis and on Trachoma.

FUMIO HAYASHI, 1933,(91) in his impressions of India, writes "I was astonished at seeing so may lepers with

syphilis, especially at Purulia and Chandkuri, and with perforation of the palate or depression of the nasal septum. At first I wondered why all lepers with positive Wassermann or Kahn Tests at the School of Tropical Medicine are treated with salvarsan, considering that many such reactions in leprosy are not caused by syphilis, but after a visit to Purulia and Chandkuri I saw that this was quite reasonable."

"At Chandkuri Leper Home I saw many trachoma cases. In Japan we have so few cases of trachoma in lepers that some leprologists consider that lepers are immune to it."

Apathy as the most typical attitude to Leprosy.

Fear of leprosy is believed to predispose to leprosy, but in India the typical and most widespread attitude to it is that of apathy or despair. As I. SANTRA (92) says, "From Rameshwarum, the southernmost temple, to Triloknath, the northernmost temple, people believe that leprosy is the result of sins committed during a past birth, and, therefore, beyond human aid."

PREDISPOSING CAUSES IN COUNTRIES OTHER THAN INDIA.

In the form of a table, the experience of some workers in other countries are given below.

TABLE XII.

Predisposing Factors to Leprosy.	Country.	Author.
Yaws, foot yaws, craw-craw, universal diet of putrescent fish, and diet defective, starvation and times of want, dense population, frequent migrations.	Gold Coast	M. B. D. DIXEY, 1930. (93)
Syphilis, gonorrhoea, skin diseases, eye diseases, dental caries.	Southern Rhodesia.	B. MOISER, 1931. (94)
Fatalistic mental attitude in some, extreme fear in others.	British Guiana.	F. G. ROSE, 1931 (95)
Sleeping sickness, venereal disease, ignorance and dirt.	Uganda.	R. G. COCHRANE, 1931 (96)
Pulmonary tuberculosis, living in low-lying areas, lack of personal cleanliness in the high lands, due to coldness of the winter, ignorance conservatism and inertia of the people.	Basutoland.	P. D. STRACHAN, 1931 (97).
Famine, anarchy, mass movements of population from China, inter-marriage and concubinage on a large scale.	Manchuria	J. L. MAXWELL, 1931. (98)
Depressed health due to bilharzia, ankylostomiasis, dysentery, malaria, syphilis, yaws & many other diseases which are rampant.	Tanganyika.	JANET MURRAY, 1931. (99).

Predisposing Factors to Leprosy.	Country.	Author.
Climatic conditions - absence of leprosy in the desert belt, heavy incidence in the equatorial territories. Cannibalism, the tribes with the heaviest incidence being cannibals.	Anglo-Egyptian Sudan.	R. G. COCHRANE, 1931. (100).
Closing of leper hospitals at the wrong time, in 1848, before the disease had been stamped out.	Iceland.	SAM BJARNHJEDINSSON, 1931. (101).
Porous thin soil on coral, with the effect on economic conditions: density of pooulation.	Cebu.	JOSE RODRIGUEZ, 1931. (102).
Depressing temperature and humidity, and heavy rainfall; tropical standards of living; deficient and monotonous diet of 'poi' and lack of meat, fresh vegetables and milk; population of greatly mixed races and large immigrations from centres of endemic leprosy.	Hawaii.	N. E. WAYSON. 1932. (103).
Devitalising diseases, the most prevalent being syphilis, yaws, bilharzia, tape-worm, scabies, chest infections, malaria and filariasis.	Garkida, West Africa.	RUSSELL L. ROBERTSON, 1932. (104).
Syphilis, tuberculosis nephritis and hookworm.	Korea.	R. M. WILSON, 1932. (105).
Damp hot climate following the rivers, and in the littoral.	Argentine.	E. P. FIDANZA, 1932. (XLVIII).

Predisposing Factors to Leprosy.	Country.	Author.
Laziness, ignorance, betel chewing, excessive smoking, interchange of pipes and drinking vessels, lack of segregation.	British Solomon Islands.	L. M. MAYBURY, 1932, (106).
Living on the borderline of starvation, the diet being mainly maize and millet, poor in protein, with little fat and vegetables. Lack of cleanliness due to scarcity of water. Poor physique, bad hygiene, debilitating diseases, as malaria, syphilis, gastro-intestinal diseases, and internal parasites (especially hookworm) are universal. No restriction of intercourse with highly infectious lepers. Lack of fear of leprosy.	Ukuguru District, Tanganyika.	C. A. WALLACE, 1932. (108).
Trypanosomiasis, ankylostomiasis, schistosomiasis, ascariasis, malaria, itch, venereal diseases.	Katanga, Belgian Congo.	E. R. KELLERSBERGER, 1932. (109)
Greater susceptibility to leprosy in youth up to 20 years. Town-dwellers are more liable also.	Surinam.	P. H. J. LAMPE, 1932. (110).
Children are prone to succumb to leprosy.	Palestine.	T. CANAAN, 1932. (111).
High temperature and humidity; dental sepsis; syphilis.	Federated Malay States.	GORDON A. RYRIE, 1933. (112).
High temperature and heavy rainfall: lack of segregation of family lepers; great prevalence of yaws.	Uganda.	L. E. S. SHARP, 1933. (113).

Predisposing Factors to Leprosy.	Country.	Author.
Trying and debilitating climate, with high mean shade temperature, usually dry, but broken by periods of high and oppresive humidity. Prevalence of bacillary dysentery and localised outbreaks of dengue and malaria. Aden is the gravitation centre for the disease of the Arabian Peninsular.	Aden.	E. S. PHIPSON, 1934. (114).
Yaws, malaria, scabies ankylostomiasis.	Uzukoli, South Nigeria.	JAMES A. K. BROWN. 1934. (113).
Great density of population in South Korea; diet mainly of rice and decayed fish.	South Korea.	R. G. COCHRANE, 1929. (116).
Periods of famine and drought, extensive floods; many endemic scourges; civil disturbances and misrule; lack of sanitation; apathy, lack of segregation, numerous leper beggars; leprosy is specially associated with water-logged and ill-drained areas; there is little relation to humidity; poverty is directly related to leprosy; Aboriginal tribes are particularly susceptible.	China.	H. FOWLER, 1929. (117).
Promiscuous mixing of races, with high rate of venereal diseases; yaws, hookworm and malaria are very common.	British North Borneo.	R. G. COCHRANE, 1929. (118).

Predisposing Factors of Leprosy.	Country.	Author.
In a case of accidental inoculation of a French doctor during excision of a leprotic nodule, leprosy manifested itself 10 years later, favoured by lowered resistance caused by a restricted diet, and a suppurating congenital hydronephrosis.	France.	E. MARCHOUX, 1934. (119).
There is probably some racial lack of resistance among the Berberis in Afghanistan, as their diet and mode of living is the same as among the Afghan tribes, which have little leprosy.	Afghanistan	H. A. LICHTWARDT, 1934. (120).
Rat leprosy. - Juxtaposition of an infected ulcer and a recent wound and flies indirectly, favour the development of rat leprosy. Secondary infections, bad nutrition, and bad hygiene favour the generalization of the bacilli of rat leprosy. In one case, a leprosy bacillus taken from a man has been transmitted to the rat.	France.	E. MARCHOUX, 1934. (121).
Prolonged qualitative and quantitative under-nourishment, particularly in youth, is the most disastrous factor in leprous areas. The common diet of polished rice and a little salted meat or fish, is poor in proteins, fats and vitamins. On the contrary under the correct diet, cases of spontaneous recovery have occurred.	Surinam.	E. G. KEIL, 1933. (122).

Predisposing Factors of Leprosy.	Country.	Author.
Low diffusible calcium of the blood serum, even though the total calcium be normal, seems to bear some relation to leprosy, for good clinical results were obtained in 70 such patients who were treated with viosterol.	Carville, Louisiana, U.S.A.	O. E. DENNEY, 1933. (123).
Poverty and a hard life; no special law to segregate; a family source of infection in a third of the cases.	Finland.	AXEL CEDERCREUTZ, 1933. (124).
The exceptional strain to which the people were subjected in the early part of the 19th century, with its wars, distresses and famines, gave rise to the last great wave of leprosy. At this time there was a great deterioration in hygiene, causing overcrowding and dirt and there was an enormous prevalence to scabies.	Norway	H. P. LIE, 1928. (125).
Broadly speaking, to live for many years in places where leprosy is endemic, especially the tropics, predisposes to leprosy; as in the case of a European aged 48. who was given by mistake a hypodermic injection with syringe and needle used on a leper, and developed leprosy in 6 months. He had gallstones, and had lived 30 years in the tropics, but no other predisposing factors were discovered.	Batavia.	C. D. DE LANGEN, 1930. (126).

Predisposing Factors to Leprosy.	Country.	Author.
So predisposed is the child to leprosy if brought up in a leprous environment, that in order to prevent leprosy developing at a later date, the child must be separated from its parents immediately after birth. At least 20%. of the children separated at nine months could not overcome infection received before they were separated.	Groot-Chatillon Dutch Guiana.	P. H. J. LAMPE, 1933. (127).

TRANSMISSION OF LEPROSY.

Unfortunately we are not in a position to make a clear and decisive statement as to the exact method by which the causative organisms are conveyed by one person to another. The difficulties in the way are peculiar to leprosy, and arise from the long and variable incubation period, the slow development of the disease and the natural tendency of patients to hide their condition as long as possible, and the very limiting fact that mankind is the only member of the animal kingdom that has ever become infected with human leprosy. In India and other tropical countries, insuperable difficulties are frequently encountered in tracing the origin of any given case of leprosy, because of the backward peoples dealt with. Such difficulties have led to a degree of dissatisfaction with the belief in the infectious nature of leprosy, resulting in new theories such as that of Cook (61) that leprosy is a deficiency disease. The earlier theory of the hereditary origin of the disease, born out of the pronouncements of DANIELSSEN and BOECK in 1847, and of the REPORT OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON in 1862, is one of merely historical interest. A great wealth of evidence soon accumulated against the theory, and ROGERS and MUIR, (128) state that every important leprosy conference from 1897 to the present date has supported the view that leprosy is a communicable disease.

In view of the lack of precise knowledge as to the trans-

mission of leprosy, it would be well, following MUIR and ROGERS, to marshal all the points bearing on it:-

1. Nodular leprosy is far more infective than nerve leprosy. The larger the number of leprosy bacilli in the body, the more there are to transmit the disease; and the nearer they are to the surface of the body, the more likely they are to be carried to other people. There is a copious discharge of the bacilli from ulcerating nodules , and if the nose is affected, large numbers of acid-fast bacilli are discharged from the nostrils, in nodular cases. On the other hand, there is an absence of lepra bacilli from the perforating ulcers of nerve cases, and not so many in the nasal discharges. In a series of 700 cases recorded by ROGERS and MUIR, the type of infective case was recorded in 113. and of these 107., or 94.7%. were nodular.

2. During the stage of febrile reaction, infectivity increases considerably. Especially if it is prolonged and severe, the febrile or 'lepra' reaction which occurs frequently in the course of the disease, leads to numerous bacilli being liberated in the discharges.

3. Variations in susceptibility with age modify the transmission of leprosy. Infants, young children, adolescents and young adults are especially susceptible, while over the age of 30. the liability rapidly decreases. No age period is entirely exempt, but the danger to young folk is much more marked. Early infections are very frequent when there are lepers living in the same houses as healthy

children. In Groot-Chatillon, as reported by P. H. J. LAMPE, (127) even when the children of leprous parents were removed at nine months, the incidence of infection was so great that he is convinced that the only safe rule is to remove such children at birth.

4. The frequency with which infection is derived from a blood relation is striking. The explanation is that many infections are those from parents and between brothers and sisters or cousins, which are likely during the very susceptible first two decades of life.

5. Conjugal or house infections occur in from 2.5%. of persons exposed to infection. Conjugal exposure to infection will be mainly during the much less susceptible later decades of life. Mc.COY and GOODHUE (129) found also in Hawaii that house infections were twice as frequent in females as males, because women spend more time at home on domestic duties in close association with any lepers in the house.

6. Infection follows the degree of contact and closeness of human intercourse. It is well recognised in India that the main town in any area forms the chief focus of leprosy, and next the villages around it are affected, beyond which lines of location of the disease branch in various directions with hardly a single instance of leper villages not forming either a focus or part of a line or group. This points to the transmission of leprosy by human intercourse.

7. The degree of infectivity is low, compared with most communicable diseases, as from 2.5%. only of persons exposed to infection, either by conjugal relationships with lepers or living in the same house with them, contract the disease.

8. Transmission by sexual intercourse is possible. While it cannot be taken as proved that the infection of leprosy is actually conveyed by the sexual act itself, it is likely that such may be the case occasionally. Leprosy must sometimes be contracted either directly or indirectly by cohabitation of the sexes, perhaps on a single occasion only, the close contact involved being very favourable for the transmission of the leprosy bacillus: especially is this the case if nasal discharge of the bacilli takes place from the leprous partner. It may be that the greater incidence of leprosy on the male sex may be due to their greater promiscuity in sexual relationships. Further L. GLÜCK, 1900, (130) has noticed leprosy lesions on the glans and prepuce as well as the vulva and anus, and that semen may contain leprosy bacilli: direct veneral contagion is, therefore, possible. ROBILIN, 1901, (131) bears this out.

9. Room and bed infections are common. ROGERS and MUIR show that sleeping in the same room and bed with lepers is a fertile source of leprosy infection. Wearing the same clothes comes under the same category. Such intimate personal contacts are frequent in the tropical countries, because of poverty and low standards of living.

The case of J. M. H. MacLEOD, 1925,[132] is an interesting British case of bed infection:-

> A boy, aged 15, was admitted to the St. Giles's Home for British Lepers in 1920, suffering from cutaneous leprosy. He was born in Lancashire and had never been out of England. His parents were healthy and came from British Guiana, bringing an elder son, who had nodular leprosy. The boy was born a fortnight after their arrival in England. During his childhood he had been in close association with his infected brother, with whom he had slept for five years, and from whom he contracted the disease.

10. Infected household servants are transmitters of leprosy. The opportunities of servants for spreading infection are similar to those of relations and intimates in a family. In tropical countries the servants are usually of low class and by their origins and personal habits likely to have a heavy incidence of leprosy. In India, outside the Goanese and Mahommedan servants of superior class, the great majority of servants of Europeans are the Untouchables, (for in Hindu ideas the European himself is an Untouchable). It is precisely the Untouchable who has the heaviest incidence of leprosy in most parts of India.

11. Infection of attendants on lepers is not common, but cases have been recorded. The reason is that standards if cleanliness and hygiene are much higher in such a class of people, and there is evidence that in cases of leprosy which have occurred among attendants, their proper standard of cleanliness and care have been departed from.

12. Close contact with lepers outside the home is also dangerous. Cases of European children with native playmates who were lepers, and of leprosy resulting from visits to native houses were leprosy existed, have been recorded. Usually close and prolonged proximity to one or more lepers is necessary for infection, but it must not be denied that casual contact is sometimes sufficient, as sleeping one night on a journey in a native hut.

13. Infection of children from wet-nurses is recorded. The infection here is probably one of contact, rather than specific secretions carrying the bacilli.

14. Infection by inoculation through the skin is considered possible by friction against bony prominences such as the temporal ridges, buttocks, etc., from clothing, bedding and sleeping mats infected with the discharges of lepers. Transmission by arm to arm vaccination was demonstrated by ARNING in 1885, in Hawaii, though possibly the occurrence of lepra reaction in lepers after vaccination means that some of the reported cases were in fact lepra reaction in unrecognised lepers. Transmission through the nasal or buccal mucous membranes, through minute abrasions or losses of surface epithelium, is considered most likely by many, but as yet no conclusive proof is forthcoming. The high frequency with which the bacilli of leprosy have been found in the nasal secretions of lepers, particularly the nodular cases, has

given rise to the idea. The frequency of nerve leprosy of the lower extremities in bare-footed races again suggests the entry of the bacillus through cracks, fissures, abrasions and ulcerations of the skin of the lower limbs.

Inoculation through wounds of the skin in various experiments have not been successful, with the doubtful exception of ARNING'S case of the convict in Hawaii, the result being vitiated by the presence of leprosy in the subject's two near relatives. Accidental infections are in a different case, as several have been met with. The most recent is the case of E. MARCHOUX, 1934.(119)

> In December, 1922, MARCHOUX removed a leprotic nodule from the arm of a patient of Professor Jeanselme, in Paris. The needle used in closing the wound pricked an assistant, making a slight superficial wound on the outer surface of the right middle finger. The needle puncture was squeezed to force a few drops of blood from it, iodine was applied, and finally the thermo-cautery. Nothing further was thought of the matter, especially in view of the negative experiments to inoculate leprosy, and of a more serious accident which happened to MARCHOUX long ago without consequences of note. However, in March 1932, the assistant, now a distinguished physician, reported that two years previously he had had a suppurative onyxitis with loss of nails of the middle and index fingers. Eighteen months after the onyxitis, he noticed that a cigarette burn was painless, both fingers being insensitive to pain, and also on examination, to heat and cold. A small piece of tissue removed from a discoloured area of skin between the index and thumb showed in a smear a large number of acid-fast bacilli: sections confirmed the diagnosis of leprosy. The patient died shortly after from a staphylococcal septicaemia.

The occurrence of accidental inoculation of man follow- an infecting injury of the skin is, therefore, possible.

MARCHOUX thinks that the site of the wound in the dermis gave the greatest chance of infecting. He points out the slow evolution of the infection, and that the first manifestation was in the form of disturbances of sensory perception.

15. Transmission of leprosy by insects is not proved. ROGERS and MUIR conclude that the evidence regarding the powers of biting insects to transmit leprosy is not sufficient to incriminate them definitely. The parasite of itch, ticks, bugs, fleas and mosquitoes have at various times been suspected, but no proof is yet available. House-flies are suspect, with more grounds for believing that they play a part in the propagation of leprosy. House-flies may absorb numerous lepra bacilli from infected ulcers, which remain unchanged in their intestines: by depositing living bacilli in their faeces and their 'vomit drop' on mucous surfaces, such as the nasal orifice, and cutaneous wounds and abrasions of healthy persons living in the immediate neighbourhood of lepers with open lesions, they may convey the disease.

STUDY OF THE POONA CASES.

During the years 1929 to 1933, a series of 190 cases of leprosy were studied by the author in Poona. These do not represent all the cases seen during that time, but only those for which adequate time could be found to secure the necessary data and make investigations. The dual nature of the work in Poona, namely control of a general hospital and of a leper asylum, made it necessary to limit the number of cases of leprosy for study, but time and opportunity have been the only limits; no artificial selection of the cases has been exercised. The work has had to be done practically single-handed, including laboratory work, as it was very desirable to have confidence in the results obtained. In taking case histories, the plan has been followed of taking them personally in every case, the presence of DR. V. S. GAEKWAD at Khondwa and of DR. D. K. GOREY at Poona being requested at as may examinations as possible, in order to check up the vernacular conversations, they being natives of the region. The principle of interrogating and conversing with each leper in his or her own vernacular was sought to be followed, because of the greater gain in accuracy of elicited facts that such a plan affords. The languages known to the author for the purpose were Marathi, Hindustani and of course English. Marathi is the most important of these, for the majority of our patients speak that language, and the other two were necessary in a few cases only. Some difficulty, however,

was experienced in getting the intimate information needed in the case of 'foreigners,' such as Tamil and Kanarese and Telegu immigrants to the District. This problem was solved as occasion offered by enlisting the aid of the linguistic knowledge of visiting doctors from these language areas. For instance, certain Tamil patients were as a closed book to us until the visit of the author's sister, DR. F. R. INNES of Vellore, North Arcot, enabled us ti have several 'field days' in eliciting the required facts. On the whole, it was possible to secure contact with every leper patient in his or her own vernacular.

Form of the Records. Special case records have been kept, in form following the suggestions of the LEONARD WOOD MEMORIAL CONFERENCE ON LEPROSY, 1931, where a useful outline of data to be obtained is given.(133)

First Grouping of the Cases. The cases are firstly divisible into two main groups. They are, 1. Outpatient Lepers attending the Wadia Hospital bi-weekly clinic, cases Nos.1 to 71 in the series: and 2. Leper Inmates of the Khondwa Leper Asylum, cases Nos.72 to 190. For some purposes it will be necessary to consider these two groups separately.

Castes of the Lepers. A definition of caste has already been given (Page 9). Caste is still more than a label in India, and when we know a person's caste, we know something definite about him. Taking all the cases, the castes represented and the number of each caste afflicted with leprosy in our series are given in the Table.

TABLE XIII.

CASTE INCIDENCE OF LEPROSY IN 190 CASES.

A.	Hindu Castes.		
	Brahmin	8)	
	Prabhu	2)	Incidence in high castes, 11.
	Rajput.	1)	
	Marwadi	3)	
	Sonar	2)	
	Wani, Lingayat	6)	
	Jain	1)	
	Gujar	1)	
	Padamsali	1)	
	Shimpi	2)	
	Teli	4)	Incidence in middle castes, 30.
	Sutar	1)	
	Koshti	3)	
	Mali	3)	
	Naidu, Tamil	1)	
	Telegu	1)	
	Dhobi	1)	
	Burud	1)	
	Goundi	1)	
	Nair, Tamil	2)	
	Kalal	1)	
	Koli	2)	Incidence in lower castes, 68.
	Devadasi	1)	
	Ramoshi	1)	
	Dhangar	2)	
	Maratha, Kunbi	55)	
	Kamati	2)	
B.	Hindu Outcastes.		
	Chambar	2)	
	Gosavi, mendicant	1)	
	Wadari, mendicant	1)	Incidence in outcastes, 30.
	Madrassi, outcaste	2)	
	Mahar	19)	
	Mang	5)	
C.	Communities outside the Hindu Caste System.		
	Beni-Israël	1)	
	Pathan	2)	
	Mahommedan	13)	Incidence in outside communities 51.
	Anglo-Indian	1)	
	Indian-Christian	34)	

The percentage incidence of leprosy in the above groups is:-

in the high castes	5.78%.
in the middle castes	15.78%.
in the lower castes	35.78%.
in outcastes	15.78%.
in outside communities	26.84%.

The Indian Christians introduce an element of difficulty in that they are a new community drawn from various Hindu castes and outcastes; and so recently have many of them come out of Hinduism that for our purposes they should be classified under their original castes. Fortunately it has been possible to obtain information as to the origins of the Christians with whom we have to deal. This is given below:-

TABLE XVI.

ORIGINS OF THE 34 INDIAN CHRISTIAN LEPERS.

Case No.	Name of Patient	Origin	Serial Numbering of each type.
85.	Manorama Bhutilal	Wani Caste	1.
1.	Dinkar Sane	Maratha Caste	1.
83.	Vitha Dhondi	Maratha Caste	2.
186.	Thakubai Sambu	Koli Caste	1.
5.	Samuel Tivari	Outcaste	1.
25.	Marybai Tivari	Outcaste	2.
29.	Gopin Augustin	Outcaste	3.
58.	Markus Kondiba	Outcaste	4.
64.	Benjamin Bhimaji	Outcaste	5.
67.	Chandanbai Kumu	Outcaste	6.
79.	Sonubai Patne	Outcaste	7.
86.	Mahadu Bala	Outcaste	8.
93.	Abel Ibasha	Outcaste	9.
95.	Lazarus Yellappa	Outcaste	10.
98.	Bhau Arjun	Outcaste	11.
112.	Pau Thomas	Outcaste	12.
114.	Malan Nana	Outcaste	13.
117.	Kamal Laxman	Outcaste	14.
119.	Mary Abel	Outcaste	15.
120.	Karonhabai	Outcaste	16.
123.	Joseph Robin	Outcaste	17.

TABLE XVI (cont)

ORIGINS OF THE 34 INDIAN CHRISTIAN LEPERS.

Case No.	Name of Patient	Origin	Serial Numbering of each type.
136.	Bhagaji Bala	Outcaste.	18.
139.	Rama Tula	Outcaste	19.
142.	Mahadu Devrao	Outcaste	20.
147.	Joseph Mathew	Outcaste	21.
153.	Tanhibai Shankar	Outcaste	22.
154.	Avda Sayaji	Outcaste	23.
157.	Tripama	Outcaste	24.
159.	Thomas Nathaniel	Outcaste	25.
177.	Taibai Ranzana	Outcaste	26.
180.	Karona Samuel	Outcaste	27.
183.	Rosemary	Outcaste	28.
185.	Bhimabai Laxman	Outcaste.	29.

It will be seen that only four of the Christians had Caste origins, and the great majority, no less than 29, had origin in outcaste communities. It is necessary, therefore, to correct some of the figures in TABLE I. The middle castes gain one, the lower castes gain four, and the outcastes gain twenty-nine.

The revised figures for leprosy incidence will now be,

in the high castes,	11 or 5.78%.
in the middle castes,	31 or 16.31%.
in the lower castes	72 or 37.89%.
in the outcastes	59 or 31.05%.
in the outside communities	17 or 8.94%.

The most striking fact that emerges from these figures is that the lower castes and outcastes have the heaviest incidence of leprosy, their figures together being 68.94%. of the total; whereas the high middle castes together account for 22.10%. only of the total incidence.

Explanatory Details about the Castes and Communities.

The Brahmin is the caste which stands at the very summit of the Hindu social and religious system, the guardian of ancient knowledge, 'twice-born,' gods upon earth. The priesthood is the prescriptive occupation of the Brahmin, but in these days he is more often a teacher, a clerk, a public servant under the Government, or even a cook and servant to his own caste. His characteristics are intellectualism, intrigue and genteel indigence. Brahmins are not many as compared with other castes, but as it happens, Poona is one of their strongholds, and they are comparatively numerous.

The Prabhu is an offshoot or subcaste of the Brahmins, and for practical purposes may be regarded as Brahmin.

The Rajput is one of the Kshatriya or Warrior castes, which are next in estimation to the Brahmins, and have provided most of the rulers and fighters of India. They are, even in their hunbler ranks, men of action and impressive bearing. The Rajput is a caste not indigenous to Poona District, as its home is in Rajputana. The name means 'descendant of kings.'

The Marwadi or Marwari is also an incomer from Rajputana. He is usually rich, and money-lending and shopkeeping his chief occupations. He is a shrewd and hard-working business man.

The Sonar is the goldsmith caste, with much business ability, which expresses itself in money-lending, banking,

and jewel-trading.

The Wani caste may be broadly interpereted as the grocer caste, that is, the smaller shopkeepers and traders. Of the four types mentioned, the Lingayat is the indigenous one. The Jain, Gujar and Padamsali are successful incomers from other parts of India. The word 'Wani' is the Marathi equivalent of the more generally known word 'Bania.'

The Shimpi is the tailor caste.

The Teli is the caste of oil-sellers and makers.

The Sutar is the carpenter and joiner caste.

The Koshti is the old indigenous weaver caste. Since the decline of weaving, many of them are to be found in other occupations, as in the Bombay mills, motor-driving, railways, etc.

The Mali is the caste of gardeners; as well as private gardens of the wealthy, they are employed in Botanical Gardens, market gardens, and fruit plantations, and sometimes even in general farm labour.

The Naiduis a south country land-owning class from the Tamil and Telegu countries.

The Dhobi is the familiar washerman. His services are in such demand that it is unusual for him to be found in another occupation than washing.

The Burud is the caste of basket-weavers.

The Goundi is the caste of stone-workers, builders and masons.

The Tamil Nair is a south country agricultural caste.

The Kalal is the indigenous caste of liquor-sellers and

toddy-sellers.

The Kolis the fisherman caste. It is most numerous on the coast, but there are genuine inland Koli castes in Poona District. They are aboriginal in origin.

The Devadasi is the temple-dancer and prostitute.

The Ramoshi is the ancient caste of watchmen. They are not far removed from a criminal tribe, and are said to have originally received their job as watchmen in the hope that they would keep other thieves away from the village or private property they were paid to watch.

The Dhangar is the Maratha country shepherd caste. They are very black in colour from their hereditary open-air life, and are regarded with affectionate amusement by the other castes, because of their great simplicity and good-heartedness.

The Maratha or Kunbi caste, with the sub-caste the Kamati, comprises the great number of farming and agricultural people in the Maratha country. Their origin is believed to be partly aboriginal. They may be described in a word as a hard-working and manly people, struggling for the most part against the hardest of conditions and poverty. They are to be found in other occupations often, as in the Army, Police, Railways, trade and even clerking and teaching. They form the most numerous caste in the population.

The Chambar is the outcaste group which deals with hides and leather, and is held in abomination because of it by Hindus.

The Gosavi and the Wadari are wandering mendicant outcaste tribes of undoubted aboriginal origins, and very wild and nomadic habits. They are comparable with the gypsies of European countries.

The Madrassi outcastes among the lepers were Tamil mendicants of the Wadari type.

The Mahar is the numerous local outcaste group, believed to be the purely aboriginal inhabitant of these regions. His position has long been one of the lowest servitude and in return for menial village services, he is allowed to live, and to take the carcases of animals dead of disease and unnatural causes for his food. His tribe often provides the village prostitutes, and the morals and civilisation of the tribe are practically non-existent. It is from this tribe of outcastes that many Christians come, and their legacy of depression and disease is probably reflected in the high incidence of leprosy in the Indian Christians. No doubt there is much more leprosy among the Mahars than is shown in the figures. The position is that when a leper is a Mahar, he is too low to care, but when he becomes a Christian, he comes for treatment.

The Mang is the last and the lowest on the list of outcastes. All that applies to the Mahar applies to the Mang, but if possible, he is even lower in the scale. The Mahar, bad as he is, looks down on the Mang. The very name 'Mang' is an expression of disgust and contempt rather than a name. It is worth repeating that the Mahars and the

Mangs are usually the only type of household servant that many Europeans can get.

The Beni-Israel are, as their name suggests, not a Hindu caste, but the Children of Israel. They are descendants of Hebrews long settled in India, mostly around the coastal towns, for over 500 years. They had almost forgotten that they were Jews, keeping only certain rites, and thought of themselves as a caste of Hinduism; but DR. WILSON of Bombay in 1830, discovered them and restored their full racial knowledge. The orthodox Jews still look askance at them, but to a Gentile eye they are a fine type of the ancient race, and if they were to be put again among the Hindus, by their habits, physique and abilities, they would rank among the high castes.

The Pathan is a Mahommedan belonging to across the North-west Frontier of India, believed to be of Semitic origin also. He is found so far south in pursuit of his great business of money-lending and battening on the Indian peasant.

The Mahommedans of Poona District are not numerous, and are of very mixed Hindu origin, being the result of inter-marriage and conversions by the early Mahommedan conquerers. In their standard of living and hygiene they are low, and are not comparable with their co-religionists in other Mahommedan countries.

The Anglo-Indian is the Eurasian community resulting from the admixture of Europeans and Indians. Their chief

occupation is to be found in the railways, and their standard of living is generally intermediate between that of the Europeans in India and the Indians. They are rather nervous and neurotic as a people. Many of them are very poor.

The Indian Christians have been described previously. The chief points about them are their low caste origins, and their state of transition from Hindu to entirely new social customs.

POPULATION BY CASTES AND COMMUNITIES.

From the statistical tables of A. H. DRACUP and H. T. SORLEY, 1931,[134] the data have been collected of the population of Poona District by chief communities, for the purpose of a comparison with the leprosy incidence in our series.

TABLE XV.

POPULATION BY COMMUNITY IN POONA DISTRICT WITH PROPORTION OF LEPERS.

Community	No. of persons.	No. of Lepers.	Percentage of Lepers.
Brahmins.	69,732.	8.	0.0101%.
Other Hindu Castes	868,564.	98.	0.0101%.
Outcastes	140,167.	50.	0.0350%.
Mahommedans	54,997.	15.	0.0270%.
Indian Christian	19,206.	34.	0.1700%.
Europeans	3,158.	0.	0.0000%
Parsees	3,473.	0.	0.0000%.
Anglo-Indians.	2,655.	1.	0.0370%.

The figures for the number of lepers in each community

can as yet represent only a partial statement of the true proportion. The highest proportion in the Table is that of the Indian Christians, indicating most probably that they come more readily than most people for treatment, and the true incidence of leprosy in their midst may not be very much greater than the figures show. The outcastes and Mahommedans also have a high proportion of lepers in the Table, and as they are ignorant peoples, it is probable that they have a higher incidence in fact, higher than other communities. The Brahmins are a very educated people, and a cleanly, and it seems that they have a low incidence in fact, as well as in the Table. Other Hindu castes have in the Table much the same proportion as the Brahmins, but it is impossible to believe that there are only 98. lepers among 868,564 of them: it is in this group that most of the hidden lepers must be looked for. The communities with the highest standard of living, namely the Europeans and Parsees, have no lepers at all. The Anglo- Indian community is such a small one, that just by having one case, the percentage of leprosy becomes high in comparison with the other communities.

TABLE XVI

OCCUPATIONS OF THE LEPERS.

Occupations.		Out-patients.	Inmates	Total	
No occupation,	adults	1.	1.	2.	
	minors.	5.	10.	15.	17.
Agriculture,	field or farm labour	8.	15.	23.	
	farmers (small-holding)	3.	3.	6.	
	minor in field work	1.	0.	1.	30.
Shopkeeping,	potatoes	1.	0.	1.	
	hardware	1.	1.	2.	
	oil	2.	3.	5.	
	cloth	3.	0.	3.	
	sweets	1.	1.	2.	
	groceries	1.	1.	2.	
	clothing	1.	0.	1.	
	tailoring	2.	2.	4.	
	grain	0.	2.	2.	
	liquor	0.	1.	1.	
	restaurant	0.	3.	3.	26.
Transport,	Railway carriage cleaner	1.	0.	1.	
	Railway ticket collector	1.	0.	1.	
	Railway public luggage coolie.	3.	0.	3.	
	Railway Company's coolie.	1.	0.	1.	
	Motor bus drivers	2.	1.	3.	
	'tanga' (public horse vehicle) drivers	1.	1.	2.	
	Private motor driver	1.	0.	1.	12.
Education,	Teacher in Primary School.	1.	1.	2.	
	Pupils in Primary School.	2.	0.	2.	
	Students at College	4.	5.	9.	13.
Domestic,	Housewives	3.	16.	19.	
	Domestic servants	0.	7.	7.	26.

TABLE XVI (cont.)

Occupations.	Out-patients.	Inmates.	Total.	
Coolies, general	5.	3.	8.	
road	2.	0.	2	10.
Millworkers.	4.	11.	15.	
Clerks	1.	2.	3.	
Priests.	0.	2.	2.	
Sempstresses.	1.	3.	4.	
Stonemasons.	0.	2.	2.	
Shepherds.	0.	3.	3.	
Carpenters.	2.	0.	2.	
Prostitutes.	0.	2.	2.	
Temple-dancer.	0.	1.	1.	
Sepoys, police	0.	1.	1.	
military	0.	1.	1.	36.
Mendicants	0.	4.	4.	
Fishermen	0.	2.	2.	
Butcher	1.	0.	1.	
Gardener in private house	1.	0.	1.	
Leatherworker.	1.	0.	1.	
Itinerant shoemaker	1.	0.	1.	
Biblewoman	1.	0.	1.	
Racecourse syce	1.	0.	1.	
Saltworker	0.	1.	1.	
Basket-worker.	0.	1.	1.	
Potter	0.	1.	1.	
Dispenser in chemists's	0.	1.	1.	
Village Watchman	0.	1.	1.	
Washerman	0.	1.	1.	
Weaver (Nagpuri)	0.	1.	1.	
Milkwoman.	0.		1.	
	71.	119	190.	

Merely by reading the above Table of occupations of the lepers it becomes borne in upon one how widespread the disease must be amongst the people. Many occupations are unrepresented, but the variety and sweep of those which are represented in a series of 190 cases leave little doubt but that few aspects of life in Poona District are untouched by leprosy.

Leprosy is evidently a disease closely bound up with human intercourse, as most of the occupations are those which have the most frequent and prolonged contact with numbers of people, or momentary contacts often repeated. Moreover it must be remembered that infection to bring about leprosy does not necessarily demand prolonged and intimate contact, as G. W. McCOY, 1914,[135] has shown. If a leper is in a position where he comes into momentary contact with great numbers of people, it is quite possible that he may convey infection to someone.

Another point brought out by a general survey of the Table is that the list of occupations of the outpatients shows the civic value of an outpatient clinic. It is reaching and treating with success, and so rendering innocuous, a body of people entrenched in fine positions for the spread of leprosy to others; it is doing this to people who would otherwise never be heard of as lepers, under present conditions of leprosy work in Poona. This aspect of the subject, as exemplified by the Wadia Hospital Clinic, so impressed COLONEL CANDY, the Civil Surgeon in Poona, that he established an outpatient clinic for lepers at the Sassoon Hospital in 1933.

Whether occupation <u>per se</u> predisposes to leprosy is a matter difficult to state categorically. It will be helpful to take the occupations in order as they stand in the Table, and to discuss them from two points of view,

1. Whether a person in any given occupation is by that

fact in danger of acquiring leprosy: and:-

2. Whether a leper in any given occupation is by that fact endowed with special opportunities of spreading leprosy.

(I) The condition of no occupation must first be dealt with. In the case of the two adults in the Table, their state of idleness depended partly on the fact that they were supported by doting parents in that state, and partly that work is hard to get. The depression in health and morale that unemployment leads to is a well-known fact of modern experience, and is well attested by a survey of 1,000. families unemployed for four years in the United States: (136) higher sickness rates, particularly in the case of the more serious illnesses that cause inability to work or confine the patient to bed, were obtained. In both of our leper cases, there was unmistakably an element of laziness, which has been already pointed out by E. MUIR, (59) as a definite predisposing cause of leprosy. For these reasons it seems true to say that 'no occupation' is a state predisposing to the disease. In the case of the minors the consideration of being employed or not employed of course does not concern us as it does in adults. Whether a leper in a state of no occupation is possessed with special opportunities of spreading the disease may be answered on the whole in the affirmative. Things may not be so bad if he stays at home in retirement, within a circle of human contacts which is limited. If, however, as these

two cases did, he indulges in promiscuous immoralities, and wanders to other villages and on pilgrimages to amuse himself in his idleness, he becomes a danger.

(II) Agricultural pursuits impress one as tending rather to raise a person's resistance to leprosy, considered per se. So agriculture should be at its best, with healthy open-air work and good food, and an adequate return from the soil. Only conditioned by poverty, poor soil, overcrowding and defective diet, will farming predispose to a disease like leprosy. Such is the state of affairs only too often in the District, hence the occurence of 30 agricultural lepers in our list of 190.

Again, with agriculture at its best, a leper in such pursuits would be benefitted and have little opportunity of passing on the disease. With its unhappy dyshygienic concomitants as found in fact, it does give him opportunities. If only the conditions of this important and predominating occupation of agriculture could be improved, we should strike a severe blow at leprosy in India. There are 102,454,147 persons engaged in agriculture in India, according to the 1931 Census.

(III) To be in the occupation of shopkeeping seems definitely to expose a person to the danger of acquiring leprosy. There is the rather surprising total of 26 shopkeepers on our list. The outstanding features of shopkeeping in India are long hours of attendance, sedentary life, long exposure to roadside dust and flies,

frequent contacts with many people among whom there may be lepers. Contrariwise, a leper who is a shopkeeper has undoubtedly special opportunities of spreading the disease. A factor of importance is that beggars, including leper beggars, are in the habit of making rounds of the shopkeepers to gain alms. Shops, more than private houses, form their beat by preference.

Among the shops represented, tailoring shops, liquor shops, and restaurants strike one as the ones likely to be the most fertile sources of infection if the shopkeeper were a leper.

(IV) Transport occupations will involve a person in danger, chiefly because of numerous contacts with the public, and on the other hand, if a leper, the public is in danger from him. One is not surprised at a railway carriage cleaner being the victim of leprosy; in the author's opinion, this is a good example of an occupation which in itself puts the person engaged in it under threat of leprosy. That lepers travel on Indian trains is a notorius fact, and whether they do so having paid their fare or not, having travelled under the seat or on it, or for a long distance or a short one, they can leave their discharges, rags, and bacilli behind them. The lot of a cleaner of third class carriages in India is not one to be envied. Poona Station is a big railway centre, from which the carriages go to big centres of population like Bombay, and the carriages are used by great numbers of people. The lower classes on

Indian trains are usually very crowded, and lepers are never evicted from carriages, and seldom asked to pay the fare, which encourages them to travel; lepers are also restless people and fond of wandering. To reverse the picture, a leper carriage cleaner may leave infection behind him in a carriage for healthy travellers to meet.

It is not pleasant to think that leper luggage coolies may lift one's baggage on arriving or leaving by train in Poona. Probably the danger is not very great, but it is a state of affairs which should be guarded against. The Company's coolies do not come much into contact with the public, and are engaged chiefly in handling merchandise.

Bus drivers in Poona District are more predisposed to sudden death than leprosy, because of their common habit of reckless driving at great speeds over the bad roads, but if lepers, they do come much into contact with the general public.

The tanga driver is the driver of the common public horse vehicle in Poona. He sits in the front seat, and the passengers sit in the rear seat facing backwards. It is a cheap form of conveyance and much used. If the public is to have some of its tanga drivers lepers, it is fortunate that they sit in front, but there is little space between driver and passenger, even if they are back to back. Tanga drivers have the habit of curling up in the back seat of the passenger to sleep, when a slack hour comes, and may thus convey the disease.

The private motor driver gets a good wage and should not be in special danger of leprosy. Unfortunately, as in the case in the Table, loose living often spoils the picture. This case was interesting in that after his employer knew his chauffeur was a leper, he still kept him in active employment, in daily contact with the children, and it was some time before the driver was released from work. Here were special chances of conveying infection to susceptible young folk.

(V) Education does not necessarily in itself run a person into the danger of leprosy, but does so indeed because of the bringing into daily contact perhaps an infective teacher and susceptible youth. Thus, we have a leper primary school teacher and leper pupils in a primary school, not in the same school, though they might well have been. College students are at a later age, amd may sustain their infection at a primary school, or their homes, or elsewhere. Nevertheless the danger is there of scholastic infection. It is at least suggestive that two of the college students attended a college where there had been in their time, a leper, the Professor of English. This man is not in this series of cases, but was attended by the author, who succeeded in getting him to leave his work, but his previous chances of spreading infection must have been enormous. It seems less likely that a teacher will get the disease from young lepers in his classes, but it is possible.

(VI) Domestic occupations fall into two classes, housewives, whose entire world is the home, and domestic servants of both sexes in the houses of Europeans and the richer Indians. One would not expect women whose chief interest is the home to run much danger of acquiring leprosy, because of their limited circle of contacts, and the fact that there are 19 examples of housewives in the series is an arresting one. The explanation was found to be that in their case a household infection already existed. A home life is no protection from the danger of leprosy if there is a leper in it. Conversely, a housewife who is a case of leprosy has special opportunity of transmitting the disease. Domestic servants are not especially placed in danger of leprosy and there are factors which militate against it, as the better housing, food and living conditions generally that they obtain. Overwork is certainly not a detrimental factor in their case. On the other hand, if a servant is already leprous when entering a household, and especially if there are children in the home, great is the risk of the disease being spread. In the same case is the one example of a gardener in a private home; by his occupation he is rather fortified against leprosy, but if he is much in contact with children he can spread it if he is a leper.

(VII) Not much can be brought home to road or general coolies. Their predispositions to leprosy and their chances of spreading it depend rather on their habits than their occupation. In the case of millworkers, however,

the occupation does run them into danger. Mill labour is new to India, and the operatives are drawn from people of other types, as agriculturalists. They are thus in a state of transition and adaptation to a new environment, and vulnerable to the attacks of disease. They knew overcrowding in their villages, but the Bombay chawls are unbelieveably worse. They had promiscuous and unhygienic ways of living in their villages, but in Bombay they learn worse. (In the venereal diseases clinic in the Wadia Hospital, Bombay, ~~is~~ was given as the place where the syphilis or gonorrhoea was acquired in a considerable number of the cases). Further, millworkers come into contact with a vastly greater number of people than they would in their villages, and possibly the change of climate to the Deccan villager may play some part in lowering health and resistance: this would not apply so much to the Sholapur Mills, for Sholapur is also in the Deccan, but Bombay to which most of them go, is possessed of a very trying climate to a Deccani. Finally, even with their steady wages they may not necessarily purchase a better diet. Adulteration of food in Bombay is scandalously common, and food is dearer, and the result is that their diet is perhaps worse than in their native villages. In the course of her researches on tropical macrocytic anaemia, LUCY WILLS, 1934,(138) found from a dietetic survey in the classes which include the millworkers "a deplorable state of multiple deficiencies:" all the diets were particularly low in Vitamins A. and C. All these factors contribute to make millworkers to be in

a danger of leprosy. Conversely, a leper employed as a millworker is in a strategic position for perpetuating the disease.

(VIII) In the case of the other occupations represented, the clerks, sempstresses, stonemasons, shepherds, carpenters, fishermen, leatherworker, salt-worker, basket-worker, potter, village watchman and weaver did not appear to have any discoverable relation to leprosy. In the case of the priests, remembering the pilgrimage habit of the Indians, and the frequenting of shrines by lepers, it was not surprising to find that each of the two priests came from a famous shrine in Poona District. Whether they obtained their infection from pilgrims, or obtained it otherwise and gave infection to pilgrims, is not easy to say.

The one temple dancer and the two prostitutes are representatives of occupations dangerous as to sustaining and to spreading the infection of leprosy, as they imply extreme promiscuity.

The sepoys in so far as they have many contacts with the public, have by their occupation some relation to leprosy, and the police are often called upon to arrest leper vagrants and escort them to the Leper Asylum. Apart from that, their disciplined life, adequate pay, and standard of hygiene and living must tend to couteract leprosy infection.

Mendicants are as a class leprous in India, and are uncontrolled wandering foci of leprosy infection. Thus in Calcutta, it is estimated that there are about 4,000 beggars,

and of these 224 are known to be lepers.(139) In Poona, 14 leper beggars have been counted, in a total number of beggars of about 150. The itinerant shoemaker as a leper is almost in the same case as the mendicant as to chances of spread of infection, and he also may visit the house of Europeans. A leper biblewoman, racecourse syce, butcher, chemist's dispenser, washerman, and milkwoman are public dangers in so far as they handle foods or clothes, and have many human contacts, but there seems nothing in their occupations which predispose to leprosy.

SUMMARY OF THE QUESTION OF OCCUPATION.

Apparently, among the occupations represented, shop-keeping, transport, schooling, domestic occupations, mill labour, temple priests, temple attendants, prostitutes and mendicants, have a definite relation to leprosy, by virtue of the danger of acquiring, and the opportunities of spreading the disease.

Sex Incidence.

Of the 190 lepers under review, 125 were males and 65 females, the males being almost twice as many as the females. This is in line with experience in the rest of India, and in most countries of the world, as described by J. LOWE,(70 & 71) and has been discussed on Page 73.

In Poona the cause of it is chiefly environmental, for men are more exposed than women to infection and to conditions predisposing to infection, as a study of the occupations of the lepers shows. A partial cause of the figures may be a hidden incidence as regards women. By the demand of social custom, women are more retiring in India than men,

and they are less wont to get attention paid to their illnesses. The true leprosy incidence in women may easily be greater than now appears.. If this be so, any future rise in the status of women in India should be followed by a rise in their leprosy incidence. Inherent physiological differences in the sexes as a cause of the disparity in incidence have not been revealed by the study of these 120 cases.

As regards sex incidence in childhood, the increased proportion of males is at variance with the conclusion of J. LOWE, that in childhood the incidence on the sexes is more equal. Our series is, however, small, as there were only 20 children who were 15 years of age and under when first seen. The details are given in the Table below:-

TABLE XVI. Sex Incidence of Leprosy in 20 Children of 15 years of age and under.

Case No.	Name of Child.	Sex.	Age (in years)
5.	Samuel Tivari.	Male	10.
15.	Dathu Mandku.	Male	15.
24.	Shankar Raghu.	Male	10.
29.	Gopin Augustin.	Male	10.
36.	Ananda Tulsiram.	Male	10.
43.	Baba Sayed Kairubhi.	Male	14.
48.	Raghu Dada.	Male	13.
50.	Khatoor Sayeddaulad.	Female	12.
56.	Babu Ganpat.	Male	13.
62.	Laxman Sitaram.	Male	7.
77.	Anusayabai Hari.	Female	11.
90.	Husain Imam.	Male	15.
96.	Babu Kondiba.	Male	11.
99.	Gaffur Chotu	Male	15.
140.	Damu Honaji.	Male	12.
142.	Mahadu Devrao.	Male	7.
146.	Tatyoba Bhau.	Male	12.
149.	Narayan Jairam.	Male	15.
170.	Bali Chandra.	Female	10.
172.	Ragunath Ramji.	Male	12.

Of 20 children, only 3. or 15%. are females. It is noteworthy that with the exception of two, the children are at about the age of puberty, and in the case of the three female children, menstruation had not begun but was not far away, judging by the onset of other developmental changes. It will be remembered that menstruation may begin at 12 years of age in Indian girls.

J. N. RODIGUEZ, 1926,[140] found that the incidence of leprosy was practically identical in both sexes up to the eighth year, after which it became 28 to 13 in the 8-12 year group; between 10 and 16 years the influences of puberty operate, and as these are naturally more pronounced in girls, the incidence amoung the females rises suddenly. The facts in our Table only support the influence of puberty, and the incidence on females certainly rises suddenly between the ages of 10 and 16 years, as below that age there were no females. Thus there were in the age group to the 9th year:- 2 males.
0 females.

and in the age group 10 to 16 years:- 15 males
3 females.

A complicating factor as far as India is concerned is that of the estimation in which sons are held as opposed to daughters, resulting in a tendency to give them the first attention when illness comes. In India, boys are thus more likely to be brought at the earliest signs of leprosy, and in our series there are two males at 7 years of age, and no females. This factor is a very real one, and may ex-

plain much of the disproportion between incidence on the sexes in childhood. It may not be the whole explanation, but it cannot be ignored. That beyond this, under the same conditions of exposure to infection, boys develop the disease more readily than girls, it was possible to find no evidence in these cases. In Japan, such a conclusion was come to from an investigation of families in which the parents suffered from leprosy, by Y. HAYASHI, 1927.(141)

The Form of Leprosy and Sex.

Study of the cases from the point of view of the severity of the disease in males and in females indicates that there is a tendency for leprosy to be severer in men than in women. The chief means of distinguishing severity lies in consideration as to whether the case is neural or cutaneous, the latter or nodular type being associated with the greater severity. In other countries some experience of this greater severity of leprosy in males has been reported, as by FUMIO HAYASHI, 1934,(142) who states that in Japan the disease is on the whole milder in women than in men. Also from Crete, EHLERS and CAHNHEIM, 1920,(143) reported that the nodular form was much more common among men than women, and a similar finding comes from Norway, by A. SAND, 1903; he also showed that female lepers had a longer expectation of life.(144)

Using the classification of cases of leprosy adopted by the International Conference at Manila in 1931, which

has been described on Page 61., the Table below has been constructed for convenience of considering the question of severity of leprosy in each sex:-

TABLE XVIII.

SEX AND FORM OF THE DISEASE IN 190 CASES IN POONA.

	Males. (125)		Females. (65).	
Type of the disease.	No. of cases.	Percentage of total No. of males.	No. of cases.	Percentage of total of females.
N-1.	18.	14.4%.	6.	9.23%.
N-2.	21.	16.8.	15.	23.07.
N-3.	8.	6.4.	16.	24.61.
C-1.	10.	8.0.	1.	1.53.
C-2.	14.	11.2.	6.	9.23.
C-3.	4.	3.2.	1.	1.53.
N-1,C-1.	8.	6.4.	4.	6.15.
N-2,C-1.	16.	12.4.	4.	6.15.
N-1,C-2.	10.	8.0.	1.	1.53.
N-2,C-2.	6.	4.8.	5.	7.70.
N-1,C-3.	1.	0.8.	1.	1.53.
N-2,C-3.	2.	1.6.	2.	3.07.
N-3,C-1.	3.	2.4.	1.	1.53.
N-3,C-3.	3.	2.4.	1.	1.53.
N-3,C-2.	1.	0.8.	1.	1.53.
Purely Neural	47	37.6.	37.	56.15.
Purely Cutaneous	28.	22.4.	8.	12.30.
Mixed	50.	40.0.	20.	30.76.
All cases partly or wholly cutaneous	78.	62.4.	28.	43.07.
All cases partly or wholly bad cutaneous (C-2 or C-3)	41.	32.8.	18.	27,69.

Taking the above data in three ways, in each one the greater severity of the disease in males in indicated. Thus 22%. of the males were purely cutaneous cases, whereas only 12%. of the females were such; in all cases grouped to-

gether of cutaneous type, including mixed cases with a cutaneous factor, 62%. of the males but only 43%. of the females are classified: taking all cases which have a C-2 and a C-3. factor as 'bad cutaneous,' we find 32%. of the males as against 27%. of the females are such.

SEX AND DURATION OF LIFE IN LEPERS.

There seemed no significant difference between male and female lepers in our series as regards duration of life. Of 26 cases in the age period 46 to 70 years, 14 were males and 12 females, as shown in the TABLE.

TABLE XIX.

LEPERS IN THE AGE PERIOD 46 - 70 YEARS.

Males.			Females.		
Case No.	Name	Age	Case No.	Name	Age.
175.	Daniel Abraham	64.	133.	Sarzabai	60.
27.	Shankar Tukarum	62.	129.	Gangutai	55.
181.	Vinayak Moolay	60.	188.	Muktabai	55.
124.	Sidhappa	55.	73.	Bhagubai	50.
26.	Khondilaka Tuka	55.	76.	Noorbi	50.
9.	Devji Yemaji	51.	128.	Saku Tai	50.
22.	Baduroa Phule	51.	135.	Govindma	50.
16.	Hari Ramu Thorat	50.	161.	Jayabai	50.
53.	Punamchand Jumarji	50.	184.	Rakhnabai	50.
71.	Syed Asadulla	50.	185.	Bhimabai	50.
91.	Tatyoba Yeshwant	50.	186.	Thakubai	50.
97.	Rama Shetiba	50.	187.	Sakhubai	50.
190.	Genu Hange	50.			
65.	Tukaram Kashiba	46.			

LEPROSY AND LITERACY.

The degree of education of the cases was studied, in order to ascertain any relation between it and leprosy. There were no University graduates among these lepers, so they were divisible into three classes, the highest stopping short of the graduate stage. The three classes are the illiterate, the literate and the well-educated: in the first are placed all those who could not read nor write; in the second are those who can read, write and count in their own vernacular; and in the third are those who have proficiency in English or other languages besides their own vernacular, but who have not passed beyond the secondary school stage. In the case of minor children, if there were no future possibility of any schooling, they were classed as illiterate; if they were at school and there were every possibility that schooling would be continued, they were classed as literate.

The general literacy of the people it is necessary to know for comparison. This may be given approximately from the EAST INDIA CENSUS, 1931,(145) The population of the Bombay Presidency is given as 26,398,997. and the number of literates 5 years of age and over is 2,278,044. Taking these figures for the purposes of calculation as 26,400,000. and 2,800,000., respectively, the percentage literacy of the general pooulation works out at 10.6%.

Using the same sources, the literacy of tne main communities in Bombay Presidency is calculated to be:-

Hindu Castes	10.2%. literacy.
Outcastes	2.8%. literacy.
Muslim	0.67%. literacy
Christian	34.06%. literacy.

The data concerning the lepers studied are given in the following TABLE:-

TABLE XX. LITERACY OF THE LEPERS BY CASTE AND SEX.

No. available.	Caste or Community.	Illiterate.		Literate		Well-educated.	
		Male	Fem	Male	Fem.	Male	Fem.
8.	Brahmin		3	2		3	
2.	Prabhu					2	
1.	Rajput			1			
3.	Marwadi		1	2			
2.	Sonar		2				
9.	Wani	5	2	2			
2.	Shimpi		2				
4.	Teli	3	1				
1.	Sutar	1					
3.	Koshti	1	1	1			
3.	Mali	2		1			
2.	Naidu		2				
1.	Dhobi			1			
1.	Burud	1					
1.	Goundi	1					
2.	Nair		1	1			
1.	Kalal	1					
2.	Koli	2					
1.	Devadasi		1				
1.	Ramoshi	1					
2.	Dhangar	1	1				
55.	Maratha Kunbi	30	19	5		1	
2.	Kamati		2				
2.	Chambar	2					
1.	Gosavi	1					
1.	Wadari	1					
2.	Madrassi outcaste	1	1				
19.	Mahar	15	4				
5.	Mang	4	1				
1.	Beni-Israel			1			
2.	Pathan	1	1				
13.	Mahommedan	9	4				
1.	Anglo-Indian					1	
34.	Indian Christian	4	6	10	10	4	
190.		87	55	27	10	11	
	Totals	142.		37.		11.	

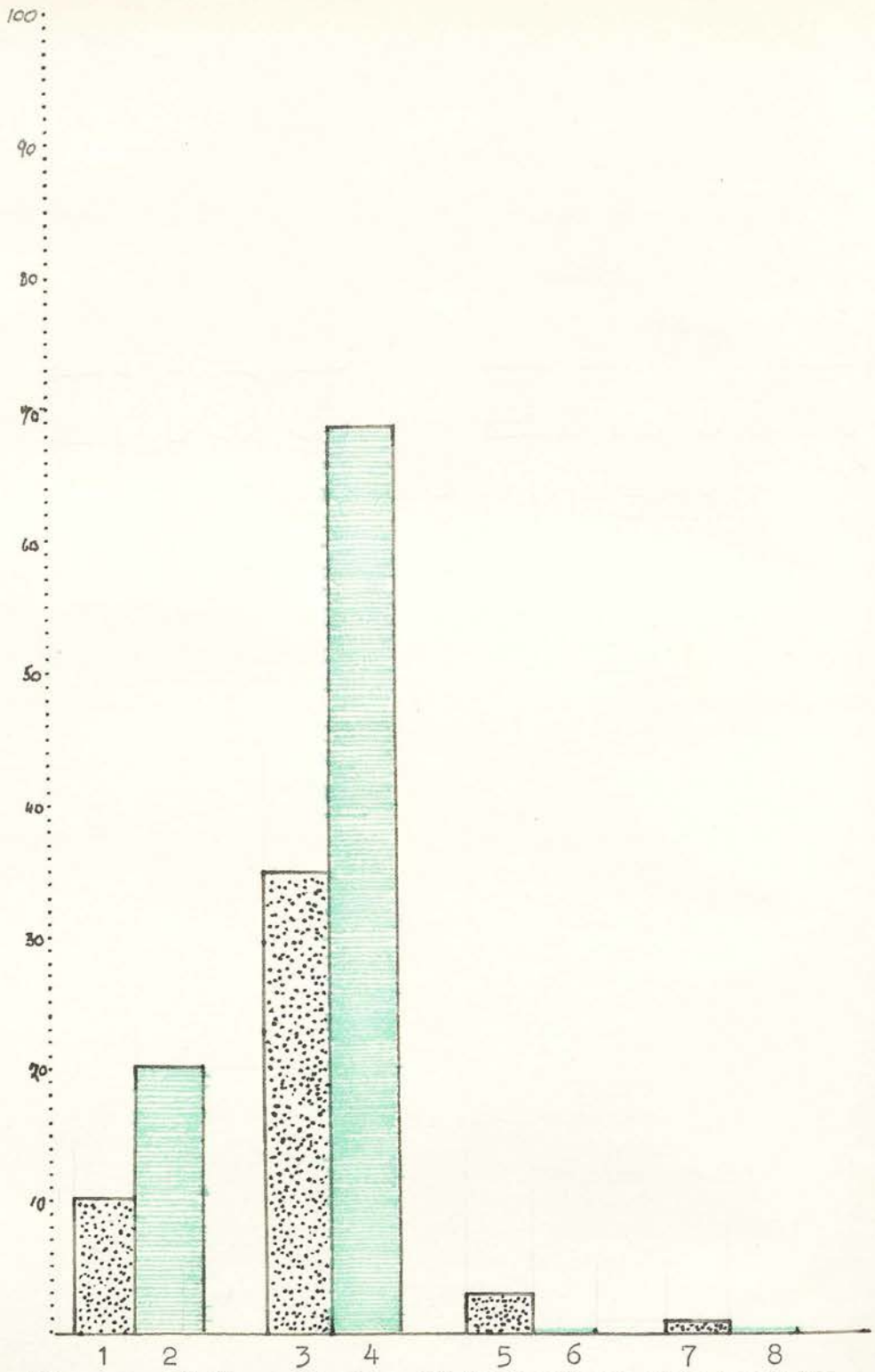


FIGURE 5 Diagram of Comparisons of Percentage Literacy in the general population and in lepers, constructed from data in the East India Census,1931, and in TABLE XX

1,Hindu Castes, general population, literacy 10.2%.
2,Hindu Castes, leper cases, literacy 20%.
3,Christians, general population, literacy 34.06%.
4,Christians, leper cases, literacy 68.5%.
5,Outcastes, general population, literacy 2.8%.
6, Outcastes, leper cases, literacy 0%.
7,Mahommedans, general population, literacy 0.67%.
8,Mahommedans, leper cases, literacy 0%.

In the case of these lepers, 48 are literate and 142 illiterate, the percentage literacy being 25.26%. Compared with the percentage in the general people of 10.6%., it is evident that some significant factor is at work. This may be expressed simply by saying that education encourages lepers to come for treatment. Our cases contain quite a number of ignorant police-ingathered lepers, but in spite of that we find that the voluntary incoming of educated people is so great as to raise the literacy rate as high as two and a half times as much as that of the general people. The influence of education is patently one to be made use of in the campaign against leprosy. In FIGURE 5., it is depicted how communities which have a reasonable degree of literacy are apparently influenced to come forward when they become lepers, whereas communities with a low degree of literacy are as yet not so influenced. The latter communities, namely the outcastes and the Mahommedans, are the ones which provide the bulk of the statutory or compelled cases, and it is this factor, rather than education, which is generally responsible for their forming part of any group of cases of leprosy.

(The data of general population literacy upon which the Diagram in FIGURE 5 is built, are derived as follows:-
Hindu Castes, population 16,622,000, containing 1,700,958. literates above 5 years of age, or 10.2%. literates.
Christians, population 317,042. containing 108,176. or 34.06%. literates.
Mahommedans, population 4,456,897. containing 312,955. or 0.67%. literates.
Outcastes, population 1,750,424, containing 49,012. or 2.8%. literates.
These figures are from the EAST INDIA CENSUS, 1931).

TABLE XXI.

AGE OF LEPERS WHEN FIRST SUBMITTING TO TREATMENT.

1-5	6-10	11-15	16-20	21-25	26-30	31-35.
0	7 (3.6%)	13 (6.8%)	24 (12.6%)	30 (15.7%)	38 (20%)	20 (10.5%)

36-40	41-45	46-50	51-55	56-60	61-65	66-70.
20 (10.5%)	12 (6.3%)	16 (8.4%)	6 (3.1%)	2 (1%)	1 (0.5%)	1 (0.5%)

It will be seen from the above TABLE that 132 cases out of 190, or 69.4%. of cases came under observation in the age periods 16 to 40 years. In the age period of 1 to 15 years, only 20 cases, or 10.5%. came: while in the age period 41 to 70 years, there were 38 cases, or 20%.

TABLE XXII.

AGE OF PROBABLE ONSET OF THE DISEASE.

1-5	6-10	11-15	16-20	21-25	26-30	31-35	36-40
11 (5.7%)	24 (12.6%)	38 (20%)	57 (30%)	34 (17.9%)	15 (7.9%)	5 (2.6%)	3 (1.5%)

41-45.	46.50.
2. (1%)	1. (0.5%).

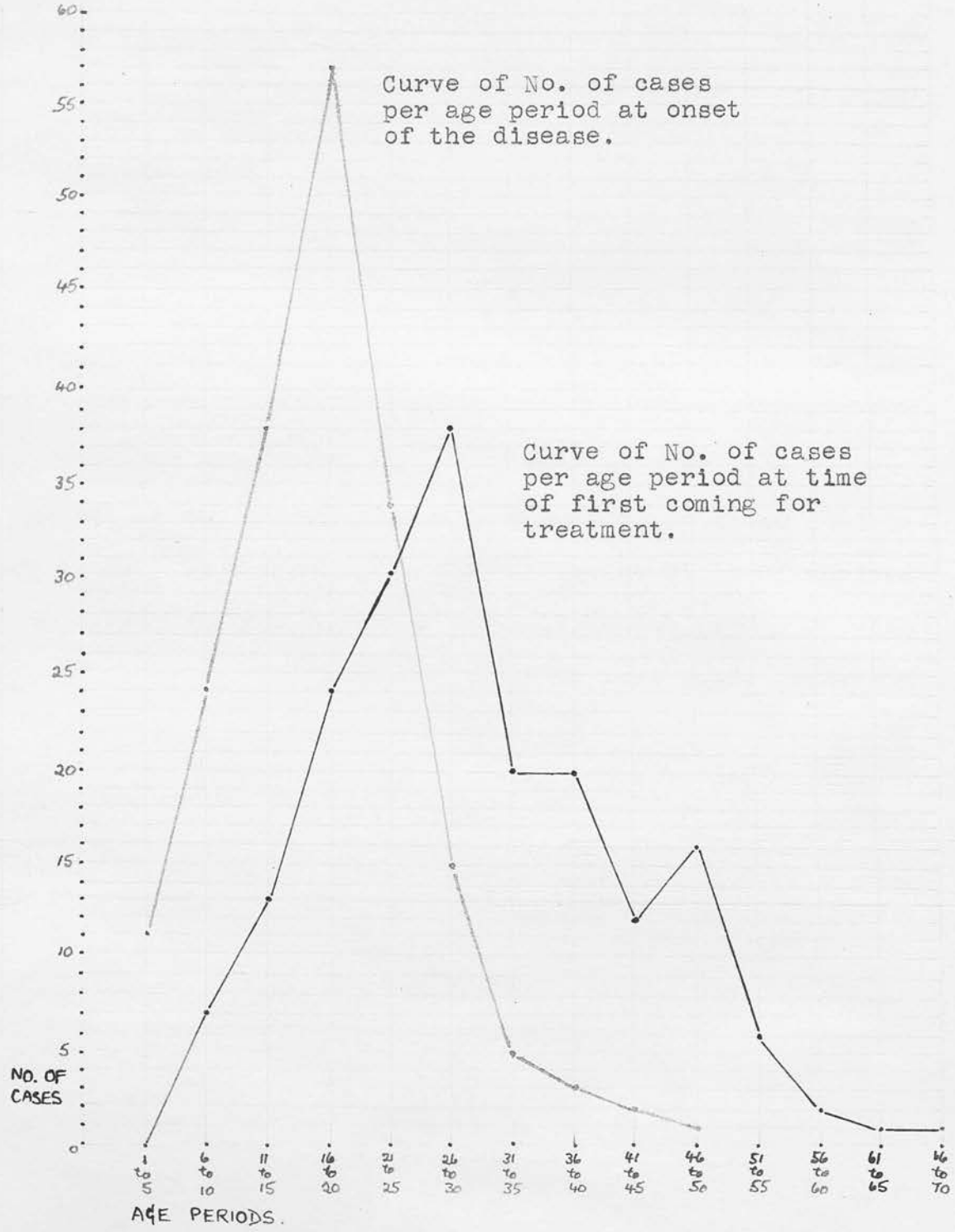


FIGURE 6 Graph of cases of leprosy per age period of onset, and of cases per age period when first seen, in 190 cases. To show the marked delay in seeking treatment after onset of the disease.

The above table was compounded from the results of special enquiry in each case as to the age of reasonable probability at which the disease originated. The main facts which emerge are:-

in the first three decades of life, 179 cases, or 94.2% of cases of leprosy began;

in the fourth and fifth decades of life, 11 cases or 5.8%;

in the second decade of life, 95 cases, or 50%.

From this it seems that leprosy is a disease sustained chiefly in the first three decades, and in that period, chiefly in the second decade.

Delay in seeking treatment, or in coming even compulsorily under treatment, is revealed by a comparison of the two previous Tables. These Tables deal with the same cases, yet right through the age periods a 'time lag' is seen. For though 11 cases had their probable time of onset at 1 to 5 years, not one came under observation at that age; though 24 cases probably originated at the age period 6 to 10 years, only 7 were first seen at that age; and the state of affairs is similar right through the age periods. There is a 'shift to the right' throughout the series, which is more fully depicted in FIGURE 6.

The factors responsible for this state of delay in seeking relief are the degree of ignorance an apathy which exists among the people, the generally low value that is put on human life, the slow development of the disease

itself and the lack of spectacular pathological effects in its early stages, and perhaps most of all the meagre resources in medical men and equipment and accommodation which are available for the treatment of leprosy. If we accept the estimate of the number of lepers in Poona District as 3,725, and consider that only some 300 are under treatment or cared for, the insufficiency of anti-leprosy resources will be appreciated. This is bound to tend to discourage lepers to come forward, and to come early.

The position is further demonstrated by a consideration of the cases of old people who came under review at age periods so late as 51 to 70 years. Some of these cases had stayed at home untreated for as long as 20 to 25 years, and only found themsleves in the leper institution by reason of destitution or vagrancy or some other cause, often with the disease 'burnt out,' and showing only the crippling secondary effects.

In order to elucidate the matter still further, the facts of duration of the disease before each case same under control, have been gathered into the following TABLE:-

TABLE XXIII. DURATION OF LEPROSY BEFORE APPREHENSION.

1 yr.	2 yrs.	3 yrs.	4 yrs.	5 yrs.	6 yrs.	7 yrs.	8 yrs.	9 yrs.
4	5	7	7	28	13	3	10	3

10 yrs.	11 - 20 yrs.	More than 20 yrs.	% within 4 yrs.
10	71	29	11%.

These figures speak for themselves. Only 11%. of the cases came under control within four years of onset of the disease. While we are not acquainted with the mode of transmission of the disease and do not know for certain at what time in its course it is infectious, there is no reason for doubting that the early stages are dangerous. The defects of the present methods of segregation in Poona District are well illustrated in the above TABLE. As many as 37%. came under control within 11-20 years from onset of their disease, and 15%. came within periods of over 20 years. Naturally the long incubation period must be allowed for, but even so, these figures are sufficiently indicative of the great delay which occurs in obtaining control of lepers.

CIVIL STATE OF THE LEPERS. TABLE XXIV.

Married.	Unmarried.	Widower or Widow.
67 cases.	99 cases	24 cases.
(35.2%).	(52.1%)	(12.6%)

The predominance of the unmarried is to be expected, as the onset of leprosy at the age which marriage is contracted tends to keep many single. In the general population the number of unmarried persons at marriageable age is negligible, but in this group of lepers over half are unmarried. It is clear that there must be a valuable social prejudice which places obstacles in the way of a leper's

marriage. Only the very obvious lepers, however, will not be able to marry: those who are unaware of their disease, or have an amount of it which is not noticeable, will be able to do so. Of this type there have been many, for taking the married, widows, and widowers together, 91 persons, or 47.4%. had entered the married state.

The group of widows and widowers proves to be almost identical with those lepers in the age group 46 to 70. as previously given in the Table of age of lepers when first submitting to treatment. An enquiry among this group made it quite clear that the manifestations of the disease had become more numerous and severe in the years following the loss of the other partner. The disruption of a well-habituated mode of life seems to have had some influence on the course of the leprosy. No doubt, in the case of widows, change in economic position from independence towards destitutism had in some cases a strong influence. It is such economic pressure in the case of younger widows that may compel them to adopt a loose life, with danger to all concerned. In the case of widowers, the loss of the wife often means a falling off in the quality and regularity of meals, and a deterioration in personal hygiene. Such, for instance was the case with regard to Baburao Phule, 51 years of age (case No.22) and Khondilaka Tukaram, 55 years of age (case No.26).

Association of the patients with lepers.

In making a study of the records to determine what

association with lepers in the home or elsewhere is reported, one realises that the sources of errors are many. In a country where leprosy is as prevalent as in India, a large proportion of the population unknowingly may come into contact with the disease. It is also probable that when the history is being taken, an association may be concealed, through the fear of the person interrogated that admission of association with a leper would contribute to making positive a diagnosis in his own case. In the Table, where the patient was emphatic that there was no previous association with lepers, the case is recorded as such, and where the patient gave hesitating or doubtful answers, the case is recorded as 'no information.' The figures in the ensuing Table indicate that association with lepers is admitted in 70%. of all cases (133 among 190), and in that the presence of the disease in a parent, grandparent, sister or brother, uncle or aunt constituted most of the acknowledged associations. There were few who acknowledged intra-marital contacts, but there were 15 who described a casual contact with a leper stranger. Prolonged and intimate contact with lepers is usually considered necessary to bring about infection, but the 45 cases in which there was absolutely no history of association with lepers, and the cases of casual contact, rather tend to oppose this assumption in some degree.

TABLE XXV.

ASSOCIATION WITH LEPERS BEFORE APPREHENSION.

Father or Mother.	Uncle or Aunt.	Grandparents.	Brother or sister.	Son or Daughter.
32	21	18	17	3

Cousin	Grand-child	Nephew or Niece	Husband or Wife.	Fellow-servants.
3	1	2	5	1 (fellow mill-worker) 1 (fellow assistant in tailor's shop) 2 (fellow house servants 4

Friend	Casual contact.	No information.	No association.
12.	2 (in motor bus) 2 (leper prostitutes) 1 (rest house) 1 (in theatre) 1 (leper beggar) 1 (leper priest) 1 (in market) 1 (in courthouse) 1 (shaved by leperbarber) 1 (in Hindu 'hotel') 1 (in tea shop) 1 (fellow opium addict) 1 (at a shrine). 15	12	45

The influence of the joint family system is in the direction of extending the range of house contacts, and, therefore, house infections. Relatives other than parents may never leave the joint household if they have leprosy, and furthermore, if leprosy is developed when they are away from it, they usually return. This explains why grandparents and

uncles and aunts play such a large part in providing contacts. As far as leprosy is concerned, the joint family system is an unfortunate one. Otherwise the very considerable influence of the more remote relatives would be avoided. Yet to destroy or modify this system in India is very difficult, for it is almost universal in Poona District, as in fact in most parts of India, and is supported by the force of ancient social custom and the pressure of economic conditions.

Rarity of Conjugal Infection.

It is striking that only five cases of conjugal associations occur in the whole series. It seems that prolonged co-habitation with a leper partner only rarely results in infection of the other partner. It may simply be that so few conjugal associations occur because as a whole lepers rarely marry, but apart from this, intra-marital infection seems to be uncommon.

TABLE XXVI. DETAILS OF THE CONJUGAL ASSOCIATIONS.

Case No.	Name	Age	Sex	Details.
3	Hashim Karim	25	M	Both partners had leprosy in their families; both developed leprosy after marriage, the husband 1 year before his wife.
16	Hari Ramu Thorat	50	M	No history of leprosy in their families: wife developed leprosy at 20 yrs. and died at 30: husband developed leprosy at 40 years.

Case No.	Name	Age	Sex	Details.
25	Marybai Tivari	40.	F.	Both partners had leprosy in their families: both had mild neural leprosy at their marriage (they each concealed the disease): two of their children became lepers.
57	Dathupant Damaji	38.	M.	Both had leprosy in their families: both developed leprosy after marriage, the husband 2 yrs. before wife.
76	Noorbi	50.	F.	No history of leprosy in their families: wife discovered her husband to be a leper after their marriage, when she was 19: wife developed leprosy at 30 years.

Of the above cases, the second and the last seem to be the most genuine ones of conjugal infection, one from the wife and one from the husband. The other three cases are ones in which both partners had leprosy in their families of origin, and, therefore, were liable to have had the disease before marriage. The third case is interesting in that both had leprosy before marriage, and it was so mild that they concealed it: whatever may have been the effects on themselves, they provided a strong association with leprosy for their children, with the result that a son and a daughter developed the disease. To be fair to them, one must also point out that the children's leper grandfather and maternal uncle used to frequent the home in their childhood.

It will be seen that when one goes more closely into the few cases of conjugal associations, the cases of reasonably probable conjugal infections become very few indeed.

Among the cases reporting casual contacts there are two which admitted sexual association on one occasion with a prostitute who was a leper (Cases Nos.10 and 53). In neither case is it possible to ascribe their disease to this contact, as there are so many confusing factors. The most that can be concluded is that they did not have the disease before the contact.

Fertility of Leper Women.

Among the female patients there were 43 women who were married or had been married.

TABLE XXVII.

FERTILITY OF 43 LEPER WOMEN.

Serial number.	Case number.	Age	Number of years a leper between 15-40.	Number of children.
1	11	25	10	3
2	13	40	15	5
3	25	40	25	4 (2 lepers)
4	55	27	9	4 (1 leper)
5	59	40	15	6
6	67	40	20	5
7	72	40	20	3
8	73	50	15	6
9	74	20	5	1
10	75	40	20	1
11	76	50	10	4
12	80	30	15	4
13	82	30	6	0
14	83	30	14	3
15	109	35	5	3
16	110	25	10	4

TABLE XXVII (cont).

Serial number.	Case number.	Age.	Number of years a leper between 15-40.	Number of children.
17	115	30	15	5
18	116	35	20	2
19	118	30	15	2
20	128	50	20	6
21	129	55	15	4
22	132	30	15	2
23	133	60	8	5
24	135	50	10	4
25	141	45	20	6
26	145	45	13	3
27	152	35	19	5
28	154	40	14	4
29	155	40	20	5
30	161	50	15	6
31	162	35	13	3
32	171	35	5	2
33	177	45	25	2
34	178	45	21	5
35	179	30	15	2
36	180	30	6	1
37	182	40	25	5
38	183	25	10	2
39	184	50	20	3
40	185	50	16	3
41	186	50	20	0
42	187	50	13	4
43	188	55	15	3

The women number 43. The total number of leprosy years between the ages of 15 and 40 is 637; that is, there is an average of 14.8. leprosy years per woman. In the time of the total number of leprosy years the women have given birth to 150 children. If they had been lepers for the whole period of fertility, taken as 25 years, they would have given birth on the average to 5.9. children each; that is their coefficient of fertility is 5.9. (The actual average of children per woman is 3.5).

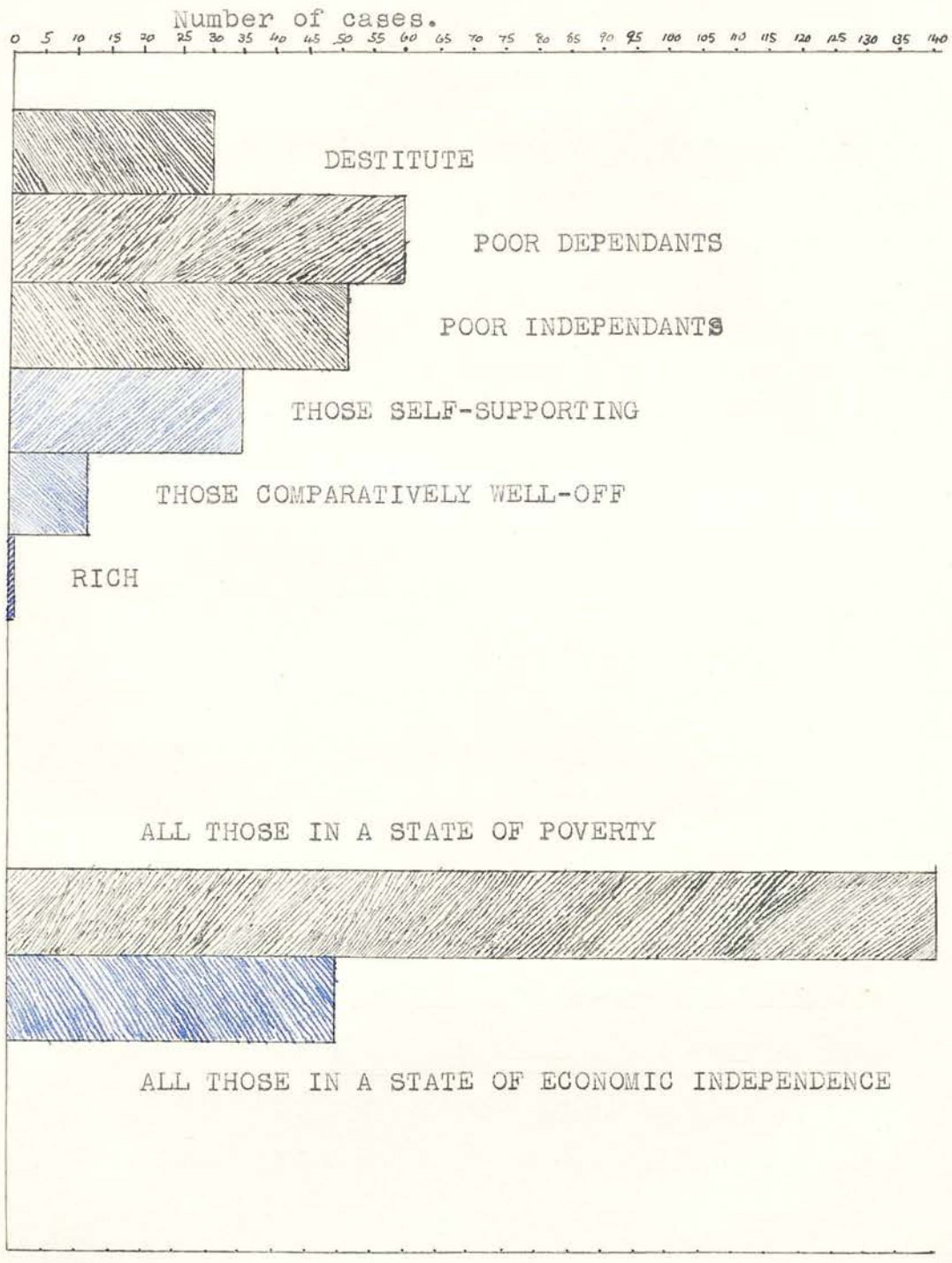


FIGURE 7 Economic position of 190 lepers.

This coefficient of fertility may be compared with that of another group of leper women in the tropics, namely, those in Surinam. For 29 women there, P. H. J. LAMPE, 1933, finds the coefficient of fertility to be 6.6, and he gives that of the non-leprous native woman in Surinam as 6.3.(146) The coefficient of fertility of the Poona cases is thus rather less than the Surinam women, which is much the same as in the non-leprous Surinam women. The figures for the non-leprous women in Poona District are not available.

Economic Position of the Lepers.

The economic state of each leper in his or her normal life before becoming a leper is of importance with regard to possible influence on the development of the disease. The results of a survey are shown below, and also in Figure 7.

TABLE XXVIII.

Destitute	29 cases.
Poor Dependants, (those wives, children and poor relations maintained at a rate less than 10 rupees, about 15 shillings per mensem).	58 cases.
Poor Independants, (those with an income of less than 20 rupees, about 30 shillings, per mensem, but still drawing some slight support from being part of a family group).	54 cases.
Self-supporting, (income 20-25 rupees , about 30-37 shillings, per mensem). ..	35 cases

Comparatively well-off, (income 24-40 rupees, about 37-60 shillings, per mensem .. .. 13 cases.

Rich, (income over 40 rupees, or 60 shillings, per mensem) .. .. 1 case.

Combining the figures for all those with the common factor of poverty, namely, the destitute, the poor dependants and the poor independants, we get 141 cases, or 74.2%. in a state of poverty. Similarly the self-supporting, the comparatively well-off, and the rich give together 49 cases, or 25.8%. in a state of economic independence.

In classifying the cases, due regard has been paid to the current standards of Indian life in the District. It has been ascertained by experiment with a students' hostel in Poona that an average healthy Indian can live, according to his present standards, on 7-12 rupees, or about 10-18 shillings, per mensem. This provides what would satisfy an Indian, but it is a low standard of living according to European ideas. Wealth in itself seems no protection against leprosy, if, as in the case of the single rich man among the patients, no use be made of it to elevate the standard of living and hygiene of its possessors. The great preponderance of the poor in the series of patients suggests that leprosy, though it may attack rich and poor, is liable to have a higher incidence among the poor. A wealthy person can if he wishes obey the rules of health, or at least obtain advantages in the way of space, air, food, housing, clothing, etc., which may be denied to

poorer people. It is probable, however, that if a rich man be fond of over-indulgence, or be careless, he puts himself in danger of diseases like leprosy, given that he lives in a country where it is endemic.

> The author, while on a visit in 1933 by the courtesy of DR. DREW to Peel Island Leper Settlement in Queensland, was informed by him of the history of a certain wealthy leper. The patient for some 10 years had been living alone on his property in the far West in a state of squalor, and also of promiscuity with the aboriginal women: he had made no use of his wealth to ensure cleanliness, regular food, or good housing. Presumably the infection was sustained from an aboriginal with whom he had been in close contact, and the man had made himself a ready soil for the growth and development of the infection.

As far as Poona District is concerned, it is justifiable to trace a direct or an indirect connection between poverty and leprosy, as in about three quarters of the cases poverty was of a degree sufficient to devitalise the people concerned.

Housing of those who had become lepers.

Bound up with the question of economic position is that of housing. It was ascertained that some of the patients had lived in houses of two to four rooms, and the others had lived in single rented rooms, either in houses of others, or in 'chawls,' or tenements, in the big cities. The average size of the rooms, in houses or tenements, was found to be eight by ten, or ten by twelve feet. In a survey of the cases, it was discovered that in a single room there may sleep and live as many as twelve persons, this being so

especially in tenement life in the industrial areas.

TABLE XXIX.

HOUSING OF THE LEPERS PRIOR TO APPREHENSION.

No. of lepers who had no housing:-	26
Lepers who had lived in a single room:-	72
Lepers who had lived in a house of 2-4 rooms:-	92
Average No. of persons amoung whom the leper dwelt in a single room:- (total No. of persons in 72 instances of living in single rooms, 409).	5.6
Average No. of persons among whom the leper dwelt in a house of 2-4 rooms:- (total No. per persons in 93 instances of living in a house, 1299).	14.1.

With such small houses and rooms, it is evident that a considerable degree of over-crowding obtained in the dwellings from which these lepers came.

It was further discovered that not only was there over-crowding of persons, but also overcrowding of houses. Except in small and hilly villages, the houses are jammed together. The principal prescribing the height of buildings in relation to width of street, and preventing the erection of houses to the last limit of boundary walls in every case, seems to be ignored by most Municipalities, with the result that the Indian city is apt to be a crowded welter of houses and shops threaded by narrow streets. The result is a massing of the population in a typical way, which gives great anxiety in times of plague and epidemic diseases, and

facilitates the spread of leprosy.

With regard to the chawls and industrial tenements, massing of the population and over-crowding is perhaps at its worst.

Personal Cleanliness.

In an effort first of all to assess the degree of personal cleanliness of the people generally, use was made of the fact that the N. M. Wadia Hospital in its city and village work reaches all classes and types of the people. On a Saturday in May (hot weather) and on a Saturday in January (cold weather) counts were made for cleanliness of every patient seen. The patients included examples of practically every class that is found in the District, and even Europeans and Parsees, (the latter are a small but affluent and well-educated business people of old Persian origin). Village centres were included in an effort to make the count as representative as possible. The criterion of cleanliness was made as simple and decisive as possible, it being noted in each case whether the person had, 1. clean clothes or not, and 2. clean body or not, on the ordinary medical examination. The leper cases under review had been dealt with as they came under control first throughout the years of survey, and in the same way.

TABLE XXX.

CLEANLINESS IN THE GENERAL POPULATION AND IN LEPERS.

	Clean in clothes and body	Not clean.
A. Result of an examination of 202 cas s on a day in May, (general cases).	100 cases, or 49%.	102 cases, or 51%.
B. Result of an examination of 152 cases on a day in January, (general cases).	69 cases or 45%.	83 cases, or 55%.
Averages for A and B.	47%.	53%.
C. Result of examination of 190 lepers.	76 cases, or 40%.	114 cases, or 60%.

In the above survey, only moderate cleanliness sufficed to prevent the patient being put in the 'not clean' class, so each doubtful case received the benefit of the doubt. Yet although judgment was on the side of leniency, the figures suggest that the general people tend to be unclean personally, and that they tend to be less clean in the cold weather. The comparison of the lepers with them shows that lepers as a class are less cleanly than the ordinary population.

Poverty is largely responsible for the low standard of personal cleanliness: soap is too dear to be bought freely, and even the use of oil as a cleanser is inhibited by its cost: clothes are few and tend to be worn long, to save the cost of washing and outlay for new clothes. The average

personal bath is taken with a small quantity of water, and no soap, and even if access to some public tap affords a more copious supply of water, it does not necessarily mean that the person is clean after the bath. In lack of personal cleanliness leprosy seems to find a greater opportunity to lodge and grow.

Pyorrhoea and Dental Caries.

The lepers were surveyed and classified according to the state of the mouth and teeth.

TABLE XXXI.

STATE OF THE TEETH OF 190 LEPERS.

Good, (clean sound teeth, with apparently no pyorrhoea, and no caries).	27 cases, or 19.4%.
Fair, (pyorrhoea beginning or moderately advanced, or some caries, but most of the teeth present)	89 cases, or 46.8%.
Bad, (advanced pyorrhoea or caries, few or no sound teeth).	64 cases, or 33.6%.
Total number with pyorrhoea or caries.	153 cases, or 80.5%.

The devitalising effect of sepsis and dyspepsia, associated with pyorrhoea and dental caries, is considered to predispose to leprosy.

Diet.

The type of diet is mostly dictated by the rule and custom of the caste or community to which a person belongs:-

1. High Caste Group:- those who are strict vegetarians and who look on meat, eggs and fish with abhorrence:
2. Middle Caste Group:- those who are vegetarians, but not strictly so, as some eat fish and eggs occasionally, and a few will not refuse mutton: the latter is, however not often obtainable:
3. Outcaste Group:- thos who will eat anything they can get, incuding carrion in some instances, but through poverty are usually confined to grain of poor quality:
4. Beni-Israel Group:- those who eat a carefully-chosen mixed diet according to Jewish custom:
5. Mahommedan Group:- those who eat a mixed diet, including beef, mutton, fowl, fish and eggs:
6. Anglo-Indian Group:- those who eat according to an Indianised European standard, that is, a mixed diet with foods prepared in the Indian way:
7. Indian Christian Group:- those who eat according to a varied standard, some following a vegetarian Hindu Caste dietary, some a diet approximating to that of the Mahommedans, some through poverty following the Outcaste low standards.

It will be convenient now to consider certain foods, consumption of which belongs to a good dietary, and see what proportion of the leper patients were in the habit of using them:-

TABLE XXXII.

CONSUMPTION OF MILK, MILK PRODUCTS AND FRESH FRUIT, AND VEGETABLES IN THE DIETARY OF LEPERS BEFORE APPREHENSION.

Fresh Milk.		Curds.		Butter.	
Daily	Occasionally	Daily	Occasionally	Daily	Occasionally
1 case or 0.5%	24 cases or 12.6%	5 cases or 2.6%	59 cases or 31%	0	21 cases or 11%
Ghi (clarified butter)		Sweetmeats containing milk or milk products.		Imported condensed milk.	
Daily	Occasionally	Daily	Occasionally	Daily	Occasionally
16 cases or 8.4%	108 cases or 56.8%	0	45 cases or 23.6%	0	12 cases or 6.3%
Fresh Fruit			Fresh Vegetables.		
Regularly	Occasionally		Regularly	Occasionally	
10 cases or 5.2%	96 cases or 50.5%		1 case or 0.5%	57 cases or 30%	

The daily or regular consumption of fresh milk, fruit, and vegetables is extremely small. Occasional consumption of milk products as curds, butter, ghi and sweetmeats is more common: likewise the occasional use of fresh fruits and vegetables. Taking it as a whole, the consumption of these fresh vitamin-containing foods is in more than half the cases almost negligible.

As the majority of cases use Bajri, dal and rice as the pillars of their dietary, it was hard to find in the whole series many examples of a reasonably good diet, because of the defects in proteins, fats and salts in these grains (previously pointed out), and the tendency throughout to rely on a few constituents. If only more variety were the rule, the defects of one foodstuff would likely be counterbalanced by another. As the present position is, there was not one dietary but had some serious defect or other. Some samples of the dietary findings are given below:-

Case No.1. Staple constituents, dal, with a little rice and fried vegetables: much spices and condiments: a little meat, fish, bajri chappaties occasionally: no milk, no fresh vegetables, fruit only rarely.

Case No.2. Staple constituents, dal, much rice, jowari bread and fried vegetables: subsidiary and occasional are milk, wheaten chappaties, curds, a little spices and chutney, fresh vegetables and fruit: never fish, meat or eggs.

Case No.3. Staple constituents are sheep and goat mutton, with a great deal of rice and spiced things: subsidiary are fried vegetables: no eggs, fish, chicken, fresh milk, fresh fruit.

Case No.4. Staple constituents are dal, rice, bajri; and subsidiary are wheat, ghi, occasional meat, fruit and vegetables; no fresh milk.

Case No.5. Staple are dal, fried vegetables, and wheat chappattees: subsidiary are dal, spices and condiments, a little fish or mutton twice a week, no fresh milk, no fresh vegetables, only a little fruit.

Case No.6. Staple are dal and rice: subsidiary are wheat bread and cooked vegetables: no fruit.

Case No.7. Staple constituents are jowari bread, dal and rice: subsidiary are fried vegetables, ghi, curds, dried fish. No fresh milk, no fresh fruit, no fresh vegetables.

Case No.9. Staple are dal and rice, bajri and millet bread: subsidiary are a little ghi and vegetables occasionally: no milk, no meat, no eggs, no fresh fruit.

Case No.10 Staple constituents are dal and rice, subsidiary and occasional are spices, wheat chapatties, curried vegetables: no fresh milk, no fresh fruits, no fresh vegetables.

Case No. 142. Dal and bajri bread as the staple constituents: no fresh milk, fruit, nor vegetables: no meat nor eggs.

Monotonous and devitalising diet is widespread among the people, and the chief defects are, a lack of good proteins, an excess of carbohydrates, a deficiency in salts, and a rarity of the vitamin-containing foods. To this may be added the excess of spices, salt and condiments due to the unattractive nature of the few constituents of the diet. Bajri, which is eaten so largely in the District, is one of the most unattractive foods in taste and appearance: and dal is very tasteless by itself.

The composition of the common Indian foods is given in the Table, the facts being derived from J. P. BOSE, 1928, (147)

TABLE XXXIII. COMPOSITION OF FOODSTUFFS IN COMMON USE.

Foodstuff.	Carbohydrate gms. per oz.	Protein gms. per oz.	Fat, gms per oz.	Calories per oz.
Bajri meal	20.8.	2.2.	0.36.	97.6.
Maize	18.3.	2.8.	1.9.	95.8.
Rice, average	23.6.	1.42.	0.43.	102.1.
Wheat, whole	19.0.	3.3.	0.8.	96.4.
Dal, average	15.9.	6.7.	0.65.	96.25.
Butter	0.0.	0.3.	25.7.	232.0.
Curds	0.8.	1.3.	1.0.	17.0.
Ghi	0.0.	0.0.	24.0.	223.0.
Milk (buffalo)	1.4.	1.2.	2.5.	33.0.
Milk(bazar cow's)	0.7.	0.7.	0.6.	10.0.
Milk (cow's, pure)	1.3.	1.1.	1.2.	20.0.
Milk (goat's)	1.1.	1.0.	1.2.	19.0.
Milk (condensed)	15.1.	2.7.	3.1.	102.0.
Fish, average	0.0.	5.5.	1.0.	31.8.
Butcher meat, ave.	0.0.	6.0.	3.0.	51.0.
Beef	0.0.	5.8.	0.99.	27.0.
Chicken	0.0.	6.1.	0.7.	31.0.
Goat mutton	0.0.	6.8.	0.7.	34.4.
Mutton	0.0.	4.5.	4.5.	60.3.
Egg, hen	0.0.	3.8.	3.3.	44.9.
Vegetable oil	0.0.	0.0.	28.4.	255.0.
Sooji (wheat)	13.5.	4.0.	0.6.	75.8.
Chapatti (wheat)	19.65.	2.67.	1.05.	101.2.
Plantain	4.0.	0.5.	0.0.	18.0.
Bael fruit	4.5.	0.18.	0.2.	20.8.
Coconut (kernel)	1.5.	1.7.	15.1.	148.7.
Coconut water	0.7.	0.4.	0.0.	3.4.
Dalim(pomegranite)	0.19.	0.18.	traces	9.0.
Guava (peru)	3.2.	traces	0.0.	13.0.
Raisins	21.6.	0.73.	0.93.	99.8.
Mango (ripe)	4.99.	0.34.	0.21.	23.2.
Orange	1.9.	0.1.	0.1.	9.0.
Milk sweetmeats	11.4.	5.16.	5.6.	120.0.
Badam (almonds	2.0.	6.7.	15.2.	176.9.
Ground nuts	2.3.	6.8.	13.4.	157.0.
Akrot (walnut)	4.8.	4.8.	19.0.	209.0.
Green vegetables	1.0.	0.25.	negligible	5.0.
Potato (Bombay)	4.3.	0.49.	0.15.	20.0.
Potato (Deshi)	5.8.	0.50.	0.1.	27.0.

It will be seen that a proper combination of the foods available would secure a better diet than is at present the

rule, but the obstacles of poverty, caste prejudice, and ignorance are formidable. There is further the drawback of a degree of adulteration of foods which would be hard to surpass in any other country.

Predisposing Diseases.

The most satisfactory survey of the other diseases which affect lepers would be a pathological one by means of autopsies. Unfortunately, permission for an autopsy is very hard, usually impossible, to obtain in these regions, and there was in any case no death among the patients under review during the time of the survey. As a result, clinical and laboratory methods alone were available in seeking to ascertain if the lepers had any diseases predisposing to the development of leprosy, and its aggravation when developed. Because of this, it would be valuable first to state briefly what E. V. PINEDA, 1924, (148) found in his pathological survey of 300 autopsies at Culion. He found that:-

1. The inmates of the Culion Leper Colony seldom die from the direct effects of the disease. In but 2.3%. of the autopsied cases could leprosy be assigned as the cause of death.

2. Tuberculosis is the most frequent cause of death among the lepers at Culion, 24%. of the autopsied cases and 42.7%. of the total deaths having been due to this disease.

3. Nephritis is second in frequency, causing death in 16.3%. of cases autopsied. It should be considered not as a complication, but as a true sequela of leprosy. Several factors in leprosy are responsible for nephritis, such as early and often extensive involvment of the skin, the absorption of

toxins from infected ulcers, the absorption of toxin from the infecting organism and from broken-down tissues: the irritant action of ethyl esters on the kidney cannot be ignored.

There is a general tendency to ascribe morbid conditions occuring in leprosy to leprosy itself, as J. M. H. MACLEOD, 1909,(149) and G. A. HANSEN, 1902,(150) early pointed out. However, we are approaching the subject from the point of view of whether morbid processes detected in the early examination of lepers, and definite history of the processes in the former life of the leper, do not refer to conditions which inclined the person to sustain leprosy, and to advance more rapidly in the disease. As far as causes of death are concerned, we have to consider whether the morbid processes which formed such are not also predisposing. PINEDA mentions two as frequent causes of death in Culion, tuberculosis and nephritis. It seems that the latter can be dismissed as a predisposing cause, being of the nature of a sequela. In the 190. cases seen in Poona, the urine examination when the case first came under review yielded the following result:- 43 cases, or 22.6%. were positive for albumen, and 31 cases, or 16.3%. were positive for casts. This indicates that a proportion of the cases were nephritic when first seen, but it is not much evidence that nephritis predisposes to the leprosy, as the disease had in most cases been in existence long enough for the nephritis to have been a sequela.

Tuberculosis is in a different position. With such

a widespread and common disease, leading to great devitalisation of the patient and wasting, it is reasonable to expect some connection with predisposition to leprosy.

The onset of pulmonary tuberculosis as a complication in advanced cases of leprosy is a common finding by most observers, (J. M. HENDERSON, 1930).(151) There were a number of such cases in our series, but they must be disregarded for our purpose, and tuberculosis at the beginning or preceding the leprosy must be sought for. The matter is complicated by the fact that true leprosy of the lungs, though rare, undoubtedly occurs, as E. MUIR, 1933,(152) has described nine cases, and quotes others before him who have described lung leprosy, such as HANSEN and LOOFT, WADE, DOUTRELEPONT, BABES, and others. Because of this complication, cases in the present series with acid-fast bacilli in the sputum were not accepted, unless guinea-pig innoculation was successful. The following are the cases in which pre-existing or concurrent early tuberculosis was traced, and which might be held to have predisposed to leprosy:-

Case No.2. Govind Bapat, Male, 18 years: N-2.C-1. leprosy began at the age of 15 years, with depigmented anaesthetic patches on back, and blisters on fingers of left hand, At the age of nine years, he had tuberculous glands in the neck, which were operated on in the hospital (confirmed by the hospital records), and the scars of these could be seen. He had been subject to fever in his childhood, and his sister died of pulmonary tuberculosis. His sputum was frequently examined, but no acid-fast bacilli were found.

Case No.6. Maruti Laxman Holi. C-3. Male, 28 years: leprosy began about the age of 22. He had had a 'weak chest' since the age of about 16 years, and cough and expectoration for 7 or 8 years. His sputum was examined, and was found

positive on staining, and also by guinea-pig inoculation, for tubercle bacilli. An aunt had died of 'wasting disease.' His grandmother was a leper, and he lived in a house containing 14 people. He was certain that chest trouble preceded the onset of leprosy by several years.

Case No.15. Dathu Mandku, 15 years: Male: N-1. leprosy began at 12 years of age, with neuralgic pains in left thigh, and an anaesthetic patch on the outer side of the left thigh, which was positive for leprosy bacilli when a skin clip was examined. He had walked with a limp since the age of 10 years, and shortly after he came under observation as a leper, a psoas abscess was aspirated, yielding typical creamy tuberculous pus. Rest and other treatment of this lesion had over a period of a year marked effect on the leprosy, as he became negative for leprosy bacilli in his one lesion, no other lesions than one on the cheeks developed, and in three years he was very fit, and classed as 'arrested' as regards leprosy.

Case No.20. Chintamani Kamalapurkar. Male. 30 years: N-1: leprosy began at about the age of 20-25 years, with lesions of lower limbs. At 19 years of age, he began to have regular fever, cough, sweating, and later wasting and expectoration. His father had died of tuberculosis, so he put himself under treatment by a country 'vaidya,' and took much medicines and tonics. On examination, exudative and cavitating lesions were found in the lungs, and the sputum was positive for tubercle bacilli, microscopically and on guinea-pig inoculation. In spite of treatment, his state has got worse in the year under observation, and lsprosy lesions of the trunk appeared: he left for his own town, and nothing more has been heard of him.

Case No.28. Sopana Choudari, Male. 25 years C-2. this man is one who came first under observation complaining of 'wasting disease' (tuberculosis is well known to the people under this name). He had lived all his life in a single room containing four people, of whom one, his aunt, was a leper, and another, his mother, also had 'wasting disease,' and he began to be weakly at 14 years of age. For about three years he had had fever and cough. The patient was thought to be a case of pulmonary tuberculosis merely, as the sputum was positive on staining, but after he had been in hospital for a few days, a bout of high fever brought out an extensive crop of cutaneous nodules, which on examination showed lepra bacilli. Later, his sputum was tested by guinea-pig inoculation, and found positive. His subsequent history was the rapid development of leprous cutaneous lesions, with many bouts of lepra fever mixed up

with tuberculous fever, until after 3 months he insisted on going home, and was lost sight of. It was the more difficult to keep him, as he had to be sent out of the hospital when leprosy was discovered, and refused to go into the Leper Asylum, and lived in the city with his people in an isolated hut. No doubt death would soon end the matter.

These five cases are the only ones in which one could have no doubt of the tuberculous factor: there were some others in which suspicion was strong, but could not be established. These cases, however, certainly suggest that tuberculosis may have a strong influence on leprosy.

Venereal Diseases. As syphilis may be taken as the chief representative of veneral diseases, attention was directed mainly to it in a survey of the cases. The Kahn Test for sera was relied on, as it can be most conveniently performed by one man for a great number of cases. E. M. YAGLE and J. A. KOLMAR, 1923,(153) state that the Kahn Test is negative in lepers' sera unless complicated by syphilis, but on the other hand, D. N. FORMAN, 1931,(154) from his experience of thousands of Kahn Tests, in both non-leprous and leprous patients, thinks that the test is sometimes positive in uncomplicated fairly marked cutaneous cases, and P. M. OTERO, 1927,(155) states that is is sometimes positive in uncomplicated cases of leprosy during the stage of reaction. In spite of these disadvantages, the general opinion of the Kahn Test is that for the most part it can be relied upon in leprosy (J. M. HENDERSON, 1930).(151)

The actual method and mode of interpretation of the results of the Kahn Test followed in the present cases was

the same as described by R. L. KAHN,(156)

So as to avoid possible influence of the lepra reaction on the Test, no serum was taken during the time of such a reaction, though it was sought to take the serum as early as possible after the case came under control. As for the possible influence of cutaneous leprosy, it was thought that by not counting all results below 'three plus' as positives, this factor would be balanced.

TABLE XXXIV.

RESULTS OF THE KAHN TEST FOR SYPHILIS IN 190 LEPERS.

Cases, type.	++++	+++	++	+	±	—
N-1.	1		3			20
N-2.	9	4	2	2		19
N-3.	3	2		6		13
C-1.		3	1	1		6
C-2.	2	4	3	2	3	6
C-3.	1	1		1		2
N-1,C-1.	3	2	3	1		3
N-2,C-1.		1	5	1	1	12
N-1,C-2.	4	2	2	1		3
N-2,C-2.	3		2		1	4
N-1,C-3.			1			1
N-2,C-3.	1		1			2
N-3,C-1.					1	3
N-3,C-3.	1					3
N-3,C-2.						2
Totals	28	19	23	15	6	99

Taking the figures in the 'four plus' and 'three plus' columns together, and disregarding all others, there are 47 cases, or 24.7% of the series, with sera positive for syphilis. Even if we reduce the figures to their lowest possible total, by taking away from the figures in the first

two columns, those which refer to C-2. and C-3. cases, pure or mixed, there still remain 28 positive cases, or 15%. Among the negatives there were 12 cases who gave a definite history of syphilis, so it seems as if the higher percentage is probably correct, because these cases almost cancel out the cases removed for being lepers of C-2 and C-3 type. On the whole, the figure of 24%. of the lepers being syphilised is probably the correct one.

It has been noted that in the series of 180 general cases at the Hospital, who were non-lepers, the Kahn test was positive in 15%. (Page 28). It seems, then, that syphilis has a close connection with leprosy, and can be considered a grave predisposing cause, and an aggravating factor in the course of the disease. When active anti-syphilitic treatment was carried out in these cases which were positive to the Kahn Test, and in those cases which admitted syphilis, the effect on the course of the leprosy was always marked, and in some cases remarkable. The degree of improvement to be expected in such cases became quickly recognised among the lepers and they willingly submitted to having blood taken and accepted full courses of treatment if the result of the blood test justified it.

Malaria.

Enquiry was made as to whether each patient gave a history of malaria in his life previous to becoming a leper, but only those were recorded who gave a history of five or more attacks. Besides this, blood films of each patient

were taken and searched for malarial parasites.

TABLE XXXV.

MALARIA INCIDENCE IN 190 LEPERS.

Those who gave a history of 5 or more attacks of malaria.	Those whose blood films contained malarial parasites at time of apprehension.
72 cases or 37.8%.	17 cases, or 8.9%.

It is evident that the influence of malaria cannot be ignored, when 37.8%. of these leper cases had had enough malarial attacks in their earlier life to make it an important devitalising factor. The percentage of blood positives was smaller, but unless the blood happened to be taken at a critical time in the malarial infection, it would not necessarily be positive. With the aid of the Melano-Precipitation Serological Test it would be possible to assess more completely the influence of malaria, but unfortunately this was not available to the author at the time. This test provides a means of testing for malarial infection when there may be no positive blood films and has recently been put on a practical basis by E. D. W. GRIEG, C. E. VAN ROOYEN and E. S. HENDRY, 1934,(157) This test can be tried in future surveys, and will perhaps confirm the fact which the available figures indicate, that malaria is a serious factor in leprosy.

Intestinal Parasites.

For concentrating eggs of helminths in faeces, a simple method of using saturated salt solution was adopted, because of lack of the Clayton-Lane centrifuge. The method used is much the same as that described by D. B. BLACKLOCK and T. SOUTHWELL, 1931,(158) (To emulsified faeces in a tube, saturated salt solution is added, and the tube shaken vigorously: then more saturated salt solution is added with a pipette until the meniscus of fluid is flush with the surface of the tube: from this a slide and coverslip preparation is made, after touching the surface and removing vertically with great rapidity).

By this means the examination of faecal specimens in 112 leper cases yielded the following results:-

TABLE XXXVI.

INCIDENCE OF INTESTINAL PARASITES IN 112 LEPERS.

Genus to which ova found belonged.	No. of cases.	Percentage.
Ancylostoma	34	30.3%.
Ascaris	14	12.4%.
Taenia	5	4.4%.
Others	7	6.2%.
Totals	60.	53.5%.

The incidence of intestinal parasites being over 50%. indicates that they also form an important factor in leprosy in these regions.

Other Predisposing Diseases.

Some of the diseases of lesser import which some of the lepers were subject to are given in the next Table. Pellagra, in view of possible relation to leprosy, was specially sought for, but no case was found among the lepers.

TABLE XXXVII.

INCIDENCE OF CERTAIN DISEASES IN 190 LEPERS.

Disease	No. of cases.	Percentage.
Dysenteries & sprue.	21	11%.
Skin diseases.	61	32.1%.
ringworms	6	3.1%.
scabies	10	5.2%.
impetigo	8	4.2%.
pityriasis	10	5.2%.
others	9	4.7%.
Guinea-worm	18	9.4%.
Filariasis	5	2.6%
Enteric	12	6.3%.
Cholera (survived in the past).	6	3.15%.
Myiasis	24	12.6%.

The Patients' Own Opinions as to their Disease.

It occurred to the author that possibly some illumination might be gained from recording the views of the patients as to why they sustained leprosy. Not every patient was intelligent enough to give a reliable answer, but after the confidence of the more thoughtful ones had been gained, they showed themselves willing to talk freely about their own disease. The results of this part of the enquiry have been so enlightening, that they are recorded in brief detail

in the next Table:-

TABLE XXXVII.

RESULTS OF ENQUIRY AS TO THE PATIENTS' OPINION OF THEIR OWN DISEASE.

Case No. of the Patient.	Remarks of the patient, or of the nearest relative, if a minor.
1.	Father of the patient ascribes his son's disease to the latter's acquiring syphilis in Bombay at the age of 25 years, and to the fact that there was a grandfather in the house a leper: he believes that leprosy often follows on syphilis, and on indulgence in toddy, and 'warm foods,' such as fish and game. The patient himself ascribes his disease to eating preserved fish, excessive cigarette smoking (200 per day), and excessive tea drinking (3 dozen cups of tea a day) in his case.
2.	The patient thinks that daily contact with a close friend of his at college who was a leper, and the tuberculous glands he had as a child, caused him to develop leprosy: he thinks that fish-eating, a damp hot climate as in Konkan, and a large amount of malaria in a region, are what makes the disease common.
3.	The patient ascribes his disease to the fact that his wife was a leper, and that they both had leprosy in their families, and to his taking heating medicine given him by a country hakim when he sustained gonorrhoea at the age of 20 years. He thinks that leprosy generally is to be ascribed to evil practices and dissipation and to fish-eating.
4.	This man's mother was a leper, but he does not ascribe his disease to that, but to sitting by a leper in a court-house one day in his 18th year. He says that leprosy is held by some to be inherited, but he thinks it is contagious: it is generally held to be predisposed to by eating 'heating foods,' such as preserved fish and game and by having syphilis.
5.	The mother of the child says that leprosy is inherited in families, as the patient's sister is a leper in Chingleput, and her own

Patients' Case No.	Remarks of Patient or Relative.
5. (cont.)	husband, herself, and an uncle were tainted with leprosy. She thinks that because she had albuminuria of pregnancy and eclampaia at the patient's birth, he may have been weakened and made more liable to leprosy: generally, eating meat, especially beef, is associated with the disease.
6.	Patient thinks that his grandmother being a leper and living in a crowded house gave him no chance, so that he first developed 'wasting disease,' and later hard work in a potato shop, and irregular meals, heat in the body due to his eating meat and fish and drinking toddy, caused him to develop leprosy: he thinks leprosy generally is due to irregular habits of life and heat in the body, (he used the same word for 'heat in the body' which is applied in Marathi to syphilis, but denies having had syphilis himself).
7.	Patient ascribes the infection in the first place to the fact that a friend of his father used to come to the house in his childhood, who had a sore of his foot and nodules on his face: also in his childhood he had to sit by a leper in a bus. In his youth also he acquired syphilis, and used to drink daily from his 18th year toddy and country brandy. He thinks that leprosy is contagious, and is the misfortune of fate (Karma), and that a leper should be considered an Untouchable. One is made more liable to leprosy by eating bad fish, eating milk and meat together, having syphilis, cohabitation during the monthly periods of wife, (this is a very widespread belief), irregularities in diet, alcholic indulgences, late hours, meals in hotels, meals at irregular times, working at writing lat at night for greed of money, overeating in the hope of getting strong.
8.	The patient thinks that his leprosy was due to his not getting enough to eat all his life.
9.	Patient says his disease was caused by his having much malaria and rheumatism, so that he could not work in the fields, and lay about at home: this led to his eating much more with no exercise, and brought out nodules on his face. He does not admit any leprosy in his family, but says that two had tuberculosis, and two had syphilis, and

Case No. of patient.	Remarks of Patient or Relative.
9. (cont.)	everybody had malaria, and there were six lepers in the neighbourhood with whom he came in contact, and from whom he may have got the infection.
10.	He does not know for certain how he acquired leprosy. When he was in Mespot, at Shaib, he was in hospital with a factured pelvis after a motor accident: a nurse gave him another patient's medicine, and after that red patches of leprosy came out on his forehead and thigh and forearm: he has had tuberculosis of the lungs, and malaria, and thinks they may have had some effect in promoting leprosy. Generally, he thinks leprosy is caused by venereal disease, and by contact with a leper with discharges.
12.	He thinks that in his own case leprosy is due to his grandfather being a leper, to his having had a lot of malaria, and to having syphilis at 17 years of age.
14.	He thinks that his aunt being a leper in the house gave him the infection, and long hours of work sitting in the shop, not enough exercise, drinking brandy too much, having syphilis and gonorrhoea at 22 years, weakened him and made him more liable to the disease.
16.	He thinks that he caught leprosy from his wife, who died a leper at 30 years: cohabitation during menstrual periods may have made him more liable.
17.	He thinks that leprosy follows venereal diseases, especially syphilis, which he had at 20 years: malaria, poor food, and hard work also helped.
18.	He thinks living in a crowded house where a brother was a leper gave him the disease, and that loose living, drinking, and syphilis made him liable.
19.	He thinks that hard work and bad habits lead to leprosy, and that it is contagious when people live crowded together.
20.	He thinks that leprosy is inherited, as his uncle was a leper.
22.	He thinks that poor food and syphilis, and malaria caused his disease.

Case No. of patient.	Remarks of Patient or Relative.
23.	He thinks that leprosy may have been caught when he had bad worms, and scratched himself at the anus a lot, and his grandmother, who was a leper used to apply cloths to allay the itching. He thinks leprosy is contagious.
25.	She thinks leprosy is hereditary, and comes out in women at the time of first menstruation, as she thinks it did in her case.
26.	He thinks that leprosy is caught by sleeping in a room with lepers, as he used to sleep with his two brothers who were lepers.
27.	He thinks that too much sexual indulgence led to impotence, and his weakness caused leprosy. He thinks that bad food (decomposed food), and syphilis may cause leprosy.
28.	He thinks that insufficient food and weakness and anything which causes heat or fever in the body lead to leprosy.
30.	He thinks his leprosy was inherited from his father, but thinks that weakness due to lack of food may have helped.
32.	He thinks that leprosy is contagious, and that he caught it from the family of a fellow-mill-worker, with whom he lived: he thinks that heating foods, as meat, fish and toddy and lack of cold foods, as fruits, make a man more liable to leprosy.
34.	He thinks that too much malarial fever caused his leprosy.
35.	He thinks that syphilis at the age of 16 years caused his leprosy.
37.	He ascribes his first attack of leprosy to malarial fever.
40.	He thinks his disease was due to great weakness following an attack of cholera, and that he caught the infection from a sister in the house who was a leper.

Case No. of Patient.	Remarks of Patient or Relative.
41.	When on a pilgrimage, he slept near a leper in a 'dharmsala,' or rest-house, and thinks he may have caught the disease from him: on his return he fell ill with bad malaria and typhoid, which weakened him greatly, and caused the disease to appear, after a long time.
42.	He thinks that his disease was due to an attack of dysentery, which left him very weak.
46.	He thinks leprosy follows bad habits, as he had syphilis long before leprosy developed, and used to drink toddy.
47.	He had guinea-worm abscess of the foot for a long time, and as at a later date leprosy developed in that foot, he thinks the guinea--worm may have caused it.
49.	He thinks leprosy follows bad habits, such as excessive toddy-drinking, excessive sexual intercourse, and cohabitation during the menstrual periods.
50.	Her parents think that she inherited the disease as her grandmother and mother were lepers.
51.	He thinks that poor food, hard work and syphilis caused the disease in his case.
53.	He ascribes his leprosy to being weakened by cholera four years before the first sign of it: later he had syphilis, with burning and heat over the body, which was made worse when a native 'vaidya' gave him mercury treatment: the heat caused by the syphilis and the mercury, was, he thinks, what brought out a crop of nodules on face and body.
55.	She thinks that leprosy was caused in her by the onset of menstruation, and subsequent excessive blood loss at her periods.
59.	She thinks her leprosy was inherited, as her mother was a leper.

Case No. of Patient.	Remarks of Patients or of Relative.
63.	He thinks that as his brother was a leper, he got the disease from him, as he used to wear his shoes: his own leprosy first developed in the feet, and his brother had sores on his feet.
65.	He ascribes his disease to travelling with a leper in a bus, and to syphilis acquired at the age of 30.
68.	He ascribes his disease to travelling and living with another leper beggar, and to bad and scanty food.
74.	She thinks that because her sister had itch and leprosy, and she got itch from her, she must have got the leprosy too.
76.	She thinks that she caught leprosy from her husband.
78.	He ascribes his leprosy to an attack of small-pox. He thinks that anything causing heat in the body leads to leprosy.
81.	She thinks that her leprosy followed an attack of syphilis.
82.	She thinks that her disease was due to being born in Penang, where there was a lot of leprosy, and that she has always had scanty menses, had something to do with it.
92.	He thinks that he got the disease from a fellow-beggar who was a leper, and thinks that heating foods cause it to come out.
102.	He thinks that he got leprosy from a friend with whom he used to sleep, and change clothes, and that leprosy generally follows syphilis and heating foods.
110.	She thinks that having children causes leprosy.
112.	He thinks that leprosy follows loose living.
120.	She thinks that her leprosy was derived from her mother (hereditary), and was started by the onset of menstruation.

Case No. of Patient.	Remarks of Patient or Relative.
123.	He thinks that leprosy was perhaps acquired by his borrowing books from a fellow-student who turned out to be a leper.
125.	He thinks, that as a priest, he acquired leprosy in some of his visits to people's houses for marriages and other religious ceremonies.
132.	She thinks that her addiction to opium must have caused the disease.
137.	He thinks that leprosy is due to eating meat and fish against the rules of caste, and to sexual excess.
138.	He thinks that due to plague in the house, and a mild attack of it himself, his leprosy arose.
143.	He thinks that excessive toddy-drinking makes people liable to leprosy, because it heats the blood.
150.	He thinks that leaving home and living in a strange place makes one liable to leprosy.
164.	He thinks leprosy due to a hot climate, and heating food and disease, like syphilis.
173.	He ascribes leprosy in his own case and in that of others to acquired syphilis: he had a primary sore at 18 years of age.
174.	He ascribes his disease to gonorrhoea 2 years before, and to leprosy in the family.
177.	She thinks that a chronic patch of ringworm on the buttock developed into leprosy because her mother was a leper.
181.	This man, a very intelligent Brahmin, thinks that psoriasis caused leprosy in his case, as he had continual irritation from this which weakened his skin.
182.	She thinks that children are very liable to get leprosy, if, as in her case, there is leprosy in the mother.

There were many patients who were content merely to ascribe their leprosy to being 'written in the forehead' or to fate, but the above 65 cases were of those who thought about the matter. Their opinions may be classified as follows:-

Bad Habits Generally.

Opium addiction	1.
Toddy or brandy drinking	8.
Excessive cigarette smoking	1.
Excessive tea-drinking	1.
Loose living and dissipation	6.
Overwork	6.
Cohabitation during menstrual periods	3.
Lack of exercise	2.
Excessive sexual intercourse	3.

Dietary.

Lack of 'cold foods' (fruits)	1.
Eating 'heating foods', as fish and game)	10.
Eating beef.	2.
Eating preserved fish	2.
Eating milk and meat together	1.
Irregular meals	1.
Overeating	1.
Starvation	3.
Poor food.	5.

Diseases.

Syphilis	21.
Gonorrhoea	4.
Tuberculosis	5.
Malaria	9.
Rheumatism	1.
Worms	1.
Cholera	2.
Typhoid	1.
Dysentery	1.
Guinea-worm abscess	1.
Psoriasis	1.
Itch	1.
Ringworm	1.
Smallpox	1.
Plague	1.
Albuminuria of pregnancy and eclampsia	1.

Contacts blamed.

House and family	15.
Visiting lepers' houses	1.
Wearing Leper's clothes	1.
Wearing leper's shoes	1.
Using Leper's books	1.
Sitting with lepers	3.
Sleeping with lepers.	4.
That Leprosy is Hereditary.	7.

Menstrual

Onset of menses	3.
Excessive blood loss at menses	1.
Scanty menses.	1.

Miscellaneous.

Impotence	1.
Damp hot climate	2.
Being born in Penang	1.
Leaving home to live in a strange place	1.
'Heat in the body'	4.
Drinking 'heating medicine'	1.
Being given another patient's medicine	1.
Mercury treatment by a native practitioner	1.
Childbirth	1.
Motor accident	1.
Children especially liable to leprosy	1.

The striking opinions recorded above from those patients who think, makes it worth while adopting a rule in every case of leprosy that the patient should be hearkened to, if and when he is encouraged to talk about his disease. Valuable clues are given as to what predisposing factors should be attended to if the disease is to be fought, both from the personal and the public health point of view. Of course, many of the patients speculations will be wild and useless,

but it has been surprising what good sense has been displayed. Leprosy is a disease of long duration, giving time for reflection, and there is besides an accumulated ancient wisdom about leprosy, for it is a disease which has long been known to the peoples of India. They are naturally a reflective and philosophic people, and were they not cursed with inertia, might well have dealt with their leprosy problem themselves: as it is, the outsider would do well to consult the opinions of the intelligent Indians on the subject, and he will make use of the people's own thoughts as a starting point in his anti-leprosy work. After all, the opinions summarised above, may not be so greatly mistaken. Even such apparently bizarre causes as 'heating foods' and 'heat in the body' have truths behind them, namely that lepra reaction may well be precipitated by 'heat' or fever, and that foods which are 'heating' are vitamin-deprived in many cases, as opposed to 'cold foods,' which fresh and rich in vitamins, and hence likely to build up resistance against leprosy. So it is with cohabitation during menses: the truth behind that may well be that a man who does such a thing is likely to be one devitalised by other bad habits, and a ready prey to leprosy. Then at first signt it seems ridiculous to blame psoriasis as a cause of leprosy: but the man's skin being injured by such a disease, and, therefore, an easy portal of entry of the leprosy bacillus, does not seem a consideration that can be neglected lightly.

On the whole. then, these opinions of the patients about their disease, will repay being taken seriously, and being used with due discrimination.

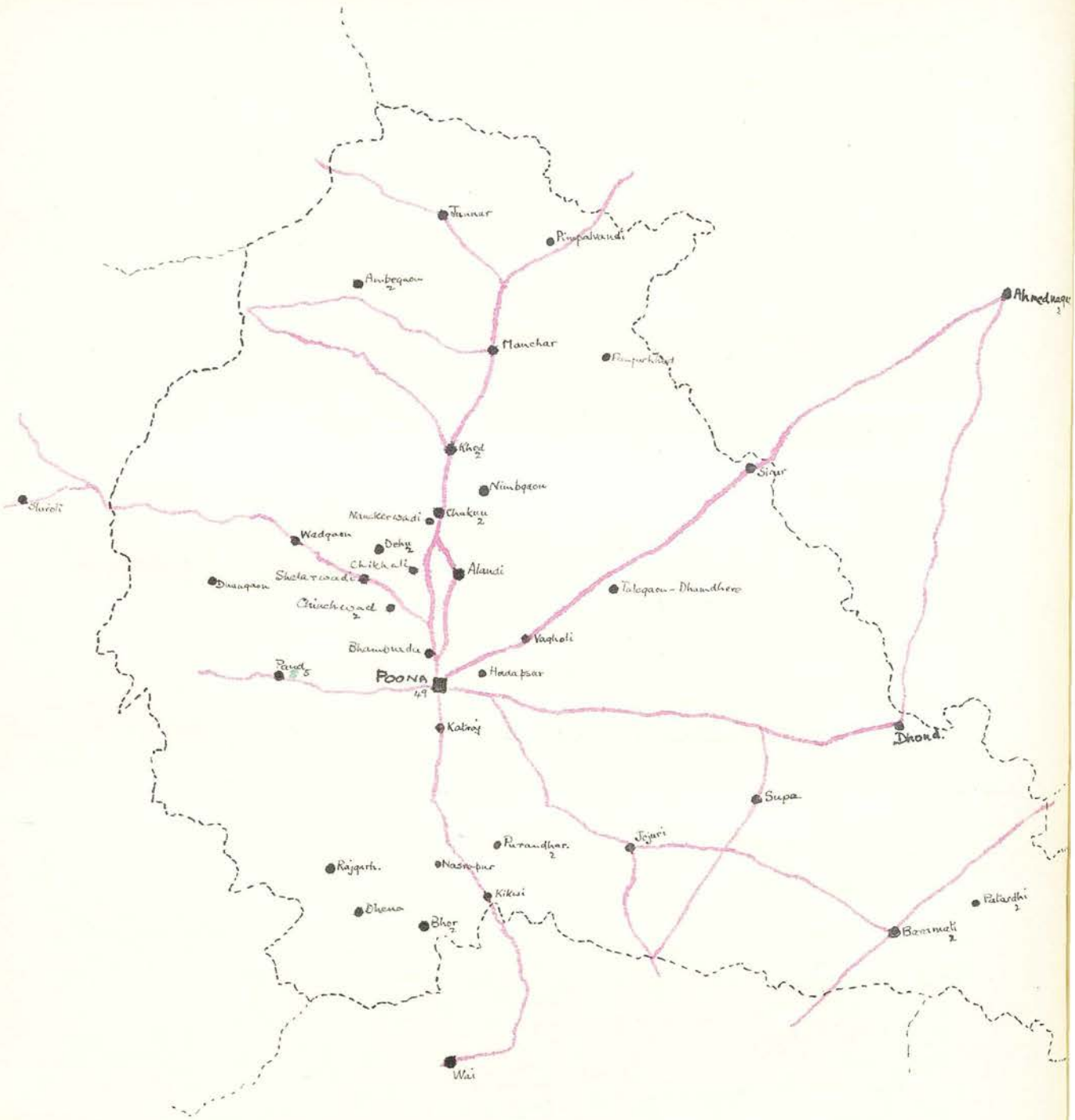
Places of Origin of the Lepers.

In Map 4. are shown the places of origin, as far as Poona District is concerned, of the lepers in the present survey. The Map shows the number who came from each place, and how the disease is scattered through the District, and in a striking way how leprosy incidence follows the main routes of communication.

There were lepers who came from neighbouring districts or even distant parts, and they are given below under their place of origin.

TABLE XXXVIII. PLACES OF ORIGIN OF LEPERS OUTSIDE POONA DISTRICT.

Penang	1.)	
Madras Presidency	5.)	
Calicut	1.)	
Goa	2.)	
Travancore	3.)	
Tanjore	1.)	
Bengal	1.)	
United Provinces	1.)	Far Places.
Nagpur	2.)	
Bijapur	2.)	
Berar	1.)	
Orissa	1.)	
Raichur	1.)	
Belgaum	9.)	
Ankleshwar	1.)	
N.W. Frontier	1.)	



Map 4. Map of Poona District.
The place names marked are those which are places of origin of the lepers in the series of 190 cases. The figures near the place names refer to the number of lepers in the series who came from that place. Villages with no number marked had only one leper representative in the series.
The remainder of the places of origin, which are outside or far from Poona District, are given elsewhere.
Main roads are marked in red.

TABLE XXXVIII. (cont.)

PLACES OF ORIGIN OF LEPERS OUTSIDE POONA DISTRICT.

Neighbouring or adjacent Districts.

Kolaba District	4.)	
Satara District	7.)	
Konkan	5.)	
Miraj State	1.)	
Kurundwad State	1.)	
Kolhapur District	4.)	Near Places.
Bombay	2.)	
Nizam's Dominions	13.)	
Thana District	7.)	
Ahmednagar District	5.)	
Sholapur District	9.)	
Phaltan	1.)	

This Table illustrates the surprising distances which lepers may travel away from their native place, and confirms how widespread leprosy is throughout India. It must be remembered also that infection must be carried in this way.

Summary of, and Conclusions from, the Survey of the Poona Cases of Leprosy.

It appears that:-

1. While high castes and communities do not escape leprosy, the heaviest incidence is on the lower castes and the outcastes;
2. Indian Christians, because of their origins being mainly the outcastes, may be included in the outcastes' leprosy figures, and more than the non-Christian outcastes do they put themselves under control when leprosy is suspected or develops.

3. The Mahommedans of Poona District have also a relatively high incidence of leprosy.

4. It is seen from the details given of the various castes and communities concerned, that those who have a higher standard of life have the lower incidence of leprosy.

5. When population figures by community, with known proportion of lepers, are studied, it is seen that the Europeans and Parsees, those with the highest standards of living, have no known lepers at all: but it is suspected that there is a large proportion of hidden lepers in the general population, comprising ordinary Hindu castes.

6. When the occupations of the lepers are grouped and studied, the variety and sweep of occupation, and the widespread nature of the spread of leprosy, becomes obvious.

7. Study of occupations of lepers also shows that leprosy is a disease of human intercourse, as most of the occupations are those which provide the most frequent and prolonged contacts with great numbers of people, or momentary contacts often repeated.

8. The civic value of an out-patient clinic under present Indian conditions is shown to lie in the fact that it reaches people who would otherwise be hidden and dangerous lepers, and takes steps to render them as innocuous as possible.

9. Lack of occupation seems to predispose to leprosy.

10. Agricultural pursuits do not, as one would expect, protect those who follow such in Poona District from the

10. (cont.) danger of leprosy, the reason lying in the very bad conditions of life which obtain.

11. Shopkeeping is an occupation which per se exposes a person to the danger of acquiring leprosy, chiefly because of the exceptionally numerous opportuniies for contact with lepers and the unhygienic conditions of Indian small shops.

12. Those engaged in transport services are in a strategic position for spreading and sustaining leprosy infection.

13. Schools and colleges are of importance because of the great numbers of susceptible persons who come into daily contact, for considerable periods of time, and the danger of a leper teacher or professor is outstanding.

14. Housewives with a limited circle of contacts are not absolved from the danger of leprosy, if there be a leper in the home.

15. Domestic servants have special opportunities for spreading leprosy, for they come into contact with young children.

16. Mill labour is a new factor in Indian life, and has taken its place prominently in the spread of leprosy: it is an occupation which predisposes to the disease.

17. Priests and shrines, prostitutes, mendicants, itinerant tradesmen, milkwomen, washermen, and several others may play an appreciable part in the spread of leprosy.

18. Sex incidence:- males are almost twice as many as females, the cause of this being chiefly environmental.

19. In childhood, the sex incidence was still overwhelmingly male, and the influence of puberty was indicated.

20. There was no evidence that boys develop leprosy more readily than girls: it is suggested that the explanation of the preponderance of males in childhood may be the greater value set on male children in India, leading to their being brought for treatment if leprosy develops, while girls might be ignored.

21. It was found that there is a distinct tendency for the disease to be severer in men than in women.

22. No significant difference was found as regards duration of life in male and female lepers.

23. Literacy and education have a valuable influence in encouraging lepers to come for treatment.

24. About 70%. of the lepers came first under observation in the age period 16-40 years: about 10%. in the age period 1-15. and 20%. in the period 41-70 years.

25. Study of the ages of probable onset of the disease revealed that 94%. of the cases originated in the first three decades of life, 50%. in the second decade, and only 5%. in the fourth and fifth decades of life.

26. A considerable amount of delay in seeking treatment and coming under control in leper cases in the District, is indicated. The factors responsible for this have been pointed out, and attention is drawn to the importance of the factor of the insufficiency of anti-leprosy resources.

27. From a study of the duration of leprosy before apprehension of the leper, it was found that only 11%. of the cases came under control within four years of the onset of the disease; 37%. came within 11-20 years, and 15%. within periods of over 20 years from the onset.

28. Civil state of the lepers:- over half were unmarried, as there is a valuable social prejudice which places obstacles in the way of a leper's marriage, and because of the onset of leprosy at the age when marriage is contracted.

29. In the case of widows and widowers the manifestations of the disease had become more numerous and severe following the loss of the other life partner, and consequent disruption of a well-habituated mode of life, change in economic position, deterioration in dietary and personal hygiene.

30. Association with lepers was admitted in 70% of the cases, and most of the acknowledged associations were with a parent, grandparent, sister or brother, uncle or aunt.

31. In 30%. of cases there was absolutely no history of association with lepers.

32. Casual contacts were described in a number of cases of which details are given.

33. The rarity of conjugal infection is shown by details of the five cases of conjugal associations, where it was possible to regard only two cases as examples of genuine intra-marital infection.

34. The coefficient of fertility of 43 leper women was found to be 5.9.;figures for non-leprous women are not available.

35. About 74%. of the lepers were found to be in a state of poverty in their previous life before apprehension , and only about 26%. in a state of economic independence. Poverty predisposes to leprosy.

36. There was but one example of a person who could be called rich among the lepers, and he had made no use of his wealth to elevate his standard of living and hygiene. Wealth is no protection against the disease, if divorced from intelligent use of it as a means to maintian good clean living.

37. A considerable degree of overcrowding and poor housing obtained among the lepers, and in the cities and villages there is also overcrowding of houses. Industrial tenements provide the worst examples of overcrowding of persons.

37(cont.) All overcrowding facilitates the spread of leprosy.

38. The standards of personal cleanliness among the general population and among the lepers was found to be low, and appreciably lower among the lepers. The causes were poverty and lack of knowledge of the rules of hygiene. A good degree of bodily cleanliness would tend to prevent the lodgment of the bacilli of leprosy.

39. About 80%. of the cases were found to be suffering from pyorrhoea or dental caries, 33%. with advanced disease of this type. The devitalising effect of sepsis and dyspepsia associated with it is considered to predipose to leprosy.

40. In an investigation of dietary it was found that the regular consumption of fresh milk, fruit or vegetables was rare; that the diets contained a few monotonous constituents, and that defects were invariable, there being a lack of good proteins, an excess of carbohydrates, a deficiency in salts, and a rarity of the use of vitamin-containing foods.

41. The composition of common Indian foodstuffs is given, and it is seen that a proper combination of the foods available would secure a better diet, but for the formidable obstacles of poverty, caste prejudice, ignorance, and the all too common

41. (cont.) adulteration of foods. In an ill-fed people disease can find easy lodgment.

42. Nephritis is common among lepers, but is regarded more as a sequela than as a contributing factor.

43. The details of five cases are given in which pre-existing or concurrent tuberculosis could be held to have predisposed to leprosy or to have aggravated the disease. Late incidence of tuberculosis is considered merely a terminal affection, and was not counted for this purpose. In one case, cure of the tuberculosis led to a marked improvement in the leprosy of the patient.

44. The Kahn Serological Test for syphilis was performed as soon as possible in all the lepers, and at such times as to avoid sources of error arising from leprosy in its acuter phases as far as possible, and from malaria, and it was concluded that about 24%. of the lepers were syphilised, as compared with a previously obtained figure of 15%. in general non-leper cases. Syphilis is considered to have a definite predisposing in- fluence on leprosy. It was found that anti-syphilitic treatment in positive cases led to marked improvement in the state of the leprosy.

45. It was found that 38%. of cases gave a history of malaria, and the devitalising influence of malaria was found to be a serious one in the direction of predisposing to an aggravating leprosy. It is pointed

45. (cont.) out that in future surveys great accuracy in assessing this factor of malaria will probably be obtained if the Melano-Precipitation Test is also used.

46. Examination of faecal specimens showed more than 50%. incidence of intestinal parasitism, revealing another important devitalising factor in Poona District.

47. Other predisposing diseases found include dysenteries and sprue, skin diseases, guinea-worm, enteric, and cholera.

48. In view of a possible relation between pellagra and leprosy, it was sought for, but no case was found among the lepers.

49. The results of an enquiry as to the patients' own opinion of their diseases are given in 65 cases. They blame as causes of their disease bad habits generally, points of dietary, diseases, contacts, heredity, menstrual influences and certain miscellaneous causes. On the whole their opinions are stimulating, enlightening and worthy of being used with discrimmination in the individual and public management of leprosy in Poona District.

50. Investigation of the places of origin of the lepers indicates that the disease is widely spread throughout Poona District and throughout India, and that it follows in its distribution the main lines of human communications. A Map of Poona District is given to show these points. (Map 4).

GENERAL SUMMARY AND CONCLUSIONS.

1. The estimate of D. E. MUIR of a million lepers in India first aroused interest in the subject of leprosy.

2. Medical charge of a general hospital in Poona and also of a leper asylum at Khondwa near Poona gave good facilities for the study of leprosy in the District.

3. Discovery of lepers admitted by mistake to the general hospital and lepers in the hospital and village dispensary often being seen, led to the founding of an out-patient leper clinic at the hospital, where the numbers attending soon reached 70.

4. The geographical features of Poona District have been described: It lies in the Tropics, in a plateau called the Deccan, mountainous to the west, flat to the east, at an altitude of 1800 feet.

5. The seasons and meteorological features of Poona District are described: there is a high average temperature.

6. Rainfall is heavy in the western parts, scanty and irregular to the east, where famine conditions often result.

7. The porous laterite soil of much of eastern Poona District corresponds with similar soil in Bengal where MUIR and SANTRA found an association with a high incidence of leprosy.

8. Poona is not a healthy part of India, as there is a high incidence of tropical endemic and epidemic diseases.

9. Ethnography of the District is dealt with and the people show to be of mixed Scytho-Dravidian origin.

10. The possibility that in the past leprosy first gained a footing among the Dravidian or aboriginal element is discussed.

11. The definition of caste is that of a group of families associated with a specific occupation and bound by certain rules.

12. Incidence of leprosy in Poona District is estimated to amount to 3,725 lepers, or a ratio of 369.0. per mille. Actual recorded figures are a fourth or fifth of this.

13. Of 4,112 villages, only 271 reported no lepers in their midst, in CHOKSY'S survey of Bombay Presidency, of which Poona District is one of the heavily affected parts.

14. By a Table of comparisons and Map 2., the correspondence pointed out by ROGERS as obtaining between leprosy incidence and high rainfall is shown not to apply very well to Poona District. Figure 1. shows what degree of correspondence there is.

15. The widespread endemicity of leprosy in India is demonstrated and some samples of recent expert surveys are tabulated.

16. A description is given of the world distribution of leprosy, and illustrated in Map 3. The Tropics bear the heaviest and widest incidence of leprosy, though colder parts are not exempt.

17. The number of lepers in the world is estimated

by ROGERS as 3,000,000.

18. The modern alertness as regards the leprosy problem is exemplified by the formation in 1931 of the International Leprosy Association.

19. A short historical sketch of leprosy relates the ancientness of the disease, the experiences of it in the Middle Ages, and the instructive history of the successful control of leprosy in Norway by systematic isolation of lepers, inter alia.

20. To obtain a picture of the background of general diseases prevalent in Poona District, 5,000. general case records from the N. M. Wadia Hospital were analysed, and the results presented in TABLE VI and FIGURE 4., and later discussed. A high incidence of tuberculosis, malaria, venereal diseases, dysenteries, helminthic diseases, myiasis, deficiency diseases and skin diseases, was obtained.

21. An account is given of the general conditions of life in Poona District. Life in villages is the most typical feature, there being 1-2 villages in every 'one minute square' of the Government Survey Maps.

22. The agricultural caste of Marathas are the strongest element in all village populations, but each village community is at its best a well-organised social unit comprised of many castes, each with its allotted duties.

23. The dwellings are small, insanitary, and overcrowded, and seldom well-built in any village. The outcastes live in wretched huts.

24. The system of conservancy in villages is the most primitive, and breeding of flies enormous.

25. The caste system in its rigid observance served to tend to restrict the spread of leprosy, but the modern loosening of caste restrictions, admixture and greater intercourse with outcastes, and the coming of the motor bus system to the villages, has led to greater spread of the disease.

26. The joint family system, under which several related families live in one house, is one of the most potent agencies in the spread of leprosy, chiefly because family contacts are preserved by it and given their best opportunity of conveying infection.

27. There is little or no attempt at segregation of lepers in the villages, but there is some restriction on marriage of lepers: it is a common practice for the presence of leprosy in a relative to be ignored through politeness.

28. Knowledge of the contagiousness of leprosy is widespread, but only the advanced cases are considered contagious: public opinion takes no account of early cases.

29. Child marriage is a debilitating factor in social life.

30. Widow remarriage is forbidden in the higher castes, a yound widow often enough being, as the result of economic pressure, driven to a life of prostitutism.

31. There is an appalling infant mortality in the villages; the village midwife is ignorant and insanitary; she believes in force as the main means of obstetric delivery.

32. Modern medical help is scanty or non-existent in most villages.

33. Sexual promiscuousness is to be regarded as normal in the social life.

34. The habit of pilgrimage is dear to the villagers. In Poona District they go to the shrines at Alandi and Pandharpur: much epidemic diseases result from these pilgrimages, and the shrines are notorious foci of leprosy, because lepers travel to the feet of the god in the hope of cure. The author counted 23 lepers in an hour at the Alandi festival.

35. The social necessity of men being shaved by the barber, and not by their ~~wen~~ own hands, helps in the spread of leprosy, as leper barbers have been observed in the act of shaving others, and non-leper barbers have been seen shaving lepers and healthy indiscriminately without cleansing their itensils.

36. The village milkman or milkwoman has been found to be leprous in some cases.

37. Toddy-drinking and drunkenness is common, especially amongst the lower castes and outcastes.

38. Opium is universally given to babies and opium

smoking and opium eating, also smoking of Indian hemp, are common habits among adults.

39. Nearly every agriculturalist is debt-ridden.

40. The average economic position of the villager is one of poverty. The average annual income per head in the village of Jategaon worked out at about Rs.33.12. 0. or £2:10: 0.

41. The land, which is the real and permanent capital, is practically unimproved, burdened with debt and progressive sub-divisions, and only in good districts where there is reliable rainfall does it support the people.

42. The larger part of Poona District, contains villages which suffer from a gamble in rain. Good seasons come 2 - 4 times in 10 years, and an average year leaves the village under-fed, more in debt than ever, and less capable than ever of obtaining, with the heavy population and present methods of cultivation, a real economic independence.

43. The price of grain dominates the cost of food in the villages, for 70%. to 80%. of the annual expenditure of food goes on grain. The grain chiefly eaten is bajri or jowar, and some pulses, rice, wheat, salt, chillies, oil, gul (sugar), and almost no milk or butter, are taken. The necessary food expenditure per annum for a man is Rs.42. (or £3: 3: 0). This diet is unsatisfactory, though customary.

44. Economic stress has driven many labourers to migrate to Bombay and Sholapur and other big centres to

work in the mills and industries. Most go only for a part of the year. The pressure on the land is reduced, but little direct financial gain accrues to the village, and the labourers return often with bad habits and diseases.

45. The soils of Poona District, and the three soil tracts are described, and the chemical analysis of black cotton soil given, and it is pointed out that soils have a relation to leprosy, because of their influence on the economic position of the people.

46. Only one limited tract of Poona District has rich soil: in the other two tracts, rainfall difficulties, and poor or porous soil, lead to poverty.

47. It is not enough to correlate leprosy incidence with high rainfall: it is necessary to consider the type of soil on which the rain falls. A soil which is not retentive of rain will not produce good crops, and will lead to a low economic standard among the people, and hence the resistance of the people to a disease such as leprosy will be low.

48. An account is given of the food grains of Poona District. The chief grains are the various millets: very little wheat and rice are grown.

49. Millet is not a perfect food grain, and approaches rice in its deficiencies, though not so bad as rice: wheat is almost a perfect food grain, but unfortunately is little used. The composition of these three grains is given.

50. Wheat and rice which are used are invariably inferior imported types.

51. The average millet diet is supplemented by pulses, such as dal, but they have the disadvantage of a low digestibility of albuminoids, and the proportion of undigested albuminoids rises when pulses predominate in the diet.

52. One of the pulses, the chick-pea or common gram, has a popular reputation in leprosy, and is sometimes ordered as the exclusive diet in that disease by the Indian 'vaidyas.'

53. Poona itself is to be regarded as an enlarged village, its general conditions of life corresponding to those in the outside villages.

54. The average inhabitant of Poona District, whether in the country village or the town, can only be described as semi-civilised.

55. A brief description is given of leprosy as a disease.

56. The classification of types of leprosy adopted since the International Conference on Leprosy at Manila in 1931, is described in its main features.

57. A study of the opinions of other workers, and their experiments, on the subject of the predisposing causes of leprosy, is now commenced.

58. E. MUIR points out that the leprosy bacillus will not grow in the healthy human body, and the soil must be prepared for its growth by some predisposing cause or other,

and he summarises these under the headings of Various Debilitating Diseases, Dietary Defects, Pernicious Habits, Certain Physiological Conditions, Climatic Conditions, Insanitary Surroundings and the Mental Attitude.

59. The JONATHAN HUTCHINSON fish theory is discussed and held to be superseded as a theory of the prime cause of leprosy, though possibly it may be allowed to rank among the predisposing causes.

60. The theory of COOK, that leprosy is a deficiency disease, is quoted and discussed, and held also to be an exaltation of a predisposing cause into a prime cause of leprosy.

61. COOK also raised the question of a connection between pellagra and leprosy, pointing out the nervous nature and the deficient dietaries commonly found in both, and the outbreak of pellagra among lepers in Dichpali in India, and in Korea, seems to support the existence of some connection between the two diseases. LOWE found in his Dichpali cases that the diet of the leper pellagra patients was markedly deficient, but the mystery of the connection between the two diseases, if any, is still unexplained.

62. A valuable survey of India made by MUIR and SANTRA in 1932, revealed as factors predisposing to leprosy or the spread of leprosy the following:- famine conditions: a people being in a state of transition from one mode of life to another: tracts of high filariasis endemicity:

mountain villages which suffer privation in the winter months: highly industrialized areas: low stage of civilisation, the semi-aboriginal especially being bery liable to leprosy: laxity of caste rules: sexual and other promiscuousness: dietary errors as a major factor: no racial predisposition to leprosy: diseases such as malaria, the dysenteries, encylostomiasis, filariasis, typhoid and influenza: the lower classes seem more liable.

63. The facts about the lack of milk in the dietary are substantiated from the Report of the ROYAL COMMISSION ON AGRICULTURE IN INDIA. In Bombay Presidency, the supply of milk, (not the consumption, which must be much less) is estimated at less than 2 oz. per head daily.

64. As regards the class incidence in leprosy, the experience in the United States is that "the social status of the patients is a cross-section of the normal populace as regards education, wealth and culture."

66. I. KERR emphasises that in women puberty, pregnancy, childbirth, lactation, menstruation and catamenia predispose to, or aggravate leprosy, and suggests a deficiency of the blood calcium incidental to these conditions as the critical factor: she thinks that syphilis, also a powerful predisposing cause, likewise depletes the blood calcium.

67. LOWE draws attention to the similarities in sex incidence in tuberculosis and leprosy.

68. BRAUL traces the onset of leprosy in eight cases to shock.

69. SANTRA, from his survey of Puri District in India, shows how overcrowding : caste laxity, floods and famines, poverty and low diet predisposes to leprosy there.

70. Oral sepsis was found by I. KERR to predispose to leprosy.

71.. In Cooch Behar, devitalising diet, worms, pyorrhoea, and the damp hot climate were incriminated.

72. U. P. GUPTA, suspects congestion at the religious festival of Jagganath at Puri, filariasis, malaria, dysentery, cholera, syphilis and gonorrhoea, skin diseases, poor diet, ignorance and lack of proper knowledge of hygiene, as the chief factors.

73. B. N. BOSE, blames callousness, ignorance of leprosy and of hygiene, smoking the same pipe as lepers, lack of houses and overcrowding, defective diet, scarcity of water for bathing and drinking, malaria, hookworm, and venereal diseases as the findings in his survey in Manbhum District.

74. In Cuttack District, U. GUPTA, found a greater incidence of leprosy in the lower castes, due to their greater degree of promiscuous intercourse: a low economic standard , poor quality diet, malaria, filariasis, syphilis, and intestinal worms were other factors.

75. JAIKARIA found in Kangra District ignorance and lack of proper sanitary knowledge, defective diet, preval-

ence of bowel complaints, venereal diseases, malaria, rickets, osteomalacia, and goitre as the predisposing factors.

76. In Burdwan District, GHOSH mentions poor soil deficient rainfall as depressing the economic state of the people, high atmospheric humidity, kala azar, fevers, intestinal diseases, venereal diseases, and defective diet, a continuous flow of immigration from areas of high leprosy endemicity, an increasing density of the population, uncleanliness, and ignorance.

77. CHATTERJI found in Midnapore that poor soil breeds lepers. He points out the striking difference in that respect between the two halves of the District. The people of the poor half eat poor diet and decomposing food, and migrate in numbers to industrial areas, where they contract venereal disease, and leprosy itself. Malaria, dysentery, typhoid, and pyorrhoea are common among them.

78. In the Central Provinces, MUKHERJI, found a high incidence of leprosy, in certain parts, due to hot moist climate, poor soil, low stage of civilisation, personal uncleanliness, insanitary surroundings, defective diet of stale rice, with no fresh foods, high incidence in Chambar caste who eat carrion, laxity of morals and great promiscuousness, venereal diseases are rife, amoebiasis, hookworm intestinal diseases, filariasis, absence of any fear of leprosy.

79. In Muzaffapur District, GUPTA incriminates

malaria, kala azar, round worm, hookworm, overwork on a poor diet, and suspects 'khesari,' as it is commonly eaten and leads to lathyrism.

80. In Tippera District, GHOSH has shown how high leprosy incidence there results from trade intercommunication with highly endemic centres, and records the lack of restriction of intercourse with highly infectious lepers, ignorance, high humidity, increasing density and overcrowding of the population, lack of knowledge of hygiene, as the chief predisposing factors.

81. In Purushothampur, PAL cites as underlying causes a hookworm infection of 80%. venereal diseases, laxity of morals, general debility and overwork, poor food and conditions of poverty and stress.

82. In Bhutan, MACDONALD SMITH, incriminates overcrowding as the explanation of the great prevalence of leprosy there. There is an unfortunate tax on houses.

83. In Kulu, JAIKARIA found prevalence of malaria, dysentery, round worms, goitre, syphilis, and gonorrhoea, ignorance and lack of knowledge of hygiene, lack of restriction of intercourse with infectious lepers.

84. In HLEGU in Burma, SANTRA records that some of the patients blamed taking mercurial preparations as the cause of their leprosy: some ascribed it to irregularities of menstruation. The native doctors of Burma incriminate eating fish, crabs, and deer. SANTRA suggests that close intermarriage, and excessive courtesy of the

Burmans which prevents restriction of intercourse with lepers, are definite predisposing causes.

85. FUMIO HAYASHI, after a visit to India, agreed to the reasonablness of regarding syphilis as an important predisposing cause in leprosy, and of treating by anti-syphilitic remedies all lepers who have a positive serological reaction for syphilis, in spite of the possibility that some positive reactions in lepers are not caused by syphilis. He commented on the frequency with which he saw trachoma in lepers, and the rarity of it in lepers in Japan.

86. The most typical attitude to leprosy in India is one of apathy and inertia.

87. The experience of workers in countries other than India have been summarised in a Table (XII), from which the ensuing list is compiled:- (The figures refer to the number of times the predisposing cause was incriminated in the Table).

Civil Conditions.
Frequent migrations 1.
Frequent immigration from endemic areas of leprosy. 1.
Mass movements of population, 2.
Dense population, 3.
Anarchy, 1.
Civil disturbances and misrule, 2.
Poverty, 2.
Depressed economic conditions, 2.
Greatly mixed races, 2.
Presence of susceptible aboriginal tribes, 1.
Racial lack of resistance, 1.
Family infection common, 1.
Strain and hard life, 2.
Numerous leper beggars, 1.
Lack of segregation of lepers, 4.
Closing of leper hospitals before leprosy was eradicated, 1.

Devitalising Diseases.

Yaws, 5.
Craw-craw, 1.
Syphilis, 8.
Gonorrhoea, 4.
Skin Diseases, 1.
Scabies, 4.
Eye diseases, 1.
Dental caries, 2.
Sleeping sickness, 2.
Pulmonary tuberculosis, 2.
Chest diseases, 1.
Bilharzia, 3.
Ankylostomiasis, 6.
Ascariasis, 1.
Tapeworm, 1.
Malaria, 6.
Filariasis, 1.
Nephritis, 1.
Gastro-Intestinal diseases, 1.
Bacillary dysentery, 1.
Dengue, 1.
Low diffusible calcium of the blood serum, 1.

Climatic and Geographical and Topographical Factors.

Living in low-lying areas, 1.
Living in the littoral, 1.
Living in water-logged and ill-drained areas, 1.
Living in towns, 1.
Living in endemic areas in the Tropics, 1.
Absence of leprosy in desert belt and heavy incidence in the equatorial belt, 1.
High temperature and humidity, 4.
Little relation to humidity, 1.
Heavy rainfall, 2.

Mental Attitude.

Fatalism, 1.
Extreme fear of leprosy, 1.
Lack of fear of leprosy, 1.
Ignorance of leprosy, 3.
Conservatism, 1.
Inertia, 1.
Apathy, 1.

Dietetic Factors.

Eating putrescent fish, 2.
Defective diet, 5.
Starvation, 3.
Famine, 3.
Prolonged under-nourishment in youth, 1.
Droughts, 1.
Floods, 1.

Habits.

Dirtiness, 4.
Bad hygiene, 3.
Intermarriage, 1.
Concubinage on a large scale, 1.
Cannibalism, 1.
Laziness, 1.
Betel-chewing, 1.
Excessive smoking, 1.
Interchange of pipes, 1.
Interchange of drinking-vessels, 1.

Soil.

Porous thin soil on coral, 1.

Childhood and Youth.

Greater susceptibility in childhood and youth, 3.

Accidental Inoculation.

By hypodermic needle previously used on a leper, 1.
By a wound from an instrument at a surgical operation on a leper, 1.

Rat Leprosy.

Inoculable from rat to rat by juxtaposition of an infected ulcer and a recent wound, by contamination of flies, and predisposed to by secondary infections in the rat, bad nutrition and bad hygiene, 1.

87. Present knowledge regarding the transmission of leprosy is discussed. It is agreed now that leprosy is a communicable disease, and the following points represent the sum of present knowledge:-

(a) Nodular leprosy is far more infective than nerve leprosy.

(b) During the stage of febrile reaction, infectivity increases considerably.

(c) Variations in susceptibility with age modify the transmission of leprosy. The young up to the age of 30. years are especially susceptible, and it is necessary to remove the children of leprous parents at birth.

(d) Infection is derived from a blood relation with striking frequency.

(e) Conjugal or house infections occur in 2.5%. of persons exposed to infection.

(f) Infection follows the degree of contact and closeness of human intercourse.

(g) The degree of infectivity in leprosy is low.

(h) Transmission by sexual intercourse is possible.

(i) Room and bed infection are common.

(j) Infected household servants are transmitters of leprosy.

(k) Infection of attendants on lepers is not common.

(l) Close contact with lepers outside the home is also dangerous.

(m) Infection from wet nurses of children is recorded.

(n) Infection by inoculation through the skin, by abrasions or wounds, is recorded.

(o) Transmission of leprosy by insects is not proved: house flies are the most suspect.

88. Following in the main the suggestions of the Report of the LEONARD WOOD MEMORIAL CONFERENCE ON LEPROSY, the author has personally surveyed 190 cases of leprosy in Poona, the summary and conclusions of which have been presented already. (Pages 178-186.)

CONCLUSION.

The subject of leprosy is a large one, and it is not proposed to carry this study of it further at this time. On return to India, the author hopes to continue the study of leprosy in Poona District, and to make an effort to place the control of the disease on a better footing. From study of the land and the people, their habits, their diseases, their mental equipment, their means of subsistence, and the public resources available, some more satisfactory method of control of leprosy may emerge. The present position is in the highest degree unsatisfactory. For thousands of lepers in the District there exists one Leper Asylum and a few clinics, and only some 300 lepers at most are under control.

ROGERS, 1931,(159) animadverts against compulsory segregation as failing to stamp out or materially to reduce the prevalence of leprosy among backward races within a reasonable time. MUIR, 1932,(160), believes that segregation in India could not possibly be carried out for frightening patients "underground," and because of lack of finance. On the other hand, we have seen the success which

has attended strict measures of isolation and control in Norway, (40) Withoug going further into the matter, it is enough to emphasise that in Poona District segregation of lepers has never been tried, strictly and efficiently, nor probably in any part of India, and one would be better convinced of the truth of the opinions of ROGERS and of MUIR in this respect, <u>if segregation or isolation had been tried.</u> There is at least enough evidence, of its value to expect that one should "move heaven and earth," at least to give it a proper trial. Further, the problem of leprosy looks staggering when considered for the whole of India, but if one adopts a District as the unit of campaign, it does not look so hopeless. If one imagines the position of a state in India, small, independent, caring for no man, which determines to essay the proper control of leprosy within its borders, one can visualise even success. Some degree of ruthlessness in the control of the liberty of the subject, and in the control of frontiers, and in the enforcement of anti-leprosy laws, and in the expenditure of money, would be demanded, but Norway has taught us that it can be done. Of course there are many difficulties, but in India it has never been tried.

In Poona District we have not even enough accommodation for the ordinary care of the lepers who come to us. We are forced to treat 70 lepers in a clinic, because there is

no room for them in a proper leper hospital. This position at least should be remedied, and if a modern leper hospital to contain 1,000 persons were built in Poona District, one is convinced that it would find occupants.

Finally, one would like to suggest the anti-leprosy measures which need to be developed in Poona District:-

(a) All measures to spread general education and literacy.

(b) Specific education about leprosy, among the people and among the existing medical personnel.

(c) Increase in medical anti-leprosy staff.

(d) Visiting every town and village in the District, and enumeration of lepers by careful and even bacteriological methods of diagnosis; at the same time carrying out propaganda and instituting treatment centres.

(e) Building adequate leper hospitals: lepers require a great deal of ordinary medical care which at present is not possible, and there is a vast field in leper sugery alone.

(f) Concerting of measures which aim at the 'bonification' of the people, as improvement of agriculture, housing, water supply, control of diseases, spread of knowledge of dietary and the like.

(g) If enough courage and enough money be obtainable, securing in the end strict control and isolation of all known lepers.

B I B L I O G R A P H Y

BIBLIOGRAPHY

1. MUIR, E. & SANTRA, I. — Indian Journal of Medical Research. XX: 421-434. Oct., 1932.

2. — Imperial Gazetteer of India. 1908. Vol. XX, p. 165.

3. — The India Office List. 1934. P. 9.

4. — Imperial Gazetteer of India. 1908. Vol. XX, p. 166.

5. ROGERS, SIR LEONARD — Trans. Roy. Soc. Trop. Med. & Hyg. 1923. XVI, p. 440.

6. — Imperial Gazetteer of India. 1908. Vol. XX, p. 177.

7. MUIR, E. & SANTRA, I. — Indian Journal of Medical Research. XX, p. 421-434. Oct., 1932.

8. — Imperial Gazetteer of India. 1908. Vol. XX, p. 181.

9. — The India Office List. 1934. P. 10.

10. SANTRA, I. — "Leprosy in India." Oct., 1933, p. 180. Published quarterly by the Indian Council of the Brit: Empire Leprosy Relief Association.

11. CILENTO, R. W. & NORTH, E. A. — Med. Journal of Australia. 2. P. 767-775. Dec., 1931.

12. CHOKSY, SIR NASARVANJI — "Leprosy in India." April, 1930. P. 41.

13. PATIL, R. B. P. C. — Bulletin No. 109 of Meterological Dept. of Gov. of India. 1921.

14. ROGERS, SIR LEONARD — Recent advances in Tropical Med. 1928. P. 323.

15. GHOSH, B. N. — Leprosy in the Cooch Behar State, 1930. "Leprosy in India." April, 1931.

16. GHOSH, B. N. — Leprosy Survey in the Howrah District. "Leprosy in India." Jan., 1931.

17. GUPTA, U. — Leprosy Survey in Cuttack District. "Leprosy in India." Oct., 1932.

18. JAIKARIA, S. S. — Leprosy in Tehsil Kangra. "Leprosy in India." Oct., 1932.

19. CHATTERJI, K. R. — Report on Leprosy Survey at Salbani, Midnapore, Bengal. "Leprosy in India." Jan., 1932.

20. MUIR, E. — The Early Diagnosis of Leprosy. "Leprosy in India." Oct., 1930.

21. SANTRA, I. — Report on Leprosy Survey at Villupuram, S. Arcot, Madras Presidency. "Leprosy in India." Oct., 1929.

22. LOWE, J. — Epidemiology of Leprosy in Hyderabad (Deccan), India. International Journ. of Leprosy, Vol. I, No. 1, Jan., 1933, p. 21.

23. COCHRANE, R. G. — Leprosy in Ceylon. "Leprosy Review", Vol. IV, No. 2. April, 1933. P. 64.

24. ROGERS, SIR LEONARD — The World Incidence of Leprosy. Trans. Roy. Soc. Trop. Med. & Hyg., 1923. Vol. XVI, p. 455. Map 2.

25. IBID — Ibid. P. 449.

26. ROGERS, SIR LEONARD — The Croonian Lectures on Leprosy Researches. Annals of Trop. Med. & Parasit., 1924. Vol. XVIII, p. 273.

27. — REPORT OF THE LEONARD WOOD MEMORIAL CONFERENCE on Leprosy, Manila, 1931. Published by the Bureau of Science of the Philippine Government, Manila. Vol. XLIV, No. 4, April, 1931.

28. STITT — Section on leprosy. "Tropical Diseases," 1929.

29. GARRISON, F. H. — "History of Medicine." 1917. P. 56.

30. IBID — Ibid. P. 57.

31. ROGERS, SIR LEONARD — Spread, Mode of Infection, and Prophylaxis of Leprosy. British Med. Journ. 1922. I. P. 987.

32. STITT — "Tropical Diseases." 1929.

33. ROGERS, SIR LEONARD — Annals of Trop. Med. & Parasit. 1924. Vol. XVIII, P. 267.

34. GARRISON, F. H. — "History of Medicine," 1917. P. 163.

35. IBID — Ibid. P. 300.

36. IBID — Ibid. P. 171.

37. IBID — Ibid. P. 228.

38. ROGERS, SIR LEONARD — Annals of Trop. Med. & Parasit. 1924. Vol. XVIII, p. 268.

39. RATTRAY, P. — Leprosy in Scotland. Caledonian Medical Journ. 14: April, 1931. Pp. 339-354.

40. LIE, H. P. — Leprosy Work in Norway. "Leprosy Review." Vol. I. No. 2, April, 1930. P. 8.

41. IBID — Ibid. P. 14.

42. BURNET — League of Nations Report, No. C.H. 887. "Report on the study tour of the Secretary of the Leprosy Commission in Europe, South America, and the Far East."

43. ROGERS, SIR LEONARD — Annals of Trop. Med. & Parasit. 1924. Vol. XVIII, p. 271.

44. MANIFOLD, J. A. & de MONTE, A. J. — 1928. Report on Investigation of Dysentery and Diarrhoea in Poona. Indian Journ. of Med. Research. Vol. XV, No. 3, p. 601.

45. MANSON-BAHR — "Manson's Tropical Diseases," 1929. P. 388. Cassell & Co., London.

46. TANDY, COLONEL T. E., R.E. — Maps Nos. 47F/N.E. and 47F/S.E., 1928. Pub. W. & A. K. Johnson, 12, Queen Street, Edinburgh.

47. DUBOIS, ABBE J. A. — "Hindu Manners and Customs." Translated by Beauchamp, P. 320.

48. MANN & KANITKAR — "Land and Labour in a Deccan Village." University Economics Series, Bombay, Study, No. 2.

49. LOWE, J. — International Journal of Leprosy. Vol. I, No. 1. Jan., 1933, p. 24.

50. BRAY, G. W. — Quoted by MANSON-BAHR, "Manson's Tropical Diseases," 1929. P. 337. Cassell & Co., London.

51. SAHASRABUDDHE, D. L. — "Soils of the Bombay Presidency." Bulletin No. 160 of 1929, pp. 40-52. Dept. of Agriculture, Bombay.

52. CHURCH, A. H. — "Food Grains of India," 1930. P. 34. Pub: Chapman & Hall.

53. MANSON-BAHR — "Manson's Tropical Diseases." 1929. P. 465.

54. ROGERS & MUIR — "Leprosy," 1925, p. 185. John Wright & Sons, Ltd., Bristol.

55. STITT — "Tropical Diseases," 1929.

56. MUIR, E. — "Leprosy, Diagnosis, Treatment and Prevention," 5th edition. Pub: Indian Council of British Empire Leprosy Relief Association, New Delhi.

57. COCHRANE, R. G. — "Leprosy, Symptoms, Diagnosis, Treatment, and Prevention." 2nd edition: Pub: Brit. Emp. Leprosy Relief Association, 29, Dorset Square, London.

58. REPORT OF THE LEONARD WOOD MEMORIAL CONFERENCE on Leprosy, Manila, 1931, pp. 457, 458. Published by the Bureau of Science of the Philippine Govt., Manila, Vol. XLIV, No. 4, April, 1931.

59. MUIR, E. "Leprosy, Diagnosis, Treatment, and Prevention," 5th edition. Pub: Indian Council of the British Empire Leprosy Relief Association, New Delhi. P. 20.

60. ROGERS & MUIR "Leprosy," 1925. P. 61. Pub: John Wright & Sons, Ltd., Bristol.

61. COOK, L. "Leprosy Review," 4: pp. 166-173. October, 1933.

62. LOWE, J. Indian Medical Gazette, 66: pp. 491-500. Sept., 1931.

63. WILSON, R. M. Pellagra in Korea, China Medical Journal, XXXIX, 1925, p. 661.

63a. WILSON, R. M. Do. XLVII, 1933, p. 287.

64. MUIR, E. & SANTRA. I. Indian Journal of Medical Research, XX, Oct., 1932: pp. 421-434.

65. EAST INDIA CENSUS, 1931. Abstract of Tables, p. 16: Pub: by H.M. Stationery Office, Edinburgh, 120, George Street: 1932. Cmd. 4194.

66. REPORT OF THE ROYAL COMMISSION ON AGRICULTURE IN INDIA, 1928: Pub. H.M. Stationery Office, Adastral House, Kingsway, London.

67. MUIR, E. "Leprosy in India," Jan., 1933. P. 32.

68. MUIR, E. Indian Journ. of Med. Research. XV, No. 1: and idem No. 2. 1927.

69. HOPKINS, R. & DENNEY, O. E. Leprosy in the U. S. Journal of the American Medical Association. XCII: pp. 191-198. Jan. 19, 1929.

70. LOWE, J. A Note on Leprosy and Sex. "Leprosy in India," Oct., 1932.

71. LOWE, J. The Sex Incidence of Leprosy. International Journal of Leprosy, Vol. II, No. 1: Jan.-March, 1934, p. 71. Pub: by International Leprosy Association, Manila, Philippine Is.

72. KERR, I. Leprosy in Women. "Leprosy Review," III: pp. 165-170: October, 1932.

73. BRAUL, J. E. Archiv. f. Dermat. u. Syph: 159, p. 308, 1930.

74. SANTRA, I. Reports on Leprosy Survey. "Leprosy in India," Vol. II, No. 2. April, 1930.

75. KERR, I. Oral Sepsis as a Predisposing Cause of Leprosy. "Leprosy in India," Vol. II, No. 1, Jan., 1930.

76. GHOSH, B. N. — Leprosy in the Cooch Behar State. "Leprosy in India," No. 2, April, 1931.

77. GUPTA, U. P. — Leprosy Survey in Puri District. "Leprosy in India," Jan., 1931.

78. BOSE, B. N. — Report of Leprosy Survey at Burrabazar, District Manbhum. "Leprosy in India," Jan., 1931.

79. GUPTA, U. — Leprosy Survey in Cuttack District. "Leprosy in India," Oct., 1932.

80. JAIKARIA, S. S. — Leprosy in Tehsil Kangra. "Leprosy in India," Oct., 1932.

81. GHOSH, B. N. — Leprosy in the Burdwan District. "Leprosy in India," Oct., 1932.

82. CHATTERJI, K. R. — Report on Leprosy Survey at Salbani, Midnapore. "Leprosy in India," Jan., 1932.

83. MUKHERJI, D. N. — Antileprosy Activities in the Chhatisgarh Division, C.P. "Leprosy in India," Oct., 1930.

84. GUPTA, U. P. — Survey of Muzaffapur District. "Leprosy in India," Oct., 1930.

85. MANSON-BAHR, P. — "Manson's Tropical Diseases," 1929, p. 606.

86. GHOSH, B. N. — Leprosy in the Tippera District. "Leprosy in India," July, 1931.

87. PAL, K. — Annual Administration Report of the District Board Leprosy Clinic, Purushothampur. "Leprosy in India," Oct., 1931.

88. SMITH, J. A. MACDONALD — Leprosy in Bhutan. "Leprosy in India," Jan., 1933.

89. JAIKARIA, S. S. — Leprosy in Tehsil Kulu. "Leprosy in India," April, 1933, p.102.

90. SANTRA, I. — Leprosy Survey Work in the Hlegu Township, District Insein, Burma. "Leprosy in India," April, 1933, p. 106.

91. HAYASHI, FUMIO — Impressions of Anti-Leprosy Work in India. "Leprosy in India," July, 1933, p. 153.

92. SANTRA, I. — Anti-Leprosy Work in India. "Leprosy Review," Vol. II, No. 1, Jan., 1931, p. 27.

93. DIXEY, M. B. D. — Work in the Gold Coast. "Leprosy Review," Vol. I, No. 2, April, 1930, p. 29.

94. MOISER, B. — A Medical Survey in Southern Rhodesia. "Leprosy Review," Vol. II, No. 2, April, 1931, p. 54.

95. ROSE, F. G. — The Leprosy Problem in British Guiana. "Leprosy Review," Vol. II, No. 2, April, 1931, p. 55.

96. COCHRANE, R. G. — Leprosy in Uganda. "Leprosy Review," Vol. II, No. 2, April, 1931, pp. 59-60.

97. STRACHAN, P. D. — Leprosy Policy in Basutoland. "Leprosy Review," Vol. II, No. 2, April, 1931, p. 65.

98. MAXWELL, J. L. — The Menace of Leprosy in Manchuria. "Leprosy Review," Vol. II, No. 1, Jan., 1931, pp. 5-7.

99. MURRAY, JANET — Work Among Tanganyika's Lepers. "Leprosy Review," Vol. II, No. 1, Jan., 1931, p. 16.

100. COCHRANE, R. G. — Leprosy in East & Central Africa. "Leprosy Review," Vol. II, No. 1, Jan., 1931, pp. 22-23.

101. BJARNHJEDINSSON, SAM — Leprosy in Iceland. "Leprosy Review," Vol. II, No. 1, Jan., 1931, p. 25.

102. RODRIGUEZ, JOSE — Cebu and its Leprosy Problem. "Leprosy Review," Vol. II, No. 3, July, 1931, pp. 96-98.

103. WAYSON, N. E. — Leprosy in Hawaii. "Leprosy Review," Vol. III, No. 1, Jan., 1932, pp. 10-12.

104. ROBERTSON, RUSSELL L. Garkida Leprosy Colony. "Leprosy Review," Vol. III, No. 2, April, 1932, p. 53.

105. WILSON, R. M. Leprosy in Korea. "Leprosy Review," Vol. III, No. 2, April, 1932, p. 81.

106. FIDANZA, E. P. Leprosy in Argentine. "Leprosy Review," Vol. III, No. 3, July, 1932, p. 109.

107. MAYBURY, L. M. Leprosy in the British Solomon Is. "Leprosy Review," Vol. III, No. 4, Oct., 1932, p. 158.

108. WALLACE, C. A. Leprosy in Ukuguru District, Tanganyika. "Leprosy Review," Vol. III, No. 4, Oct., 1932, p. 159.

109. KELLERSBERGER, E. R. Some Facts About Leprosy in the Katanga, Belgian Congo. "Leprosy Review," Vol. III, No. 4, Oct., 1932, p. 164.

110. LAMPE, P. H. J. Het Geneeskundig Tijdschrift voor Nederlandsch Indie: afl. 15. Deel 72, 1932, pp. 946-952.

111. CANAAN, T. Ridding Palestine of Leprosy. "Leprosy Review," Vol. IV, No. 3, July, 1933, p. 95.

112. RYRIE, GORDON A. Leprosy in the Federated Malay States. "Leprosy Review," Vol. IV, No. 4, Oct., 1933, pp. 138-139.

113. SHARP, L. E. S. Leprosy in Uganda. "Leprosy Review," Vol. IV, No. 4, Oct., 1933, p. 151.

114. PHIPSON, E. S. Aden and its Leprosy Problem. "Leprosy Review," Vol. V, No. 1, Jan., 1934, pp. 4-6.

115. BROWN, JAMES A. K. The Leprosy Colony, Uzuakoli, South Nigeria. "Leprosy Review," Vol. V, No. 1, Jan., 1934, p. 38.

116. COCHRANE, R. G. "Leprosy in the Far East," 1929, p. 18. Pub: World Dominion Press, 1, Tudor Street, London, E.C.4.

117. FOWLER, H. "Leprosy in the Far East," 1929, p. 22.

118. COCHRANE, R. G. "Leprosy in the Far East," 1929, p. 52.

119. MARCHOUX, E. Un cas d'inoculation accidentelle du bacille de Hansen en pays non lepreux. International Journal of Leprosy, Vol. II, No. 1, Jan.-March, 1934, pp. 1-4.

120. LICHTWARDT, H. A. Leprosy in Afghanistan. International Journal of Leprosy, Vol. II, No. 1, Jan-March, 1934, pp. 75-76.

121. MARCHOUX, E. The Leprosy of Rats. International Journ. of Lepr., Vol. II, No. 1, Jan.-March, 1934, pp. 88-95.

122. KEIL, E. G. — The Importance of Nutrition in the Prevention and Cure of Leprosy. Internat. Journ. of Lepr., Vol. I, No. 4, Oct., 1933, pp. 393-397.

123. DENNEY, O. E. — Notes on the Work of the U.S. National Leper Home. Internat. Journ. of Lepr., Vol. I, No. 4, Oct., 1933, pp. 399-405.

124. CEDERCREUTZ, AXEL — Leprosy in Finland. Internat. Journ. of Lepr., Vol. I, No. 2, April, 1933, p. 199.

125. LIE, H. P. — Why is Leprosy Decreasing in Norway? A Paper read at the 7th meeting of the Scandinavian Dermatologists' Society, held at Oslo, May, 1928. Trans. Roy. Soc. Trop. Med. & Hyg., XXII, 1929, p. 357.

126. DE LANGEN, C. D. — Superinfection with Leprosy. Transactions of the 8th (Bangkok) Congress of the Far Eastern Association of Tropical Medicine, 1930: Bangkok, 1931, Vol. II, p. 499. Condensed in Internat. Journ. of Lepr., Vol. I, No. 2, April, 1933, pp. 221-222.

127. LAMPE, P. H. J. — Fate of Children Born of Leprous Parents in the Groot-Chatillon Leprosy Asylum. Internat. Journ. of Lepr., Vol. I, No. 1, Jan., 1933, pp. 5-15.

128. ROGERS & MUIR — "Leprosy," 1925, p. 66 et seq.

129. McCOY & GOODHUE — Quoted by ROGERS & MUIR, "Leprosy," 1925, p. 80.

130. GLÜCK, L. — 1900. Zur Kenntnis der leprösen Affektionen an der Glans penis. "Lepra," Vol. I, Fasc., 1.

131. ROBILIN — 1901. Leprosy Affecting the Penis. British Medical Journal, (extracts from current literature), May, 3: 1902.

132. MACLEOD, J. M. H. — Contact Cases of Leprosy in the British Isles. British Medical Journal, Vol. I, p. 107, Jan. 17, 1925.

133. — REPORT OF THE LEONARD WOOD MEMORIAL CONFERENCE on Leprosy, 1931. Pp. 465, 475, 476. Pub: by the Bureau of Science of the Philippine Govt. Vol. XLIV, No. 4, April, 1931.

134. DRACUP, A. H. & SORLEY, H. T. — Census of India, 1931. Vol. III, Part II, Bombay Presidency Statistical Tables. Imperial Table XIII, p. 353: Imperial Table XIX, p. 452: Provincial Table II, pp. 470-473. Obtainable at Office of the High Commissioner for India, India House, Aldwych, London, W.C.2.

135. McCOY, G. W. A Statistical Study of Leprosy in Hawaii. Public Health Bulletin, No. 66, Sept., 1914, p. 22. Washington, Govt. Printing Office.

136. ANNUAL REPORT FOR 1933 OF THE SURGEON-GENERAL OF THE PUBLIC HEALTH SERVICE OF THE UNITED STATES. U.S. Govt. Printing Office, Washington, D.C. Reviewed in British Medical Journal, Aug. 18, 1934, p. 313.

137. EAST INDIA CENSUS, 1931. Abstract of Tables, p. 12: Pub: by H.M. Stationery Office, 1932, at 120, George Street, Edinburgh.

138. WILLS, LUCY British Medical Journal, June 2, 1934, p. 1002.

139. SYMPOSIUM ON LEPER BEGGARS IN CALCUTTA. "Leprosy in India," July, 1933, p. 155.

140. RODRIGUEZ, J. N. Philippine Journal of Science, XXXI, 1926, p. 115.

141. HAYASHI, Y. Nippon Iji Shu-ho, 1927, 1665 & 1666.

142. HAYASHI, FUMIO International Journal of Leprosy, Vol. II, No. 1, Jan.-March, 1934, p. 65.

143. EHLERS & CAHNHEIM "Lepra," 2, 1902, p. 29.

144. SAND, A. "Lepra," 3, 1903, p. 7.

145. EAST INDIA CENSUS, 1931. Abstract of Tables, 1932, p. 8, Table III: p. 13, Table VIII: p. 14, Table IX: p. 16, Table XI.

146. LAMPE, P. H. J. International Journal of Leprosy, Vol. I, No. 1, p. 5. Jan., 1933.

147. BOSE, J. P. 1928. A Handbook on Diabetes Mellitus, p. 164 et seq. Pub. Thacker Spink & Co., Calcutta.

148. PINEDA, E. V. Pathological Survey of the Causes of Death in Lepers at Culion. Journal of the Philippine Islands Medical Association, Vol. IV, No. 5, May, 1924, pp. 169-178.

149. MACLEOD, J. M. H. 1909. British Journal of Dermatology, 21, pp. 309-323.

150. HANSEN, G. A. "Lepra," 3, 1902, p. 268.

151. HENDERSON, J. M. Indian Medical Gazette, Vol. LXV, No. 2, Feb., 1930, p. 100.

152. MUIR, E. Leprosy of the Lungs. "Leprosy in India," Vol. V, 1933, p. 72.

153. YAGLE, E. M. & KOLMER, J. A. 1923. The Kahn Precipitation Test in Leprosy. Archiv. Dermat. & Syph. Vol. VIII, No. 2.

154. FORMAN, D. N. Leprosy in an Urban General Dispensary. "Leprosy Review," Vol. II, No. 2, p. 74, April, 1931.

155. OTERO, P. M. 1927. A Survey of 42 cases of leprosy isolated at the Insular Leprosarium (Preliminary Report), Porto Rico Health Review, No. 2.

156. KAHN, R. L. The Kahn Reaction. "Leprosy Review," Vol. I, No. 1, Jan., 1930, p. 10. (Also, "The Kahn Test, A Practical Guide," Balliere, Tindall & Cox, London, 1928.)

157. GREIG, E. D. W. van ROOYEN, C. E. & HENDRY, E. B. Observations on the Melano-Precipitation Serological Reaction in Malaria. Trans. Roy. Soc. Trop. Med. & Hyg., Vol. XXVII, No. 2, Aug., 1934, pp. 175-191.

158. BLACKLOCK, D. B. & SOUTHWELL, T. "Human Parasitology," p. 108. London, H. K. Lewis & Co., Ltd., 1931.

159. ROGERS, SIR LEONARD "Leprosy Review," Vol. II, No. 3, July, 1931, p. 102.

160. MUIR, E. "Leprosy Review," Vol. III, No. 1, Jan., 1932, p. 35.

A P P E N D I X

CASE NOTES and PHOTOGRAPHS.

CASE NOTES

1. DINKAR SANE, M. 29, origin Paud: Christian of Maratha origins: student: self-supporting: single: literate: cleanly: moderate pyorrhoea: N-2 type: grandfather was a leper: lives in house containing 11 people: probable age of onset, 10: urine, albumin and casts, positive: Kahn test positive, 'four plus'.

2. GOVIND BAPAT, M. 18, origin Poona City: Brahman (Konkani): student: poor: single: literate: cleanly: teeth fair: N-2,C-1 type: friend at college a leper, but no leper relatives: lives in small house containing 9 people: probable age of onset, 15: urine, albumin and casts negative: Kahn negative: had tuberculous glands at 9 years, operated on in hospital, sister died of pulmonary tuberculosis.

3. HASHIM KARIM, M. 25, origin Poona City: Mahommedan: butcher: poor independent: married: illiterate: not cleanly: teeth bad: N-1,C-2 type: wife a leper, both had leprosy in their families: lives in single room containing 5 people: probable age of onset, 20: urine, albumin positive, casts negative, blood film, malaria, positive: Kahn negative, but definite history of syphilis at 20 years: also had gonorrhoea.

4. M. B. GAEKWAD, M. 26, origin Poona City: Maratha: student: poor dependent: married: literate in vernacular: cleanly: teeth fair: N-1,C-2 type: mother a leper, and no other relatives: lives in single room containing 3 people: probable age of onset, 18: urine, albumin and casts, negative: history of malaria more than 5 attacks: Kahn positive, 'three plus': his wife left him when he was seen to be a leper.

5. SAMUEL TIVARI, M. 10, origin Poona City: Christian of outcaste origins: minor, at school: poor dependent: literate: cleanly: teeth good: N-1 type: sister (18 years of age) a leper: lives in a house containing 15 people: probable age of onset, 2 years: urine, albumin positive, casts, negative: Kahn negative: father and mother also had mild leprosy.

6. MARUTI LAXMAN HOLI, M. 28, origin Poona City: Mali: potato shopkeeper: poor dependent: married, wife separate: literate in the vernacular: not cleanly: teeth fair: C-3 type: grandmother a leper: lives in a house containing 14 people: probable age of onset 22: urine, negative: Kahn negative, but definite history of syphilis at 22 years: weak chest since age of 16, cough and sputum for many years before onset of leprosy: sputum positive and g.p. inoculation positive: his aunt died of wasting disease.

7. MARTIN NARAYAN KULKARNI, M. 43, origin Kolaba District: Prabhu: clerk: poor dependent: married: literate: cleanly: bad dental caries: N-1, C-2 type: no relative a leper, but once sat by a leper in the bus: lives in single room with 6 people: probable age of onset, 33: urine, albumin, positive, casts, negative: blood film, malaria, positive: Kahn test positive, 'four plus': a friend of his father used to come to the house in his childhood, who had nodules on the face.

8. KESU YESU, M. 30, origin Poona City: Mahar: carriage cleaner in Poona Station: poor independent: married: illiterate: not cleanly: bad teeth: C-2 type: no information as to contact: lives in a hut of one room, containing 7 people: probable age of onset, 16: urine, negative: history of malaria, more than 5 attacks: Kahn negative, but definite history of syphilis at 18 years.

9. DEVJI YEMAJI, M. 51, origin Alandi, Poona District: Maratha: farm labourer: poor independent: married: illiterate: not cleanly: teeth bad: N-1, C-2 type: son a leper: lives in a house containing 20 people: probable age of onset, 41: urine, positive for albumin and casts: blood film positive for malaria: Kahn positive, 'two plus'.

10. A. DUTTON, M. 32, origin Poona City: Anglo-Indian: ticket-collector on Poona Station: married: well-educated: cleanly: teeth bad: N-2 type: no relative a leper, but contact with leper prostitute: lives in a single room containing 5 people: urine, negative: malaria, history of more than 5 attacks: Kahn negative: probable age of onset of leprosy, 20.

11. SUNDRABAI KISAN, F. 25, origin Poona City: Shimpi: housewife: married, 3 children: poor dependent: illiterate: not cleanly: teeth fair: N-1 type: grandmother a leper: lives in single room containing 5 people: urine, negative: Kahn negative, but definite history of syphilis at 14: probable age of onset of leprosy, 14, or 15.

12. BABU DHONDIBA, M. 20, origin Divalgaon: Maratha: smallholder farmer: married: literate in vernacular: cleanly: good teeth: N-1,C-1 type: grandfather a leper: lives in house containing 16 people: urine, positive for albumin and casts: blood film positive for malaria: Kahn positive, 'four plus': probable age of onset of leprosy, 15.

13. BHAGUBAI RAOJI, F. 40, origin Nimbgaon: Maratha: farm labourer: married, 5 children: illiterate: not cleanly: teeth bad: N-2 type: uncle a leper: lives in a single room containing 6 people: urine, negative: malaria, history of 5 attacks: Kahn, negative: probable age of onset of leprosy, 25 years: poor dependent.

14. SHAIKH UMAR SHAIK DAWOOD, M. 35, origin Poona City: Mahommedan: shopkeeper: self-supporting: married: literate in Urdu: not cleanly: teeth fair: N-1, C-2 type: aunt a leper: probable age of onset, 17: lives in house containing 22 people: urine, negative: Kahn positive, 'four plus': has had gonorrhoea.

15. DATHU MANKU, M. 15, origin Kurundwad: Chambar: leatherworker: poor, dependent: single: illiterate: not cleanly: teeth good: N-1 type: father a leper: probable age of onset, 10 years: lives in single room containing 4 people: urine, positive for albumin and casts: Kahn negative: walked with a limp on examination, and had done so since 10 years of age; psoas abscess developed and was aspirated; leprosy improved and was arrested in 3 years.

16. HARI RAMU THORAT, M. 50, origin Nirowdi: Maratha: farm labourer: poor, independent: widower: illiterate: not/

not cleanly: teeth bad: N-2, C-1 type: wife a leper, developed leprosy at 20, died at 30: probable age of onset, 40: lives in a single room containing 3 people; urine, positive for albumin and casts: malaria, history of more than 5 attacks: Kahn positive, 'two plus'.

17. GANU BODUJI, M. 36, origin Khed Chakun: Maratha: farm labourer: poor, independent: married: illiterate: cleanly: teeth fair: N-1 type: brother a leper: probable age of onset, 22: lives in a house containing 10 people: urine, negative: malaria, history of 5 attacks: Kahn negative, but definite history of syphilis at 20 years.

18. GULAM KHAN, M. 24, origin Poona City: Mahommedan: motor bus driver: self-supporting: single: illiterate: not cleanly: teeth fair: N-1, C-2 type: brother a leper: probable age of onset, 20: lives in single room containing 4 people: urine, positive for albumin and casts: malaria, history of 5 attacks: Kahn positive, 'four plus'.

19. SOMA MUKHUN, M. 40, origin Malwad: Teli: oilshop: self-supporting: married: illiterate: not cleanly: teeth bad: N-1,C-1 type: sister a leper: lives in a house containing 10 people: probable age of onset, 25: urine positive for albumin and casts: Kahn positive, 'four plus'.

20. CHINTAMANI KAMALAPURKAR, M. 30, origin Ahmednagar: Brahman: teacher in primary school: literate: poor, dependent: married: not cleanly: teeth fair: N-1 type: uncle a leper: lives in a single room containing 4 people: probable age of onset, 20: urine negative: Kahn negative: at 19 years, began to have cough and sputum, fever, sweats, and wasting; father died of tuberculosis; sputum positive on staining and on g.p. inoculation; got worse in a year under observation, and then left.

21. JAWAHRLAL NYALCHAND, M. 40, origin Poona Cantonment: Marwadi: cloth shopkeeper: self-supporting: literate: married: cleanly: teeth bad: N-2 type: no information as to contact: lives in a house containing 15 people/

people: probable age of onset, 14: urine, negative: Kahn negative, but definite history of syphilis at 20 years.

22. BABURAO PHULE, M. 51, origin Poona City: Mali: private house gardener: destitute: illiterate: widower: not cleanly: teeth bad: C-3 type: brother a leper: housing, none: probable age of onset, 42: urine, positive for albumin, negative for casts: blood film, positive for malaria: Kahn positive, 'four plus'.

23. PANDARINATH BHAU, M. 42, origin Wadgaon: Maratha: farm labourer: poor, independent: illiterate: married: not cleanly: teeth fair: C-1 type: grandmother a leper: lives in single room containing 7 people: probable age of onset 16: urine, negative: malaria, history of more than 5 attacks: Kahn negative.

24. SHANKAR RAGHU, M. 10, origin Shelarwadi: Maratha: minor, helps in fields: poor, dependent: illiterate: not cleanly: teeth good: N-1 type: mother a leper: lives in single room containing 5 people: probable age of onset, 2 to 3 years: urine negative: Kahn negative.

25. MARYBAI TIVARI, F. 40, origin Poona City: Christian of outcaste origins: housewife: widow: poor, dependent: literate: 2 children, lepers: husband a leper, concealed before marriage: lives in a house containing 15 people: probable age of onset, 12: urine negative: malaria, history of more than 5 attacks: Kahn negative. Type N-1.

26. KHONDILAKA TUKARAM, M. 55, origin Bihudi: Maratha: farm labourer: destitute: illiterate: not cleanly: teeth bad: C-1 type: 2 brothers lepers: housing, none: probable age of onset, 19: urine negative: malaria, history of 5 attacks: Kahn negative, but definite history of syphilis at 22 years. Falling off in food and cleanliness after wife died.

27. SHANKAR TUKARAM, M. 62, origin Junnar: Mahar: coolie on roads: destitute: illiterate: not cleanly: teeth/

teeth bad and few: N-2 type: grandchild a leper: housing, none: probable age of onset, 46 years: widower: urine positive for albumin and casts: Kahn positive, 'four plus'.

28. SOPANA CHOUDARI, M. 25, origin Talegaon-Dhamdere: Maratha: farm labourer: poor, dependent: single: illiterate: cleanly: teeth fair: C-2 type: aunt a leper: lives in a single room containing 4 people: probable age of onset, 14: urine, positive for albumin, negative for casts: Kahn positive, 'two plus': he had lived with a leper aunt and a tuberculous mother, and began to be weakly at 14 years; for about 3 years he had fever and cough, and came into hospital for that; when in hospital fever brought out an extensive crop of cutaneous lesions which proved to be leprosy: later, his sputum was found positive for tubercle by staining and g.p. inoculation; both diseases rapidly developed, and he left.

29. GOPIN AUGUSTIN, M. 10, origin Poona City: Christian of outcaste origins: minor, at school: poor, dependent: literate: cleanly: fair teeth: N-1 type: cousin a leper: lives in a house containing 21 people: probable age of onset, 5: urine, negative: Kahn negative.

30. ASHEBA VITHOBA, M. 22, origin Jejuri: Mang: coolie on roads: poor, independent: illiterate: single: not cleanly: teeth good: C-1 type: father a leper: lives in a hut of a single room containing 6 people: probable age of onset, 16: urine, positive for albumin, negative for casts: malaria, history of more than 5 attacks: Kahn positive 'three plus': has had gonorrhoea.

31. HARI DHARMA, M.40, origin Kikwi: Mahar: coolie: poor, independent: illiterate: married: not cleanly: teeth bad: C-2 type: uncle a leper: lives in a hut of one room containing 3 people: probable age of onset, 16: urine, positive for albumin and casts: malaria, history of 5 attacks: Kahn positive, 'four plus'.

32. RAMCHANDRA MAHADU, M. 31, origin Baramati: Maratha: millworker: self-supporting: illiterate: single: cleanly: teeth fair: N-1, C-1 type: no relative a leper, but lives with a fellow millworker who became a leper/

leper: lives in a single room containing 7 people: probable age of onset, 21: urine, negative: Kahn negative, but definite history of syphilis at 20 years.

33. RAMA RANGU, M. 20, origin Rhangaon: Mahar: coolie at Poona Station: destitute: single: illiterate: not cleanly: teeth good: N-2 type: father a leper: housing, none: probable age of onset, 17: urine, negative: Kahn positive, 'two plus'.

34. TULSIRAM GOPAL, M. 35, origin Nanakerwadi, Chakun: Maratha: smallholder farmer: comparatively well-off: married: illiterate: not cleanly: teeth fair: N-2 type: no relative a leper, but had a friend in the house for 2 years who was a leper: lives in a house containing 5 people: probable age of onset, 22: urine negative: malaria, history of more than 5 attacks: Kahn negative.

35. ISMAIL JALABI, M. 27, origin Poona Cantonment: Mahommedan: shopkeeper: comparatively well-off: single: illiterate: not cleanly: teeth good: N-2 type: grandmother a leper: lives in a house containing 12 people: probable age of onset, 18: urine positive for albumin and casts: Kahn positive, 'four plus'.

36. ANANDA TULSIRAM, M. 10, origin Chakun: Mahar: minor, no occupation: poor, dependent: illiterate: not cleanly: teeth bad: N-2 type: father and mother lepers: lives in single room containing 9 people: probable age of onset, 2: urine negative: blood film positive for malaria: Kahn negative.

37. GOVIND LAXMAN, M. 20, origin Dhena, Bhor State: Maratha: farm labourer: poor, dependent: single: illiterate: cleanly: teeth good: N-1 type: uncle a leper: lives in house containing 12 people: probable age of onset, 15: urine, negative: Kahn negative.

38. ABDUL RAHMAN, M. 33, origin Poona City: Mahommedan: cloth shopkeeper: poor, independent: single: illiterate: cleanly: teeth fair: N-2, C-2 type: cousin and/

and brother a leper: lives in house containing 9 people: probable age of onset, 9: urine, negative: malaria, history of 5 attacks: Kahn negative, but definite history of syphilis at 21.

39. MAHADU TUKARAM, M. 20, origin Poona City: Mang: general coolie: destitute: single: illiterate: not cleanly: teeth bad: N-1, C-1 type: friend a leper: housing, none: probable age of onset, 17: urine, positive for albumin, negative for casts: malaria, history of more than 5 attacks: Kahn positive, 'three plus'.

40. DHONDIBAI YESHWANT, F. 30, origin Shrigonda: Maratha: housewife: poor, dependent: single: illiterate: cleanly: teeth good: N-1 type: sister a leper: lives in a house containing 11 people: probable age of onset, 13: urine, negative: Kahn negative.

41. KASHINATH BOLUJI, M. 35, origin Katraj: Maratha: small holder farmer: poor, independent: married: illiterate: not cleanly: bad teeth: N-1 type: no leper relatives, but slept in a resthouse on a pilgrimage where there was a leper: lives in a house containing 7 people: probable age of onset, 30: urine negative: malaria, history of more than 5 attacks: Kahn negative.

42. MAHADU LAXMAN, M. 33, origin Poona City: Mahar: general coolie: poor, independent: married: illiterate: not cleanly: teeth fair: C-2 type: grandfather a leper: lives in a hut of a single room containing 8 people: probable age of onset, 21: urine positive for albumin and casts: Kahn positive, 'three plus'.

43. BABU SAYED KAIRUBHI, M. 14, origin Poona City: Mahommedan: minor, no occupation: poor, dependent: single: illiterate: not cleanly: teeth good: N-1, C-1 type: grandmother a leper: lives in a single room containing 10 people: probable age of onset, 10: urine, positive for albumin, negative for casts: blood film positive for malaria: Kahn negative.

50. KHATOOR SAYEDDAVLAD, F. 12, origin Bhamburda: Mahommedan: minor, no occupation: poor, dependent: single: illiterate: not cleanly: teeth bad: N-2, C-1 type: grandmother and mother lepers: lives in a single room containing 5 people: probable age of onset, 6 years: urine, positive for albumin, negative for casts: malaria, blood film positive: Kahn positive, 'three plus'.

51. GOPAL HARI, M. 32, origin Poona City: Mahar: railway coolie: poor, independent: married: illiterate: not cleanly: teeth fair: N-1, C-3 type: brother a leper: lives in a single room containing 5 people; probable age of onset, 25 years: urine, negative: malaria, blood film positive: Kahn positive, 'two plus'.

52. CHINTU VITHU, M. 40, origin Ambegaon: Mang: railway public luggage coolie: poor, independent: married: illiterate: not cleanly: teeth bad: N-1, C-2 type: no information as to contact: lives in a hut of a single room containing 5 people: urine, negative: Kahn positive, 'four plus': probable age of onset of leprosy, 21 years.

53. PUNAMCHAND JUMARJI, M. 50, origin Poona City: Chambar: itinerant shoemaker: poor, independent: single: illiterate: not cleanly: teeth bad: C-3 type: contact with a leper prostitute at about age of 25: lives in a house containing 9 people: probable age of onset, 30 years: urine, positive for albumin and casts: Kahn positive, 'three plus'.

54. AMBU TUKA, M. 30, origin Ambegaon: Mahar: general coolie: poor, independent: single: illiterate: not cleanly: teeth fair: N-1 type: friend a leper: lives in a house containing 20 people: probable age of onset, 22: urine, negative: malaria, history of more than 5 attacks: Kahn negative, but history of syphilis at 18.

55. SITABAI SITARAM, F. 27, origin Patarwadi: Koshti: millworker: poor, dependent: married, 4 children: illiterate: cleanly: teeth good: N-1, C-1 type: son a leper: lives in a single room containing 7 people: probable age of onset, 18 years: urine negative: Kahn positive, 'two plus'.

56. BABU GANPAT, M. 13, origin Khed Chakun: Teli: oil-seller: poor dependent: single: illiterate: not cleanly: teeth fair: N-1, C-2 type: father a leper: lives in a single room containing 7 people: probable age of onset, 8: urine positive for albumin and casts: malaria, history of 5 attacks: Kahn negative.

57. DATHUPANT DAMAJI, M. 38, origin Sholapur: Jain Wani: cloth shopkeeper: comparatively well-off: married: illiterate: cleanly: teeth fair: N-2 type: wife a leper, both having leprosy in their families, and developing it after marriage, the husband 2 years before wife: lives in a house containing 15 people: probable age of onset, 22: urine positive for albumin, negative for casts: Kahn positive, 'four plus'.

58. MARKUS KONDIBA, M. 18, origin Chinchwad: Christian of outcaste origins: student: poor, dependent: single: literate: cleanly: teeth good: N-1 type: uncle a leper: lives in house containing 11 people: probable age of onset, 16 years: urine negative: malaria, history of 5 attacks: Kahn negative.

59. SONUBAI GANGARAM, F. 40, origin Supa: Mahar: housewife: poor, dependent: married, 6 children: illiterate: not cleanly: teeth fair: N-2 type: mother a leper: lives in a house containing 17 people: probable age of onset, 25: urine negative: Kahn positive, 'three plus'.

60. ISHWAR WAGMARE, M. 20, origin Poona City: Mahar: private motor driver: comparatively well-off: married: illiterate: cleanly: teeth good: N-2 type: no information as to contact: lives in a single room containing 4 people: probable age of onset, 15: urine negative: malaria, history of more than 5 attacks: Kahn negative.

61. SHAIKHUSAIN SHAIKRAHMAN, M. 25, origin Poona City: Mahommedan: tailor: poor, independent: single: illiterate: not cleanly: teeth bad: N-2 type: contact with a leper assistant: lives in single room containing 3 people: probable age of onset, 20: urine negative: Kahn negative, but history of syphilis at 16.

62. LAXMAN SITARAM, M. 7, origin Patarwadi: Koshti: minor, no occupation: illiterate: cleanly: good teeth: N-1 type: uncle a leper: lives in single room containing 4 people: probable age of onset, 6 years: urine negative: blood film positive for malaria: Kahn negative.

63. SAKHARAM ZOTICHAND, M. 31, origin, Phaltan: Gujar Wani: grocer and shopkeeper: rich (over Rs. 45 per mensem): literate in vernacular: not cleanly: teeth bad: N-1, C-2 type: brother a leper: lives in house containing 25 people: probable age of onset, 20 years: urine, positive for albumin and casts: Kahn positive, 'two plus': single.

64. BENJAMIN BHIMAJI, M. 20, origin Poona City: Christian of outcaste origins: carpenter: poor, independent: single: literate: cleanly: teeth good: friend a leper: lives in a house containing 20 people: N-2 type: probable age of onset, 16: urine, negative: malaria, history of more than 5 attacks: Kahn negative.

65. TUKARAM KASHIBA, M. 46, origin Poona City: Mahar: general coolie: destitute: married: illiterate: not cleanly: teeth bad: N-1, C-1 type: casual contact with lepers in a bus: housing, none: probable age of onset 40 years: urine, positive for albumin and casts: Kahn positive 'four plus': admits having syphilis at age of 34.

66. KODURAO SUKMAT, M. 30, origin Chinchwad: Marwadi: clothing store shopkeeper: self-supporting: single: literate in the vernacular: cleanly: teeth fair: N-3 type: grandfather a leper: lives in a house containing 16 people: probable age of onset, 25: urine, negative: malaria, history of more than 5 attacks: Kahn negative.

67. CHANDANBAI KUMU, F. 40, origin Poona City: Christian of outcaste origins: Biblewoman: poor, dependent: widow, 5 children: literate: cleanly: teeth bad: N-1 case: no information as to contacts: lives in a single room with 6 people: probable age of onset, 20: urine, negative: blood film, positive for malaria: Kahn positive, 'two plus'.

68. NARASAPPA RAJAUNA, M. 22, origin Sholapur: no occupation: single: destitute: illiterate: not cleanly: teeth fair: N-2 type: contacts with leper beggar: housing, none: age of onset, 16: urine, positive for albumin, negative for casts: malaria, more than 5 attacks: Kahn positive, 'one plus'. Caste Padamsali.

69. MAHADU SHRIPAT, M. 18, origin Hadapsar: Maratha: motor bus driver: poor, independent: single: illiterate: cleanly: teeth fair: N-1 type: father a leper: lives in a house containing 6 people: probable age of onset, 15: urine, negative: malaria, more than 5 attacks: Kahn negative.

70. DURGA MAHADU, M. 19, origin Poona City: Mahar: railway public luggage porter: poor, independent: single: illiterate: not cleanly: teeth fair: N-1, C-1,type: friend a leper with whom he slept: lives in single room containing 2 people: probable age of onset, 14: urine negative: Kahn positive, 'one plus'.

71. SAYED ASADULLA, M. 50, origin Poona Cantonment: Mahommedan, race course syce: poor, independent: married: illiterate: cleanly: teeth bad: N-2,C-3 type: no information as to contact: lives in a single room containing 5 people: probable age of onset, 35: urine, positive for albumin and casts: malaria, history of 5 attacks: Kahn negative.

72. AMINABAI PATHAN, F. 40, origin Poona City: Pathan: housewife, husband a sepoy: widow: 3 children: poor, dependent: illiterate: cleanly: teeth fair: N-2 type: son a leper: lives in single room containing 5 people: probable age of onset, 20: urine, negative: Kahn negative.

73. BHAGUBAI, F. 50, origin Pimpalwadi: Dhangar: shepherd: poor, dependent: widow, 6 children: illiterate: not cleanly: teeth bad: N-3 type: sister a leper: lives in house containing 14 people: probable age of onset, 25: urine, negative: malaria, more than 5 attacks: Kahn negative.

74. TUKKIBAI, F. 20, origin Chikhli: Maratha: field worker: poor independent: married, 1 child: illiterate: cleanly: teeth fair: N-2, C-1 type: sister a leper: lives in single room containing 4 people: probable age of onset, 14: urine negative: Kahn negative.

75. AMBUBAI BHIKU, F. 40, origin Takli, Ahmednagar District: Maratha: field worker: poor, dependent: widow, 1 child: illiterate: not cleanly: teeth bad: N-3 type: niece a leper: lives in single room containing 2 people: probable age of onset, 20: urine negative: Kahn negative.

76. NOORBI, F. 50, origin Shikrapur: Mahommedan: housewife: poor, dependent: married, 4 children: illiterate: cleanly: teeth bad: N-2 type: husband a leper, discovered after their marriage, no history of leprosy in their families: lives in house containing 10 people: probable age of onset, 30: urine negative: Kahn negative.

77. ANUSAYABAI HARI, F. 11, origin Sholapur: Maratha: minor, no occupation: destitute: single: illiterate: not cleanly: teeth good: C-2 type: mother a leper: housing, none: age of onset, 10 years: urine, positive for albumin and casts: Kahn positive, 'three plus': syphilis may be inherited.

78. ANANT APPAJI , M. 45, origin Poona City: Goundi: stonemason: self-supporting: married: illiterate: not cleanly: teeth bad: N-2,C-1 type: grandfather a leper: lives in a house containing 14 people: probable age of onset, 30: urine, negative: malaria, more than 5 attacks: Kahn positive, 'one plus'.

79. SONUBAI PATNE, F. 20, origin Poona City: Christian of outcaste origins: sempstress: poor, dependent: single: literate: cleanly: teeth fair: N-2 type: friend a leper, with whom she slept: lives in a house containing 20 people: probable age of onset, 6 years: urine, negative: blood film positive for malaria: Kahn negative.

80. TULSABAI DEMA, F. 30, origin Satara: Maratha: housewife: poor, dependent: married, 4 children: illiterate: cleanly: teeth bad: N-2,C-1 type: uncle a leper: lives in house containing 8 people: probable age of onset, 12: urine negative: Kahn negative.

81. CHANDRABAI BHARUM, F. 25, origin Belgaum: Kanadi Devadasi: temple dancer and prostitute: poor, dependent: single: illiterate: not cleanly: teeth good: N-2 type: casual contacts numerous with leper priests and pilgrims: lives in single room containing 4 people: probable age of origin, 17 years: urine, positive for albumin and casts: malaria, 5 attacks: Kahn positive 'four plus': remarkable improvement in leprous lesions after anti - syphilitic treatment.

82. JANIKAMA, F. 30, origin Penang: Madrassi Nair: housewife: destitute: married, no children: illiterate: no leper relatives or history of contacts, but says leprosy was prevalent in Penang, where she spent her youth: cleanly: teeth bad: N-2 type: housing, none: probable age of onset, 24 years: urine negative: Kahn negative.

83. VITHABAI DHONDI, F. 30, origin Malwan, Konkan: Christian of Maratha origins: housewife: destitute: married, 3 children: illiterate: not cleanly: teeth bad: N-2 type: grandmother a leper: housing, charity: probable age of onset, 16: urine, negative: malaria, history of more than 5 attacks: Kahn negative.

84. MANIBAI RHADU, F. 20, origin Paud: Maratha: field labourer: poor, independent: single: illiterate: not cleanly: teeth fair: N-2 type: cousin a leper: lives in house containing 12 people: probable age of onset, 10: urine negative: malaria, 5 attacks or more: Kahn positive, 'two plus'.

85. MANORAMABAI BHUTILAL, F. 25, origin Ankleshwar: Christian of Gujar Wani origins: housewife: poor, dependent: single: literate: cleanly: teeth good: N-1, C-1 type: mother a leper: lives in single room containing 3 people: probable age of onset, 17: urine negative: Kahn positive, 'two plus'.

86. MAHADU BALA, M. 18, origin Ahmednagar: Christian of out-caste origins: millworker: poor, independent: single: illiterate: cleanly: teeth fair: C-1 type: father a leper: lives in single room containing 9 people: probable age of onset, 12 years: urine negative: Kahn negative.

87. MARUTI DAGADU, M. 18, origin Poona City: Maratha: tanga driver: self-supporting: single: illiterate: not cleanly: teeth fair: C-2 type: casual contact with a leper in the market: lives in single room containing 3 people: probable age of onset, 14: urine negative: blood film positive for malaria: Kahn positive, 'two plus'.

88. YAKOB NATHANIEL, M. 25, origin Jalna: Nizam's Dominions: Christian of Beni-Israel origins: domestic servant: self-supporting: single: literate: cleanly: teeth fair: N-2, C-1 type: fellow-servant a leper, with whom he slept: lives in single room containing 3 people: probable age of onset, 19: urine negative: Kahn negative.

89. NANA BABAJI, M. 30, origin Bhor: Maratha: farm labourer: poor, independent: married: illiterate: not cleanly: teeth bad: N-2 type: father a leper: lives in a house containing 11 people: probable age of onset, 25: urine negative: malaria, more than 5 attacks: Kahn negative.

90. HUSAIN IMAM, M. 15, origin Gulbarga, Nizam's Dominions: Mahommedan: minor, no occupation: destitute: single: illiterate: cleanly: teeth good: N-2 type: father a leper: housing, none: probable age of onset, 4 years: urine negative: malaria, history of 5 attacks: Kahn negative.

91. TATYOBA YESHWANT, M. 50, origin Thana: Parbhu: sepoy: self-supporting: widow: well-educated: cleanly: teeth fair: N-2,C-2 type: casual contact with leper in courthouse: lives in single room containing 4 people: probable age of onset, 30: urine positive for albumin and casts: Kahn doubtful positive.

92. SARJUDAS BHAUSA, M. 35, origin N.W. Frontier: Gosavi mendicant: destitute: single: illiterate: not cleanly: teeth bad: N-2, C-1 type: friend a leper: housing, none: probable age of onset, 20: urine negative: malaria, more than 5 attacks: Kahn positive 'two plus', history of syphilis.

93. ABEL IBASHA, M. 30, origin Travancore: clerk: Christian of outcaste origins: poor, independent: single: literate: cleanly: teeth good: N-2, C-1 type: father a leper: lives in single room containing 7 people: probable age of onset, 15: urine negative: Kahn negative.

94. DHONDUSA, M. 35, origin Belgaum: grocer shopkeeper: comparatively well-off: married: literate: not cleanly: teeth fair: N-2 type: brother a leper: lives in house containing 7 people: age of onset, 24: urine negative: Kahn positive 'four plus', history of syphilis.

95. LAZARUS YELLAPPA, M. 25, origin Tirthpuri, Nizam's Dominions: Christian of outcaste origins: student: destitute: single: literate: not cleanly: teeth fair: C-1 type: grandfather a leper: housing, none: probable age of onset, 20: urine, negative: Kahn negative.

96. BABU KONDIBA, M. 11, origin Poona City: Maratha: poor, dependent: single: illiterate: minor, no occupation: not cleanly: teeth good: N-1 type: mother a leper: lives in a house containing 12 people: age of onset, 6: urine negative: Kahn negative.

97. RAMA SHETIBA, M. 50, origin Sholapur: Maratha: millworker: self-supporting: married: illiterate: not cleanly: teeth bad: N-2, C-3 type: no information as to contact: lives in single room containing 5 people: probable age of onset, 30: urine negative: Kahn negative.

98. BHAU ARJUN, M. 30, origin Kodoli, Kolhapur District: Christian, formerly Mang: no occupation: destitute: single: illiterate: not cleanly: teeth bad: N-2 type: grandmother a leper: housing, none: age of onset, 15: urine negative: Kahn positive 'four plus'.

99. GAFFUR CHOTU, M. 15, origin Aurungabad, Nizam's Dominions: Pathan: minor, no occupation: destitute: single: illiterate: not cleanly: teeth fair: N-1 type: uncle a leper: no housing: age of onset, 3: urine negative: blood film positive for malaria: Kahn negative.

100. LOKAPPA, M. 25, origin Belgaum: Maratha: farm labourer: poor, independent: single: illiterate: not cleanly: teeth fair: C-2 type: shaved once by a leper barber: lives in house containing 12 people: age of onset, 20: urine negative: Kahn negative.

101. ALLAH BUX, M. 18, origin Poona City: Mahommedan: shop-assistant (hardware store): poor, independent: single: illiterate: cleanly: teeth good: C-1 type: uncle a leper: lives in house containing 6 people: age of onset, 16: urine negative: malaria, history of more than 5 attacks: Kahn positive, 'two plus'.

102. RAMA RAGHU, M. 30, origin Thana: Mahar: beggar: destitute: single: illiterate: not cleanly: teeth bad: N-2, C-2 type: friend a leper: no housing: probable age of onset, 12: urine, positive for albumin and casts: Kahn positive, 'four plus'.

103. GANPAT PHAKIRA, M. 25, origin Panvel, Konkan: Maratha: salt worker: poor, independent: single: illiterate: cleanly: teeth fair: N-2, C-1 type: father a leper: lives in single room containing 2 people: age of onset, 10: urine negative: malaria, 5 attacks: Kahn negative.

104. VELAYUDHAN, M. 30, origin Madras Presidency: Madrassi Nair: Hindu hotelkeeper: self-supporting: married: literate: not cleanly: teeth fair: C-2 type: casual contacts with lepers in his 'hotel': lives in house containing 4 people: age of onset, 25: urine negative: Kahn negative.

105. GURAPPA, M. 30, origin Belgaum District: Lingayat Wani: shopkeeper: poor, independent: married: illiterate/

illiterate: cleanly: teeth fair: N-2, C-1 type: no history of association with lepers: lives in house containing 17 people: probable age of onset, 14: urine, positive for albumin and casts: Kahn, positive 'two plus'.

106. VEDGIRI, M. 25, origin Madras Presidency: Madrassi outcaste: beggar: destitute: single: illiterate: not cleanly: teeth fair: C-2 type: friend a leper: no housing: probable age of onset, 20: urine negative: malaria, more than 5 attacks: Kahn positive 'two plus'.

107. RAMSINGH VITHULSINGH, M. 35, origin Baramati: Rajput: military sepoy: self-supporting: single: literate: cleanly: teeth good: no history of contact: lives in single room containing 2 people: probable age of onset, 22: urine, negative: Kahn positive 'four plus'.

108. RADHABAI BHAU, F. 25, origin Kolhapur: Mang: prostitute: destitute: single: illiterate: not cleanly: teeth fair: N-3 type: father a leper: no housing: age of onset, 5: urine, positive for albumin and casts: malaria, more than 5 attacks: Kahn positive 'one plus'.

109. MARINATTU, F. 35, origin Calicut: Madrassi outcaste: beggar: destitute: married, 3 children: illiterate: not cleanly: teeth fair: N-1, C-2 type: no history of association with lepers: lives in a single room containing 5 people: probable age of onset, 30: urine negative: Kahn positive, 'one plus'.

110. RADHABAI KRISHNA, F. 25, origin Goa: Sonar: housewife: comparatively well-off: married, 4 children: illiterate: cleanly: teeth fair: N-2, C-2 type: no history of contact: lives in house containing 10 people: age of onset 10: urine negative: Kahn positive 'two plus'.

111. MANJULABAI HANUMANT, F. 30, origin Paud: Mahar: coolie: poor, independent: single: illiterate: not cleanly: teeth fair: C-2 type: no history of contact: lives in single room containing 4 people: probable age of onset, 25: urine negative: malaria, more than 5 attacks, Kahn positive, 'one plus'.

112. PAU THOMAR, M. 25, origin Tanjore: teacher in primary school: Christian of outcaste origins: self-supporting: single: literate: cleanly: teeth good: N-2, C-1 type: no history of contact: lives in single room containing 4 people: age of onset, 18: urine negative: malaria, more than 5 attacks: Kahn positive 'two plus'.

113. TANHIBAI PANDU, F. 25, origin Satara: Maratha: field labourer: poor, dependent: single: illiterate: not cleanly: teeth fair: N-1, C-1 type: uncle a leper: lives in house containing 10 people: age of onset, 21: urine negative: Kahn positive 'three plus'.

114. MALAN NANA, F. 25, origin Thana: Christian of outcaste origins: housewife: poor, dependent: single: literate: cleanly: teeth fair: she gives no history of contact: lives in house containing 11 people: N-1, C-1 type: age of onset, 19 years: urine negative: malaria, more than 5 attacks: Kahn negative.

115. BHAGIBAI VITHUL, F. 30, origin Kolaba District: Maratha: field labourer: self-supporting: married, 5 children: illiterate: cleanly: teeth good: N-3 type: no association with lepers: lives in house containing 23 people: probable age of onset, 13: urine negative: malaria, more than 5 attacks: Kahn, doubtful positive.

116. GANGABAI JOSE, F. 35, origin Thana: Mahar: prostitute: destitute: widow, 2 children: illiterate: not cleanly: teeth fair: N-2, C-2 type: friend a leper: no housing: probable age of onset, 15: urine, positive for albumin and casts: Kahn positive 'four plus'.

117. KAMALABAI LAXMAN, F. 30, origin Bombay: Christian of outcaste origins: student: poor, dependent: single: literate/

literate: cleanly: teeth fair: C-2 type: no association with lepers: lives in house containing 16 people: probable age of onset, 15 years: urine negative: malaria, more than 5 attacks: Kahn, doubtful positive.

118. HAMPAMA, F. 30, origin Aurungabad, Nizam's Dominions: Maratha: field labourer: poor, dependent: married, 2 children: illiterate: cleanly: teeth fair: N-2, C-2 type: no association with lepers: lives in house containing 9 people: age of onset, 14: urine negative: Kahn negative.

119. MARY ABEL, F. 25, origin Madras Presidency: Christian of outcaste origins: sempstress: poor, dependent: single: literate: cleanly: teeth good: N-3 type: father a leper: lives in house containing 14 people: age of onset, 4 years: urine negative: malaria, more than 5 attacks: Kahn negative.

120. KARONHABAI, F. 20, origin Poona City: Christian of outcaste origins: sempstress: poor, dependent: single: literate: cleanly: teeth fair: N-2 type: mother a leper: lives in house containing 20 people: age of onset, 12: urine negative: blood film positive for malaria: Kahn positive 'three plus'.

121. DINABAI SADANAND, F. 20, origin Secunderabad, Nizam's Dominions: Maratha: domestic servant: poor, independent: single: illiterate: not cleanly: teeth fair: N-2 type: fellow house servant a leper: lives in single room containing 2 people: probable age of onset, 16: urine negative: Kahn negative.

122. KISAN AMRUT, M. 30, origin Shahapur, Thana District: Maratha: cafe or teashop worker: self-supporting: single: illiterate: not cleanly: teeth bad: N-2, C-1 type: contacts with lepers in the teashop: lives in house containing 14 people: age of onset, 19: urine negative: malaria, 5 attacks: Kahn, doubtful positive.

123. JOSEPH ROBIN, M. 30, origin Bengal: Christian of outcast origins: student: poor, dependent: single: literate: cleanly: teeth fair: C-2 type: no history of/

of leper contacts: lives in house containing 9 people: probable age of onset, 25: urine negative: Kahn negative.

124. SIDHAPPA, M. 55, origin Ambarh, Nizam's Dominions: Maratha: smallholder farmer: self-supporting: married: illiterate: not cleanly: teeth bad: N-3, C-1 type: no history of contact: lives in house containing 16 people: age of onset, 20: urine negative: Kahn negative.

125. NARHARI SAKHARAM, M. 45, origin Poona City: Brahman: priest: comparatively well-off: married: literate: cleanly: teeth fair: N-3, C-3 type: uncle a leper: lives in house containing 6 people: probable age of onset, 25: urine negative: malaria, more than 5 attacks: Kahn negative.

126. BUDHERAM, M. 45, origin United Provinces: Teli: oil-worker: poor, independent: married: illiterate: not cleanly: teeth bad: N-3 type: no history of contact: lives in house containing 17 people: probable age of onset, 20: urine negative: Kahn positive 'one plus'.

127. ANYAPPA SANTU, M. 30, origin Belgaum: Wadari mendicant: beggar: destitute: single: illiterate: not cleanly: teeth fair: C-3 type: no history of contact: no housing: probable age of onset, 15: urine negative: malaria, more than 5 attacks: Kahn negative.

128. SAKUBAI TAI, F. 50, origin Sholapur: Brahman: housewife: poor, dependent: widow, 6 children: illiterate: cleanly: teeth fair: N-3 type: sister a leper: lives in house containing 20 people: probable age of onset, 20: urine negative: malaria, more than 5 attacks: Kahn positive 'one plus'.

129. GANGUTAI RAIKAR, F. 55, origin Poona City: Brahman: housewife: poor, dependent: widow, 4 children: illiterate: cleanly: teeth bad: N-3, C-3 type: no history of contacts: lives in house containing 11 people: probable age of onset, 25: urine negative: malaria, more than 5 attacks: Kahn negative.

130. RADHABAI YENKAMA, F. 25, origin Bijapur: Lingayat Wani: domestic servant: poor, independent: single: illiterate: not cleanly: teeth fair: N-2 type: no history of contact: lives in single room containing 4 people: age of onset, 20: urine negative: Kahn negative.

131. NAGIBAI LAXMAN, F. 25, origin Sholapur: Maratha: field worker: poor independent: single: illiterate: C-2 type: no history of contact: lives in house containing 17 people: probable age of onset, 19: urine negative: Kahn doubtful positive.

132. JANKIBAI MARWADI, F. 30, origin Satara: Marwadi: housewife: comparatively well-off: widow, 2 children: illiterate: is an opium addict: not cleanly: teeth bad: N-3 type: contacts with fellow opium-addict who was a leper: lives in house containing 19 people: age of onset 11 years: urine, positive for albumin and casts: Kahn positive 'one plus'.

133. SARZABAI, F. 60, origin Nagpur: Shimpi: tailor: comparatively well-off: widow, 5 children: illiterate: not cleanly: teeth bad: N-3 type: no history of contact: lives in single room containing 8 people: probable age of onset, 32: urine, negative: malaria, more than 5 attacks: Kahn negative.

134. BHUMIBAI LINGANA, F. 20, origin Daulatabad, Nizam's Dominions: Kamati: domestic servant: poor independent: single: illiterate: cleanly: teeth good: C-1 type: no history of association with lepers: lives in single room containing 4 people: probable age of origin, 15: urine, positive for albumin and casts: Kahn positive, 'one plus'.

135. GOVINDMA, F. 50, origin Madras Presidency: Tamil Naidu: housewife: self-supporting: widow, 4 children: illiterate: cleanly: teeth fair: C-3 type: mother a leper: lives in house containing 7 people: probable age of onset, 30: urine negative: Kahn positive, 'one plus'.

136. BHAGAJI BALA, M. 45, origin Dehu: Christian of outcaste origins: sweetseller: poor, independent: single: illiterate: cleanly: teeth bad: N-2, C-1 type: no history of contact: lives in house containing 19 people: probable age of onset 30: urine negative: malaria, more than 5 attacks: Kahn negative.

137. KESHAO NARAYAN JOG, M. 30, origin Satara: Brahman: priest: self-supporting: single: literate: cleanly: teeth bad: N-3,C-1 type: contacts with lepers at temple: lives in house containing 21 people: probable age of onset, 10 years: urine negative: malaria, more than 5 attacks: Kahn doubtful positive.

138. BHIMAPPA BELAPPA, M. 30, origin Belgaum: Lingayat Wani: grain shop: self-supporting: single: illiterate: cleanly: teeth good: C-2 type: no history of contact: lives in house containing 21 people: probable age of onset, 25: urine negative: Kahn positive, 'one plus'.

139. RAMA TULA, M. 30, origin Jeur, Ahmednagar District: Christian of outcaste origins: tailor: poor, independent: single: literate: not cleanly: teeth fair: N-2 type: no history of contact: lives in single room containing 6 people: age of onset, 19: urine negative: Kahn positive, 'one plus'.

140. DAMU HONAJI, M. 12, origin Dehu: Mahar: minor, no occupation: destitute: single: illiterate: not cleanly: teeth good: N-1,C-1 type: father a leper: no housing: age of onset, 4 years: urine positive for albumin and casts: Kahn positive, 'two plus': malaria, history of 5 attacks.

141. SUBHABAI LAXMAN, F. 45, origin Malegaon: Maratha: field labourer: self-supporting: married, 6 children: illiterate: cleanly: teeth bad: N-3,C-1 type: no history of contact: lives in house containing 12 people: probable age of onset, 20: urine negative: Kahn negative.

142. MAHADU DEVRAO, M. 7, origin Poona City: Christian of outcaste origins: minor, no occupation: destitute: literate, as far as schooling has gone: not cleanly: teeth good: N-1 type: grandfather a leper: no housing: age of onset, 5: urine negative: blood film positive for malaria: Kahn negative.

143. SITARAM SHIVLAL, M. 25, origin Berar: Kalal: liquor-seller: self-supporting: single: illiterate: not cleanly: teeth fair: C-2 type: no history of contact: lives in single room containing 8 people: probable age of onset, 19: urine negative: Kahn negative.

144. TUKARAM CHIMA, M. 25, origin Poona City: Maratha: millworker: poor independent: single: illiterate: not cleanly: teeth fair: N-2,C-1 type: friend a leper: lives in single room containing 11 people: age of onset, 12: urine negative: malaria, more than 5 attacks: Kahn negative.

145. SAKUBAI RAMA, F. 45, origin Thana: Mahar: coolie: destitute: widow, 3 children: illiterate: not cleanly: teeth bad: N-3 type: no history of contact: no housing: probable age of onset, 17: urine negative: malaria, 5 attacks: Kahn negative.

146. TATYOBA BHAU, M. 12, origin Kolhapur: Mang: destitute: minor, no occupation: illiterate: not cleanly: teeth fair: C-1 type: uncle a leper: no housing: age of onset, 10: urine, positive for albumin and casts: Kahn positive, 'three plus'.

147. JOSEPH MATHEW, M. 20, origin Madras Presidency: Christian of outcaste origins: student: poor dependent: single: literate: cleanly: teeth fair: C-2 type: grandmother a leper: lives in house containing 12 people: probable age of onset, 9: urine negative: Kahn negative.

148. SOLOMON, M. 25, origin Orissa: Christian of Nepali descent: student: poor, dependent: single: literate: cleanly: teeth good: C-2 type: no history of contact: lives in house containing 17 people: probable age of onset, 19: urine negative: Kahn negative.

149. NARAYAN JAIRAM, M. 15, origin Bombay: Maratha: minor, no occupation: destitute: single: illiterate: not cleanly: teeth fair: N-2,C-1 type: father a leper: no housing: age of onset, 4 years: urine negative: malaria, 5 attacks: Kahn negative.

150. IRAK, M. 40, origin Kadirabad, Nizam's Dominions: Maratha: farm labourer: poor, independent: single: illiterate: not cleanly: teeth bad: N-3,C-1 type: no history of contact: lives in house containing 23 people: probable age of onset, 15 years: urine positive for albumin and casts: Kahn negative.

151. SHIDAPPA, M. 35, origin Raichur: Maratha: farm labourer: poor, independent: single: illiterate: not cleanly: teeth bad: N-2,C-2 type: no history of contact: lives in house containing 14 people: age of onset, 15: urine negative: Kahn positive 'two plus'.

152. YENKAMMI NURSINGH, F. 35, origin Hyderabad: Teli: oilseller: poor, dependent: married, 4 children: illiterate: cleanly: teeth fair: N-3 type: no history of contact: lives in house containing 19 people: age of onset, 16 years: urine negative: Kahn positive, 'two plus'.

153. TANHIBAI SHANKAR, F. 20, origin Miraj: Christian of Mang origin: domestic servant: poor, independent: single: illiterate: cleanly: teeth good: N-2 type: aunt a leper: lives in house containing 11 people: probable age of onset, 12 years: urine negative: Kahn positive, 'three plus'.

154. AVDA SAYAJI, F. 40, origin Alibag, Kolaba District: Christian of outcaste origins: housewife: poor, dependent: widow, 4 children: illiterate: cleanly: teeth bad: N-2, C-3 type: no history of contact: house containing 10 people: probable age of onset, 26: urine negative: malaria, more than 5 attacks: Kahn positive 'four plus'.

155. BHAGUBAI MAKAJI, F. 40, origin Aurungabad: Kamati: shepherd: self-supporting: married, 5 children: illiterate/

illiterate: not cleanly: bad teeth: N-1,C-3 type: no history of contact: lives in house containing 13 people: probable age of onset, 20: urine negative: malaria, more than 5 attacks: Kahn negative.

156. DAULAT BALA, M. 25, origin Satara (Wai): Burud: basket-worker: poor, independent: single: illiterate: not cleanly: teeth fair: N-2,C-2 type: uncle a leper: lives in house containing 12 people: probable age of onset, 15: urine negative: Kahn positive 'four plus'.

157. TRIPAMA, M. 40, origin Belgaum: Christian of outcaste origins: potter: poor, independent: single: illiterate: not cleanly: teeth fair: N-3,C-3 type: no history of contact: lives in house containing 16 people: probable age of onset, 20: urine negative: malaria, more than 5 attacks: Kahn negative.

158. MAHADU HARI, M. 40, origin Paud: Maratha: tea-shop worker: self-supporting: married: illiterate: not cleanly: teeth bad: N-2,C-2 type: no history of contact: lives in single room containing 7 people probable age of onset, 25: urine, negative: malaria, history of more than 5 attacks: Kahn negative.

159. THOMAS NATHANIEL, M. 30, origin Travancore: Christian of outcaste origins: self-supporting: single: dispenser in chemist's shop: literate: cleanly: teeth good: N-2,C-1 type: no history of contacts: lives in single room containing 4 people: probable age of onset, 18: urine negative: malaria, 5 attacks: Kahn negative.

160. SITARAM CHOKU, M. 20, origin Paud: Mahar: coolie: poor, independent: single: illiterate: not cleanly: teeth fair: C-2 type: grandfather a leper: lives in single roomed hut containing 7 people: age of onset, 9: urine negative: Kahn positive 'four plus'.

161. JAYABAI MAVLI, F. 50, origin Poona City: Maratha: field labourer: poor, dependent: widow, 6 children: illiterate: not cleanly: teeth bad: N-3 type: no history of contact: lives in house containing 11 people: probable/

probable age of onset, 25 years: urine positive for albumin and casts: Kahn positive, 'four plus'.

162. RADHABAI KRISHNA, F. 35, origin Goa: Sonar: domestic servant: poor independent: married, 3 children: illiterate: cleanly: teeth fair: N-2,C-3 type: no history of contact: lives in single room containing 5 people: age of onset, 22: urine, negative: malaria, more than 5 attacks: Kahn positive, 'one plus'.

163. LAXMAN ANYAPPA, M. 30, origin Belgaum: Ramoshi: watchman: self-supporting: married: illiterate: not cleanly: teeth fair: N-3 type: father a leper: lives in house containing 9 people: probable age of onset, 10: urine negative: malaria, more than 5 attacks: Kahn negative.

164. NAMA DHARMA, M. 35, origin Roha, Kolaba District: Maratha, (Agarhi): farm labourer: self-supporting: single: illiterate: not cleanly: teeth fair: N-1, C-2 type: no history of contact: lives in single room containing 3 people: probable age of onset, 30: urine negative: Kahn positive 'three plus'.

165. POCHA, M. 30, origin Yaktuni, Nizam's Dominions: Dhobi: washerman: poor independent: single: literate: cleanly: teeth fair: N-3 type: no history of contact: lives in single room containing 12 people: age of onset, 10 years: urine, negative: Kahn negative.

166. BABU BALA, M. 27, origin Poona City: Maratha: motor bus driver: self-supporting: single: literate: not cleanly: teeth fair: C-2 type: brother a leper: lives in single room containing 6 people: probable age of onset, 24: urine negative: malaria, more than 5 attacks: Kahn doubtful positive.

167. SATWA RAOJI, M. 35, origin Purundhar: Koli: fisherman: poor, independent: single: illiterate: not cleanly: teeth fair: N-3 type: no history of contacts: lives in house containing 13 people: age of onset, 14 years: urine, negative: malaria, 5 attacks: Kahn positive 'one plus'.

168. RAGHU GOPAL, M. 40, origin Dhond: Koli: fisherman: poor, independent: married: illiterate: no information as to contact: lives in house containing 9 people: probable age of onset, 19: urine negative: malaria, more than 5 attacks: Kahn positive 'four plus': not cleanly: teeth fair: N-3,C-3 type.

169. SANTU ZOGU, M. 45, origin Rajgarh, Bhor state: Maratha: smallholder farmer: poor independent: married: literate: not cleanly: teeth bad: N-3, C-2 type: brother a leper: lives in house containing 20 people: probable age of onset, 25: urine negative: Kahn negative.

170. BALIBAI CHANDRA, F. 10, origin Roha, Kolaba District: Maratha: minor, no occupation: poor, dependent: single: illiterate: cleanly: teeth good: N-1 type: mother a leper: lives in house containing 14 people: age of onset, 9 years: urine negative: blood film positive for malaria: Kahn positive 'two plus'.

171. SHIVLINGA, F. 35, origin Bijapur: Lingayat Wani: housewife: poor dependent: married, 2 children: illiterate: not cleanly: teeth bad: N-2,C-2 type: no history of contact: lives in house containing 18 people: age of onset, 30 years: urine negative: Kahn negative.

172. RAGUNATH RAMJI, M. 12, origin Chakun: Mali: minor, no occupation: single: illiterate: not cleanly: teeth fair: N-2 type: father and mother lepers: lives in house containing 16 people: age of onset, 4 years: urine negative: Kahn negative.

173. CHAKU SHIVARI, M. 30, origin Manchar: Dhangar: shepherd: self-supporting: married: illiterate: not cleanly: teeth bad: N-3 type: uncle a leper: lives in house containing 15 people: probable age of onset, 10 years: urine negative: Kahn positive, 'three plus'.

174. KAMU BAPU, M. 25, origin Nagpur: Nagpuri Koshti: weaver: self-supporting: married: literate: not cleanly: teeth fair: N-1 type: aunt a leper: lives in house containing 26 people: probable age of onset, 20: urine negative: malaria, more than 5 attacks: Kahn negative.

175. DANIEL ABRAHAM, M. 64, origin Alibag: Beni-Israel: oilshop: self-supporting: married: literate: cleanly: teeth bad: C-1 type: no history of association with lepers: lives in house containing 19 people: probable age of onset, 30: urine negative: malaria, more than 5 attacks: Kahn negative.

176. ANUSAYABAI ALISAHEB, F. 16, origin Sholapur: Mahommedan: millworker: poor, independent: single: illiterate: not cleanly: teeth fair: C-2 type: mother a leper: lives in a single room containing 14 people: age of onset, 14: urine negative: Kahn positive, 'three plus'.

177. TAIBAI RANZANA, F. 45, origin Purundhar: Christian of outcaste origins: milkwoman: poor, independent: married, 2 children: illiterate: cleanly: teeth bad: N-2 type: mother a leper: lives in house containing 8 people: probable age of onset, 10: urine negative: Kahn negative.

178. GITABAI DHONDI, F. 45, origin Sholapur: Maratha: millworker: self-supporting: married, 5 children: illiterate: not cleanly: teeth bad: N-2,C-2 type: no history of contact: lives in single room containing 8 people: probable age of onset, 19: urine negative: Kahn negative.

179. GANGUBAI NAIDU, F. 30, origin Sholapur: Telegu Naidu: millworker: self-supporting: married, 2 children: illiterate: not cleanly: teeth fair: N-2 type: no history of contact: lives in single room containing 4 people: probable age of onset, 14 years: urine negative: Kahn positive 'three plus'.

180. KARONA SAMUEL, F. 30, origin Poona City: Christian of outcaste origins: millworker: poor, dependent: married, 1 child: literate: cleanly: teeth fair: N-1 type: no history of contact: lives in single room containing 3 people: urine, negative: malaria, more than 5 attacks: Kahn negative: probable age of onset, 24.

181. VINAYAK MOOLAY, M. 60, origin Poona City: Brahman: clerk: comparatively well-off: married: well-educated: cleanly: teeth bad: N-2,C-1 type: no history of contact: lives in house containing 21 people: probable age of onset, 35: urine negative: malaria, more than 5 attacks: Kahn negative.

182. GANGUTAI PANDURANG, F. 40, origin Poona City: Brahman: housewife: poor, dependent: widow, 5 children: illiterate: cleanly: teeth fair: N-3 type: mother a leper: lives in house containing 18 people: probable age of onset, 15: urine negative: Kahn positive 'four plus'.

183. ROSEMARY, F. 25, origin Travancore: Christian of outcaste origins: domestic servant: poor dependent: married, 2 children: literate in her own vernacular: cleanly: teeth good: C-2 type: uncle a leper: lives in single room containing 4 people: probable age of onset, 10 years: urine negative: Kahn positive 'three plus'.

184. RAKMABAI YESU, F. 50, origin Poona City: Maratha: millworker: poor, dependent: widow, 3 children: illiterate: not cleanly: teeth fair: N-3 type: no history of contact: lives in single room containing 10 people: probable age of onset, 20: urine negative: malaria, more than 5 attacks: Kahn negative.

185. BHIMABAI LAXMAN, F. 50, origin Thana: Christian of Maratha origins: millworker: poor, dependent: widow, 3 children: illiterate: cleanly: teeth bad: N-3 type: sister a leper: lives in single room containing 6 people: probable age of onset, 24: urine negative: Kahn positive 'three plus'.

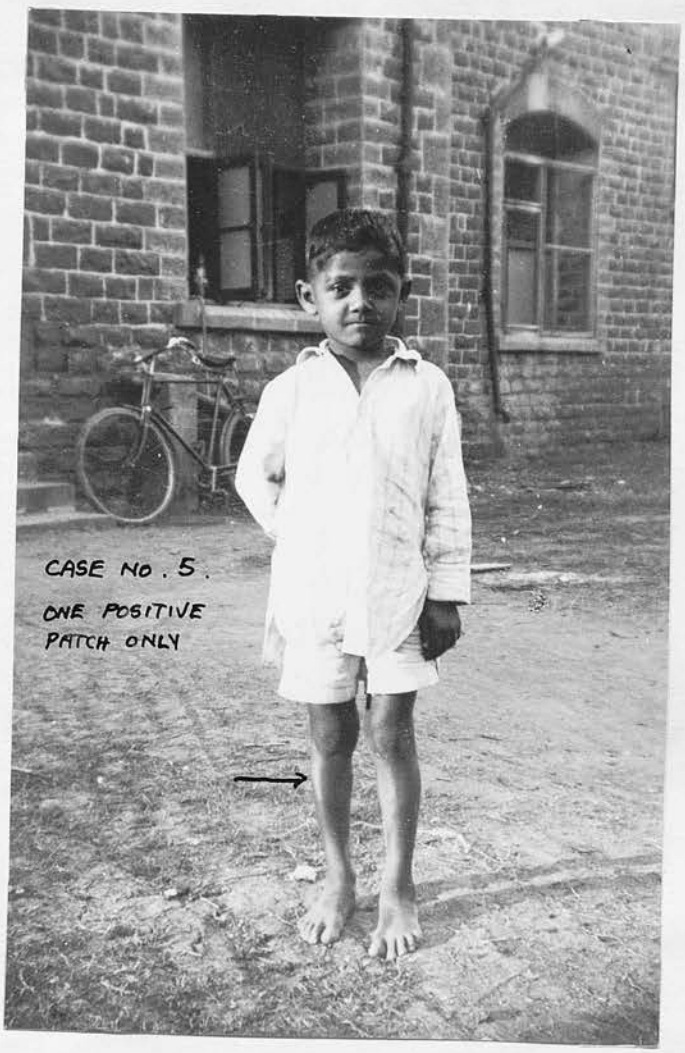
186. THAKUBAI SAMBHU, F. 50, origin Shiroli, Kolaba District: Christian, formerly Koli: housewife: destitute: married, no children: illiterate: not cleanly: teeth fair: N-3 type: no history of contact: no housing: probable age of onset, 20 years: urine negative: malaria, more than 5 attacks: Kahn negative.

187. SAKHUBAI LAXMAN, F. 50, origin Bhor: Maratha: field worker: destitute: widow, 4 children: illiterate: not cleanly: teeth bad: N-3,C-2 type: no history of contact: lives in single room containing 12 people: probable age of onset, 27: urine negative: Kahn negative.

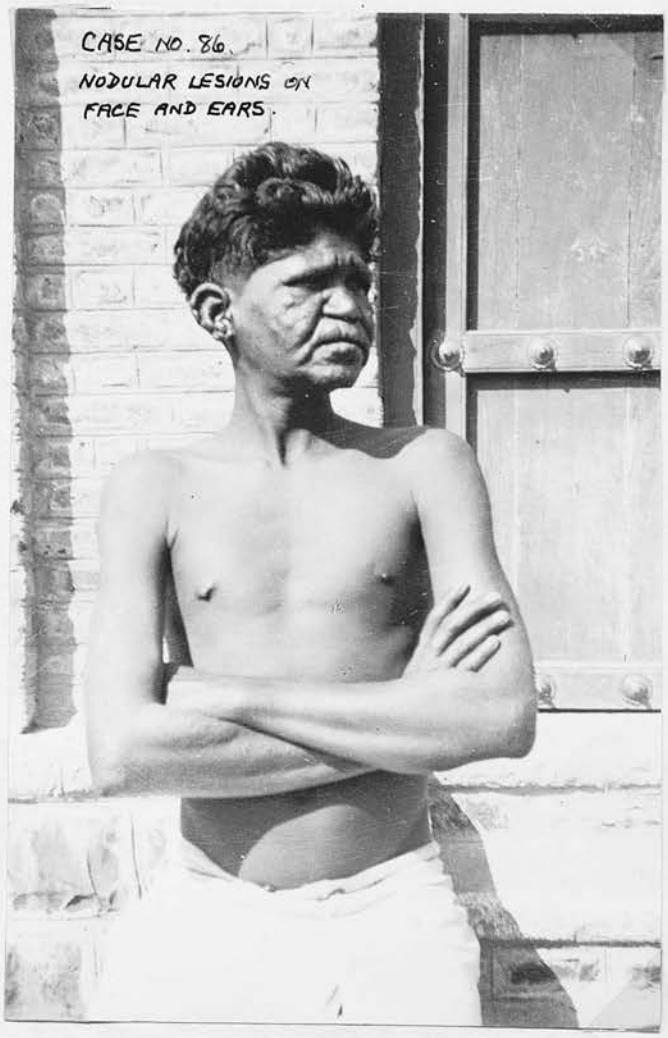
188. MUKTABAI SHRIPATI, F. 55, origin Poona City: Maratha: field worker: poor, dependent: widow, 3 children: illiterate: not cleanly: teeth bad: N-3 type: mother a leper: lives in house containing 30 people: probable age of onset, 25 years: urine negative: Kahn positive 'four plus'.

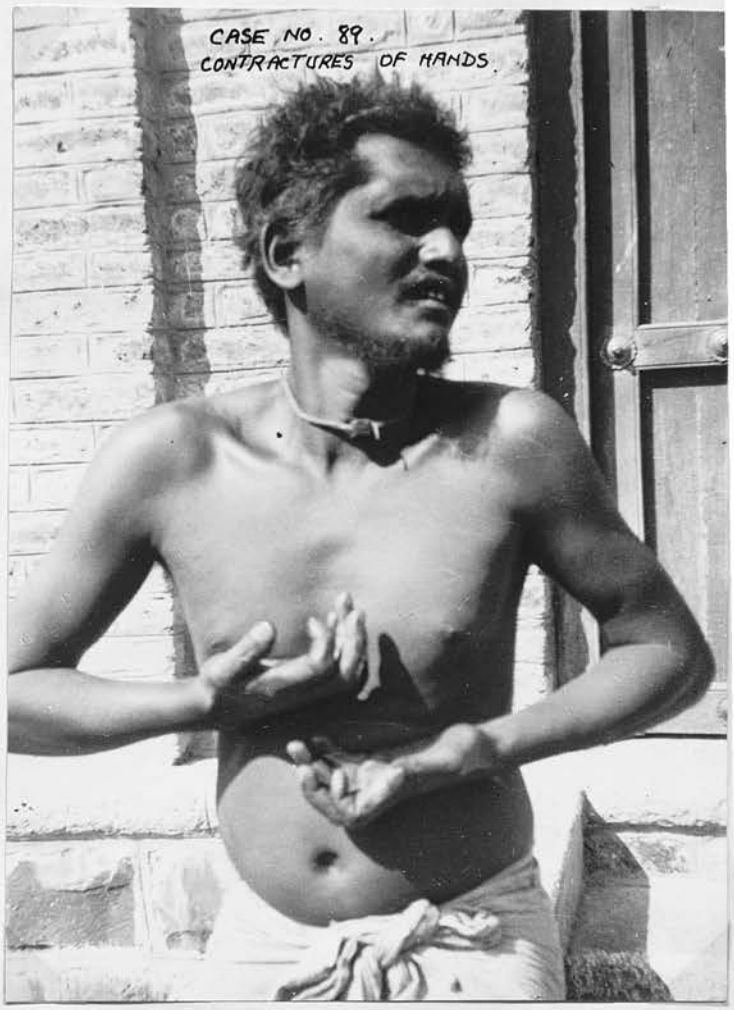
189. APPA YELLAPPA, M. 25, origin Belgaum: Lingayat Wani: mason: poor, independent: single: illiterate: not cleanly: teeth fair: N-2,C-1 type: no history of contact: lives in house containing 15 people: probable age of onset, 13 years: urine, negative: Kahn positive 'two plus'.

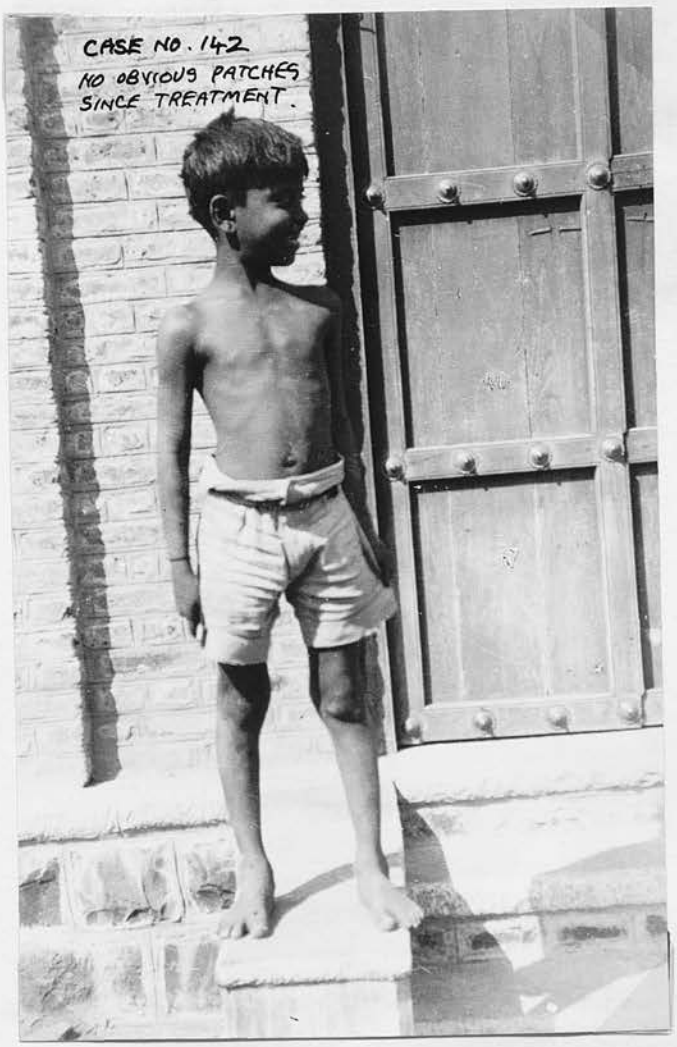
190. GENU HANGE, M. 50, origin Bharatgaon, Bhor State: Maratha: smallholder farmer: self-supporting: married: literate: not cleanly: teeth bad: N-3 type: brother a leper: lives in house containing 12 people: probable age of onset, 35: urine negative: malaria, more than 5 attacks: Kahn negative.


CASE NO. 5.
ONE POSITIVE
PATCH ONLY

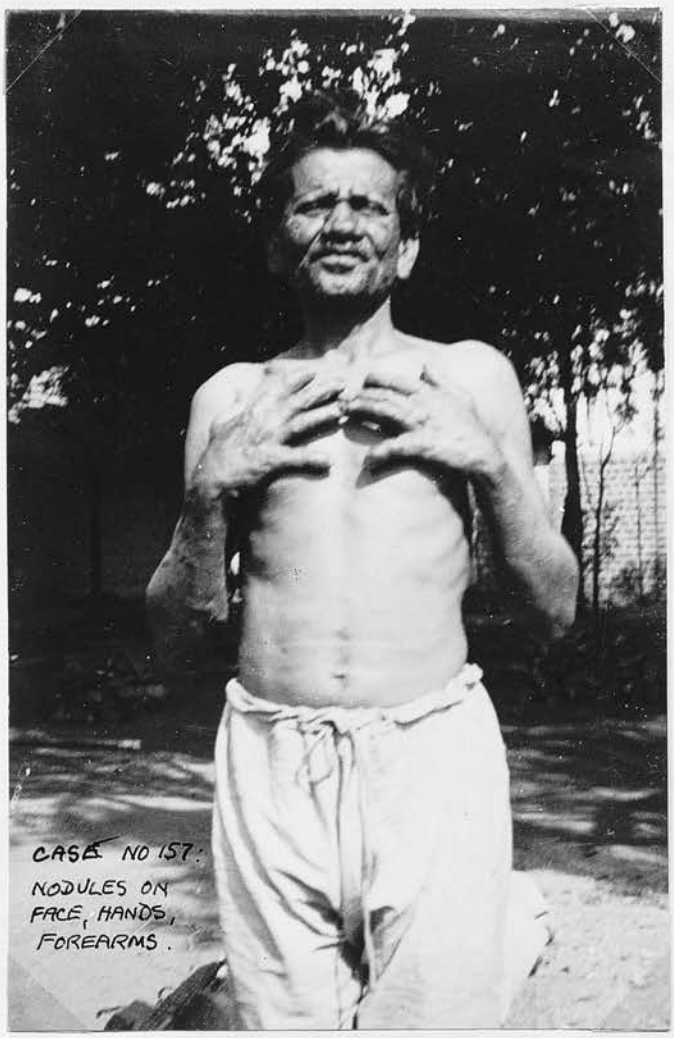

CASE NO.
50.
DIRT AND
NEGLECT
WERE
MARKED.

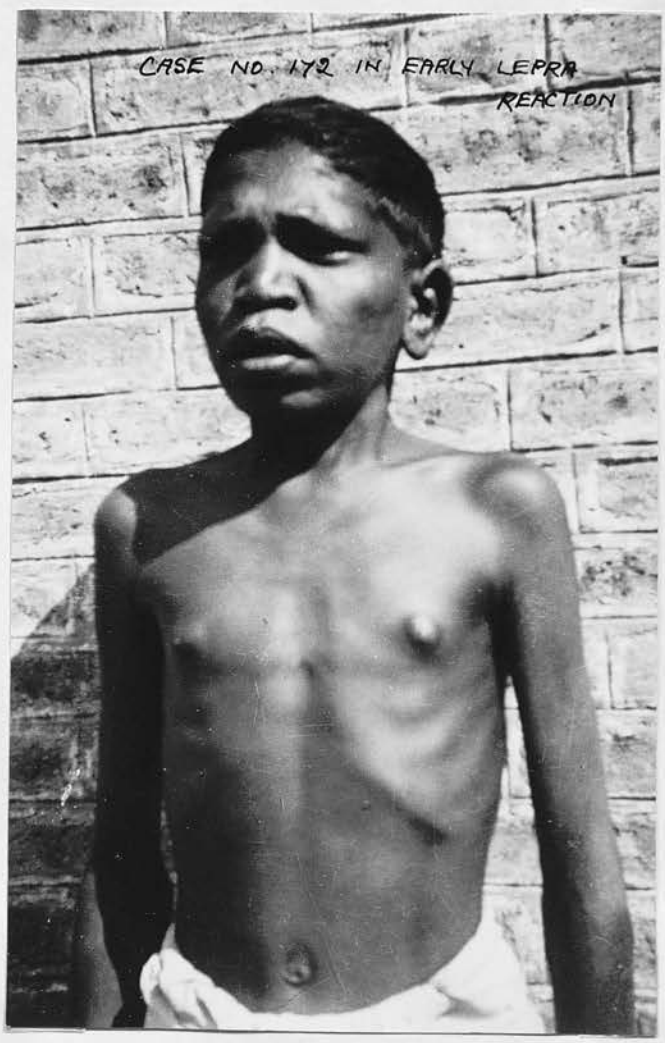

CASE NO. 86.
NODULAR LESIONS ON FACE AND EARS.

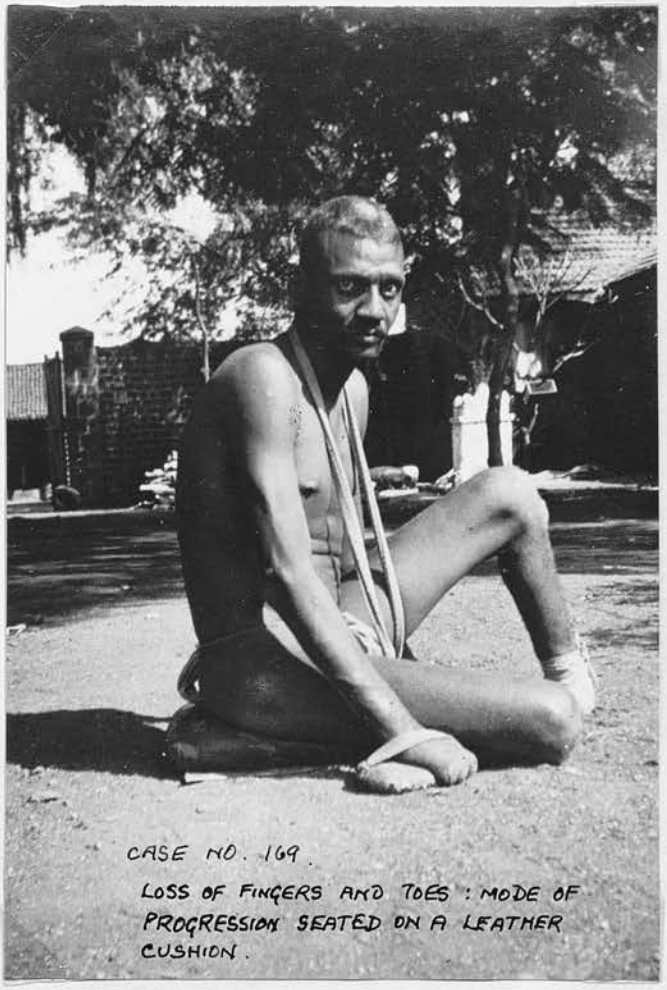

CASE NO. 89.
CONTRACTURES OF HANDS.



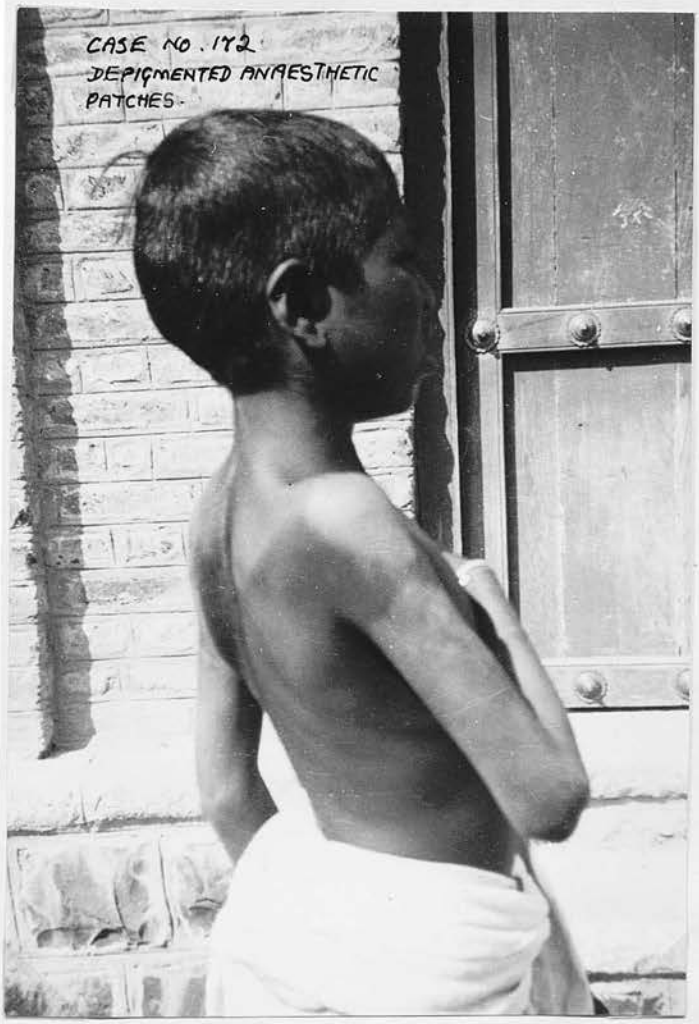
CASE NO. 118
N-2, C-2 LEPER WOMAN
WITH CHILD OF 10 MONTHS FROM WHOM SHE
REFUSES SEPARATION.


CASE NO 157:
NODULES ON
FACE, HANDS,
FOREARMS.


CASE NO. 172 IN EARLY LEPRA
REACTION



CASE NO. 169.
LOSS OF FINGERS AND TOES : MODE OF PROGRESSION SEATED ON A LEATHER CUSHION.

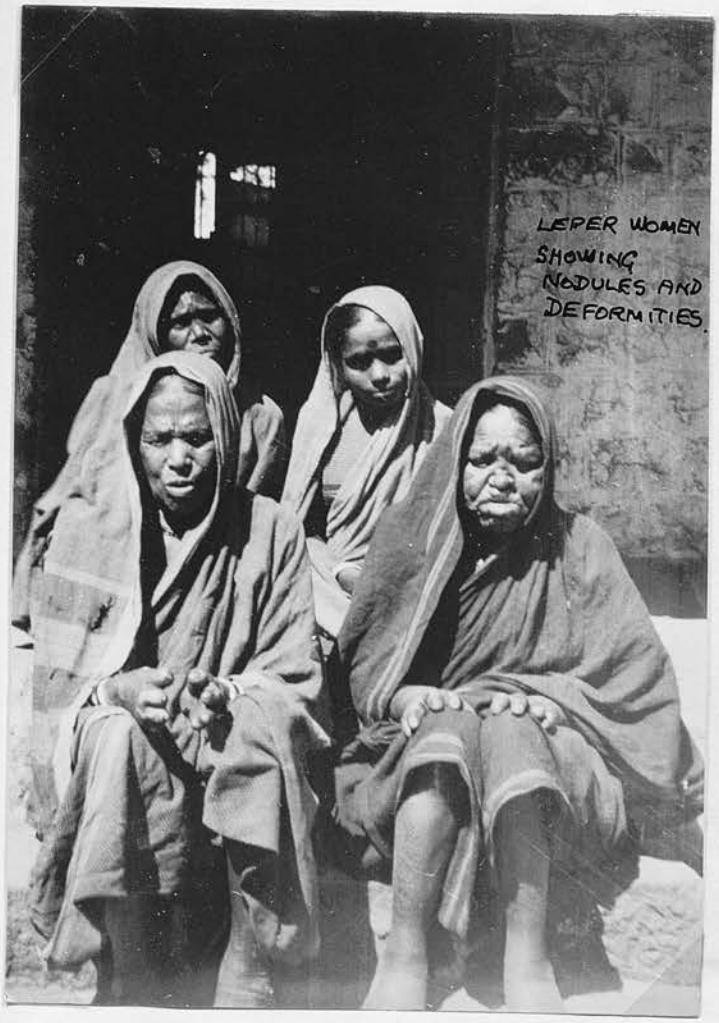


CASE NO. 172.
DEPIGMENTED ANAESTHETIC PATCHES.

CASE NO. 181.

CASE NO. 181. TRUE PSORIASIS IN A LEPER (N-2, C-1)

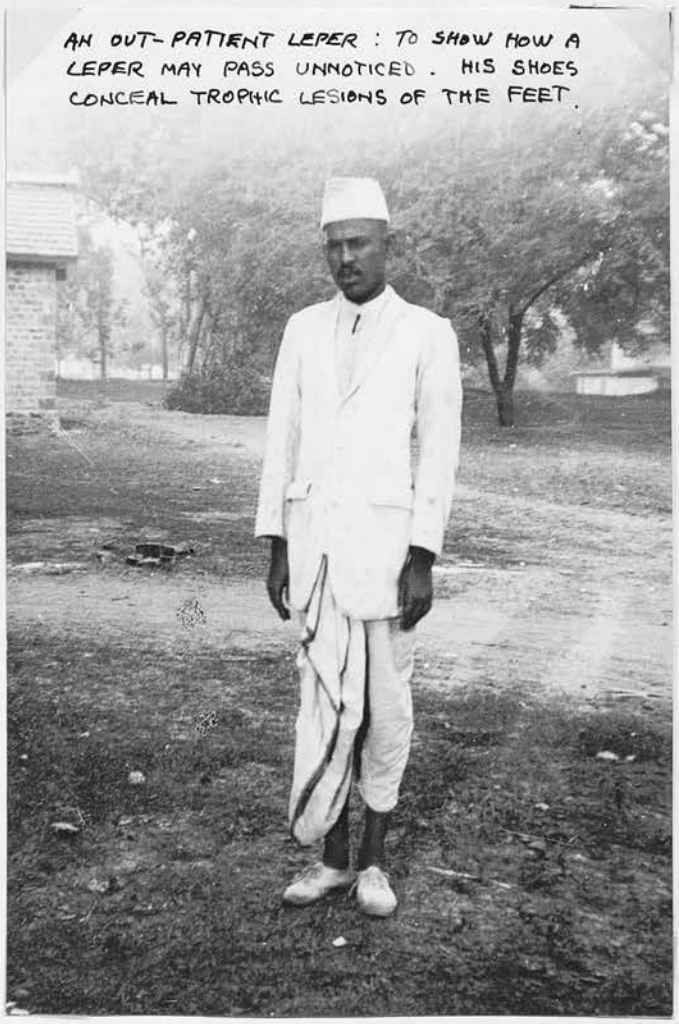
CASE NO. 181



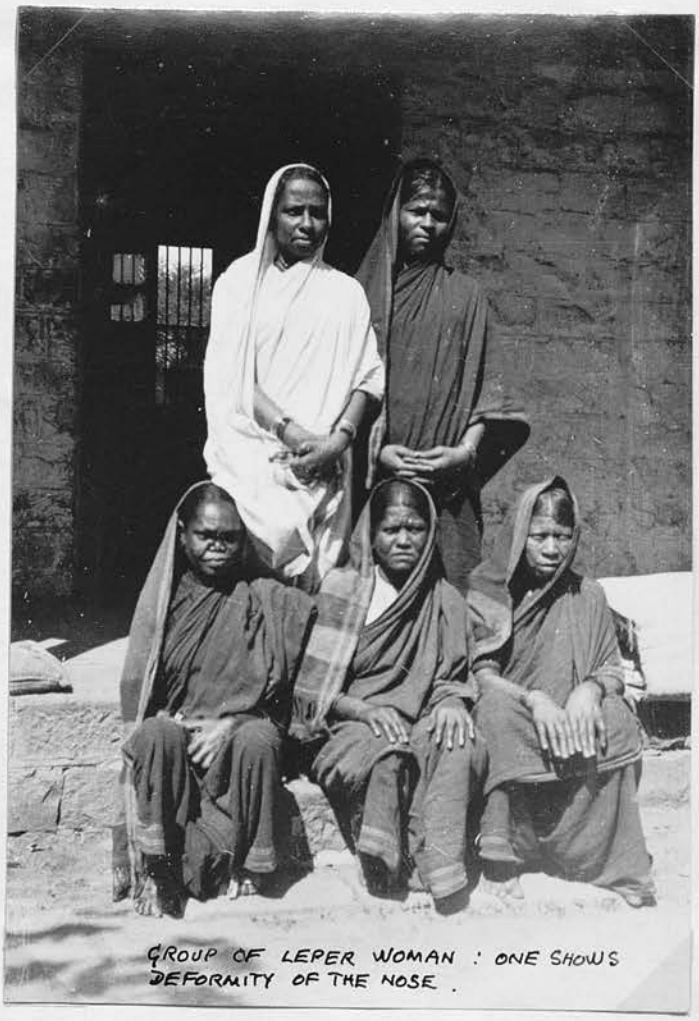
LEPER WOMEN SHOWING NODULES AND DEFORMITIES.

LEPER MOTHERS WITH CHILDREN FROM WHOM THEY REFUSE TO BE SEPARATED.

GROUP OF LEPER WOMEN: ALL ARE DEFORMED, BUT MANY SUCCEED IN HIDING IT.

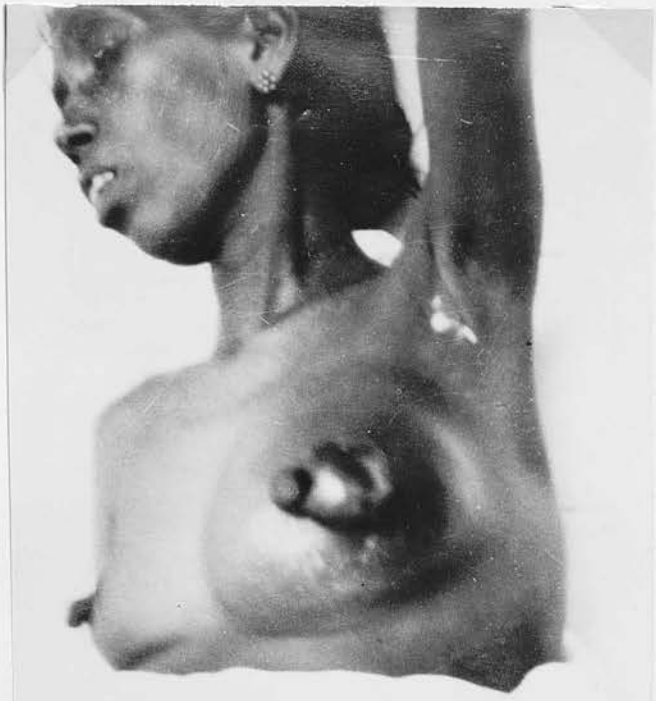


AN OUT-PATIENT LEPER : TO SHOW HOW A LEPER MAY PASS UNNOTICED. HIS SHOES CONCEAL TROPHIC LESIONS OF THE FEET.



GROUP OF LEPER WOMAN : ONE SHOWS DEFORMITY OF THE NOSE.

LEPER WOMEN, WITH DEFORMITIES.



THE FRIENDS OF THIS PATIENT MISTOOK THE SCAR IN THE AXILLA FOR LEPROSY : SHE WAS FOUND TO BE SUFFERING FROM CHRONIC SYPHILITIC MASTITIS AND ADENITIS, AND NO LEPROSY.