LEPROSY REVIEW

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THE BRITISH EMPIRE LEPROSY RELIEF ASSOCIATION.

VOL. X. NO. 4. OCTOBER, 1939.

Principal Contents:

The Distrinution of Leprosy in North-West China.

Fungus Infections in Leprosy

The Record of a Leprous Village.

Resistance and the Typing of Leprosy Skin Lesions.

Reviews.

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EDITORIAL

One of the dangers of the war is that it should be allowed unnecessarily to paralyse our efforts to help those in need and distress. While it is necessary that the exigencies of the war should receive first place, the claim of the fight against disease and distress should not be forgotten. The daily work in a leper settlement may need as much pluck and patience as fighting in the trenches. The British Colonies have one and all shown their loyalty to the Mother Country in this time of danger, and our Association will do its utmost to maintain the fight against leprosy and to help our fellow subjects in the colonies afflicted with this disease. In this we trust that we shall continue to receive the support of the British public.

* * *

SUGGESTIONS FOR INVESTIGATIONS IN LEPER SETTLEMENTS

The writer has from time to time been consulted by those in charge of leper settlements as to what investigations they could usefully undertake in the course of their daily work. Facilities for complicated laboratory research are available only to the few who have the necessary leisure, apparatus and trained staff; but there are many lines of investigation which the busy settlement doctor can fit in along side his daily clinical and administrative work. With rich clinical material at hand, with his knowledge of the people, and the good will and confidence he has already won, he is in a position to gather facts and try out living experiments which would be difficult or impossible to the laboratory worker, or the pure scientist.

The intensity of the instinct which prompts research varies in different individuals, but many doctors, once they realise that a problem is within their power to investigate, are glad to spend time and energy on its solution, and in doing so find the interest and the scope of their ordinary routine amplified.

The Leonard Wood Memorial Conference on Leprosy in 1931 made a number of suggestions on research into epidemiology, clinical studies and therapeutic experimentation, diet, etiology and pathogenesis, biochemistry and pharmacology. While many of these problems require special laboratory facilities, there are some which are quite within the scope and opportunities of the leper settlement doctor.

Epidemiology. Under this heading the Leonard Wood Memorial Conference Report states :—" It is believed that studies in the epidemiology of leprosy are greatly needed. There are serious defects in our knowledge of the incidence with regard to race, geography, environment, occupation, climate, family history, age, sex, and diet; also the incubation, duration, apparent spontaneous disappearance, incidence among healthy attendants in leprosaria, spread in newly invaded areas, apparent immunity in certain districts or areas as compared with others, etc." The more recent International Congress in Cairo has furnished most useful recommendations for epidemiological investigation (*Leprosy Review* IX-4, Oct. 1938, p.152-6). The inexpert worker will find these recommendations a guide as to how he should set about a survey in his own area; and when several surveys based upon a uniform standard have been completed it should be possible to compare accurately leprosy as it exists in different areas and countries. Such accurate comparisons have never yet been possible; when they are available they may form a basis for investigating the obscure causes of variation in the types and severity of the disease. Take for instance ten per cent of lepromatous cases in the Belgian Congo (*Leprosy Review*, X-1, Jan. 1939, p.25) compared with 56 per cent in Burmese villages (*Leprosy Review*, X-3, July 1939, p.189). These are only rough calculations made during hurried visits to limited areas, but they suffice to show the great need for careful and detailed survey along standard lines.

What is the cause of higher resistance in one community as compared with another? Many suggestions have been made, such as inherited resistance due to gradual elimination of susceptibles throughout many generations; diet or food poisoning (*Leprosy Review*, X-2, Apr. 1939, p.112-4); and climate. There seems little doubt that leprosy is particularly severe in those of mixed European and Indian race; but it is difficult to say whether that is due to any physiological pecularities or to the general adverse conditions under which Anglo-Indians so often live. Among patients admitted to the Pretoria Leper Institution, almost a hundred per cent. of Europeans and only twenty-five per cent. of Africans are lepromatous in type; yet they have lived in the same climate and malnutrition is perhaps equally common in both communities. What is the reason?

The Lepromin Test (*Leprosy Review*, V-2, Apr. 1934, p.83) is simple to carry out, and is the one test in leprosy which can lay claim to specificity. Dr. Rotberg's article reviewed in this journal (*Leprosy Review*, X-2, Apr. 1939, p.130-2) suggests many useful applications of this test in investigating the question of resistance.

Dr. Hayashi's *Age Distribution Curve in Leprosy* appearing in the International Journal of Leprosy, reviewed in this Journal, (*Leprosy Review*, X-3, July 1939, p.192) might be profitably repeated wherever leprosy is common. This paper raises the interesting question : does the age curve move to the right and the average age of lepers increase as the disease tends to die out of a community?

On page 208 of this issue, an article is republished which shows a method of conducting and recording a survey in a limited area, and dealing particularly with family history. In addition to the general value of the information gathered, such a survey may be used with considerable effect in persuading the inhabitants immediately concerned to take precautionary measures against the spread of leprosy infection.

This number also includes a note on the *Distribution of Leprosy in N.W. China*, showing a wider and less detailed survey which has gathered together valuable information.

The article by Keil on *Hereditary Factors in Leprosy* in our last issue suggests another possible line of approach to the question of immunity.

We would also refer to the survey of the Solomon Islands by J. Ross Innes, appearing in this Journal in July, 1938, and the *Field Study of Leprosy in Cebu*, by Doull and others, in the *International Journal of Leprosy*, IV-2, Apr.-June, 1936, both of which are full of useful suggestions.

Clinical Studies. The clinical manifestations vary in different countries, both in nature and in proportion. For instance, in West Africa doubts have been expressed by workers as to whether certain macules were of the neural or lepromatous type, and whether they were residual or active. In recent years very careful and detailed studies of leprides, or leprous macules, have been made by several workers, and especially by Wade, and recorded in the *International Journal of Leprosy*. A short paper on page 221 of this issue seeks still further to simplify this subject. With the help of these descriptions it should be possible to classify most cases according to the Cairo Congress classification. In doubtful cases, however, where clinical and bacteriological examinations still leave doubt, histological examination is necessary. Many of our readers have no laboratory facilities; but it is not difficult for them to collect material by biopsy and send it to those who would be willing to carry out and report on such examinations. Remove a small eliptical piece of skin, making incisions deep enough to include subcutaneous tissue. Place this tissue in 70 per cent alcohol and

change the alcohol after 6, and again after 24 hours. Then send in a tightly corked tube, lightly stuffed with cotton, and with enough alcohol to saturate the cotton. An accurate description of the patient and of the lesion, and if possible a close-up photograph, should accompany the tissue. Once there is certainty of the types and subtypes of lesions it is important to make accurate classifications and to compare the differences of types in each area.

The onset of leprosy in children is another subject which requires careful and prolonged study. There is still uncertainty about the earliest signs in children and these vary in different countries according to the colour of the skin and other factors. A careful record, with repeated examinations extending over a period of years, is necessary to clear up these points. For guidance on this subject see the article by Cochrane and Rajagopalan (*International Journal of Leprosy*, VI-3, July-Dec. 1938, p.325).

In an editorial in the January-March, 1939, number of the *International Journal of Leprosy*, Professor Marchoux refers to this subject, in connection with the future of non-leprous contacts in which gland puncture reveals lepra bacilli. "This can be ascertained," he says, "only by leprologists who observe their patients for years, or by those who, by keeping records of the results of their examinations, will enable their successors to find out what becomes of an infection verified by a puncture."

The lepromin test has already been referred to above; it should be of great value in combination with clinical studies.

Therapeutic Experiments. Doctors in charge of leper settlements are in a unique position for testing various forms of treatment. It is always well first of all to become familiar with standard methods, chaulmoogra oil or esters being used for special treatment and the general condition of the patient being toned up by removal of complications and by well-regulated regimen. But on the standard treatment as a basis there are many forms of treatment, both for the disease itself and for complications which can be tried out.

The sedimentation test, carried out regularly at weekly or two-weekly intervals, has been found by those who have given it a fair trial to be an invaluable help in regulating treatment and eliminating complicating conditions. With the aid of this test it is possible to make tentative experiments with other drugs without undue risk to the patient, since the slightest deterioration of health which may occur as the result of the treatment is recorded in the most delicate manner by the sedimentation index. The most powerful reaction producer in leprosy is potassium iodide,

so much so that some authorities consider it should not be used at all. The writer, however, is of the opinion that when used as he has recommended (*Lep. Diag. Treat. and Prev.*, Sixth Edition, p.181) with the aid of the sedimentation test, this drug may be of great value in carefully selected cases, and it is possible that careful experimentation might still further extend its safe use.

Among the complications of leprosy which have been studied, but the treatment of which still requires improvement, are lepra reaction, trophic ulcers, nerve and bone pains.

Animal Inoculation. The study of leprosy is still handicapped by lack of suitable experimental animals. Recent reports of successful inoculation of human leprosy in the golden hamster (Cricetus auretus) still await general confirmation; but the *Rodentia* seems to be the most promising natural order. There are innumerable forms of rodents within reach of leprosy workers which could be easily caged, fed on various diets, and inoculated with leproma suspension; success might be of immense value in opening up the path for further research.

These are a few out of many problems which do not require whole-time research workers, but may be gradually solved in the course of every day routine by doctors in charge of leper institutions who are willing to co-ordinate their work, observe closely and record their observations.

* * *

CORRECTION

We regret two mistakes which occurred in *Leprosy Review*, Vol. X, No. 3, page 194. On line 3, in place of 240,000 read 24,000; on line 9, in place of 150,000, read 1,500.

THE DISTRIBUTION OF LEPROSY IN NORTH WEST CHINA

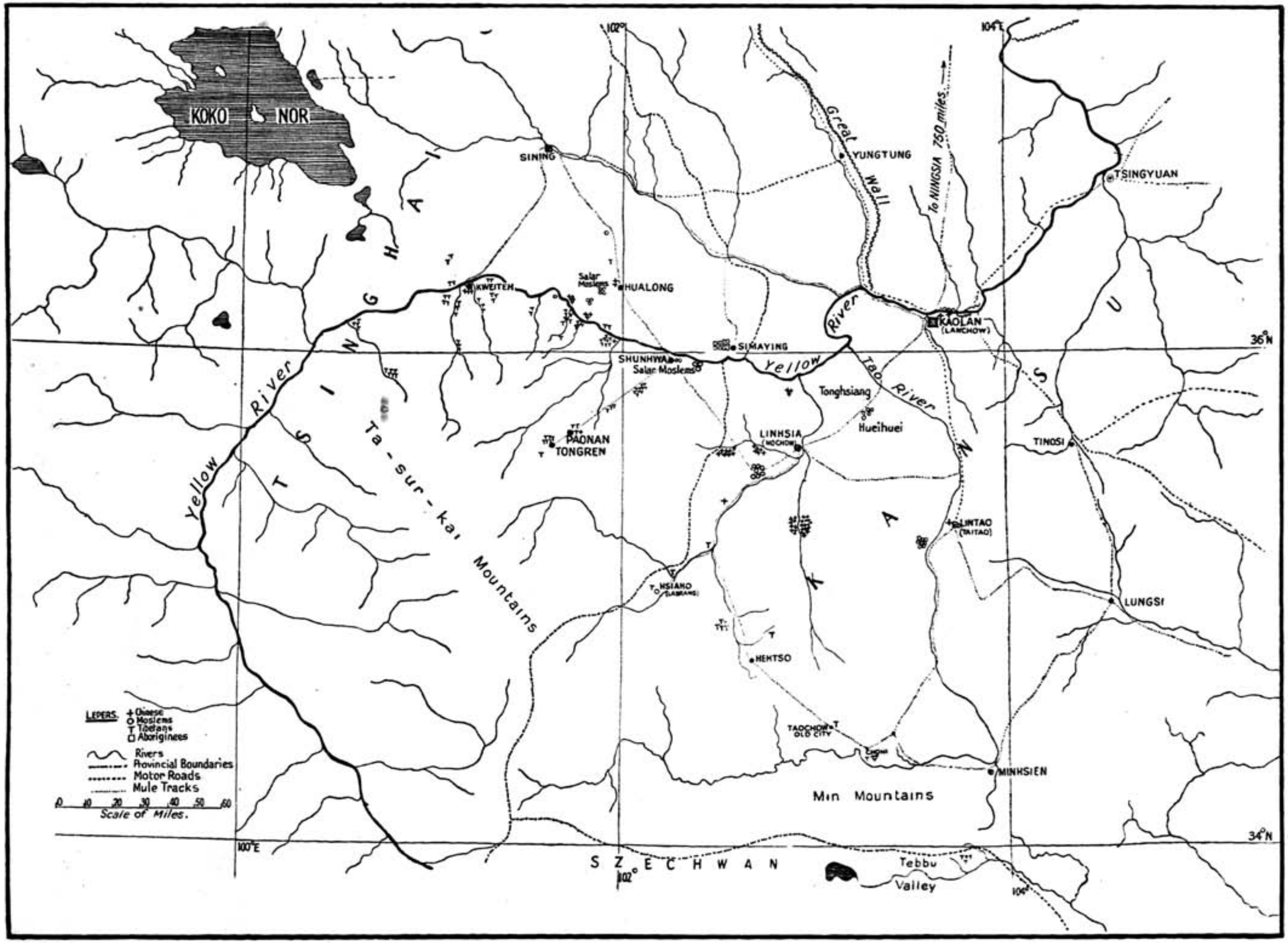
ROBERT PEARCE.

The provinces of Tsinghai and Kansu are the home of a number of races. The Chinese and Moslems have military and political supremacy throughout the area. The bulk of Tsinghai is populated by Tibetans, whose language and customs differ widely from those of their rulers. In Kansu the Chinese and Moslems predominate. The latter are of different stock from the Chinese, and among themselves own different ancestries and languages, although their religious and military strength is one. There are also a number of aboriginal tribes related to the Mongols. These races tend to live separately, and even in the larger cities it is customary to find the Moslems congregated in one of the suburbs. Chinese is the official language, but a knowledge of the local tongue is necessary if one works in the country among the natives.

The population is largely confined to the river valleys where there is watered ground suitable for raising crops. Between the rivers the mountains are high, and offer only scanty herbage for the grazing of sheep and goats. Northward of a line joining Shunwa, Linhsia, and Lintao the soil is loess. Southward, black earth and limestone rock becomes increasingly evident. In places sandstone and granite appear. At the higher altitudes the valleys broaden out into rolling grasslands which in turn give place to wild rock mountains, 15,000 to 25,000 feet high.

Moslems and Chinese occupy and farm most of the fertile tracts of land. Above them sedentary Tibetans raise a little grain which they barter with the nomad Tibetans of the grasslands for cattle and sheep. Among the sedentary Tibetans are trading posts occupied by Moslems and Chinese merchants. These are invariably situated close to Tibetan monasteries.

The staple item of diet of all except the nomad Tibetans is flour made into a dough with water, cut into strips and boiled. Vegetables are sometimes added to this meal. Coarse bread is baked or steamed, and eaten with boiling water or tea. Meat is eaten by the country Chinese on rare occasions—perhaps once or twice a year. Those living in the cities taste meat more frequently. On the whole, the Moslems eat more meat than do the Chinese, and those in the Linhsia (Hochow) district are said to eat a great deal of meat. The aboriginal tribes live on a diet poorer than that of the Chinese. The Tibetans are different again. The nomads eat vast quantities of meat, raw or partially cooked, and take a little flour mixed with tea and butter. The sedentary


KOKO NOR
SINING
YUNGTUNG
Great Wall
To NINGSIA 750 miles
TSINGYUAN
KWEITEH
Salar Moslems
HUALONG
KAOLAN (LANCHOW)
SIMAYING
SHUNHWA
Salar Moslems
Yellow River
Tonghsiang
Tao River
Hueihuei
PAONAN
TONGREN
LINHSIA
TINGSI
LINTAO (TAITAO)
TSINGHAI
KANSU
Ta-sur-kai Mountains
HSIAHO (LABRANG)
LUNGSI
HEHTSO
TAOCHOW OLD CITY
MINHSIEN
Min Mountains
SZECHWAN
Tebbu Valley
LEPERS.
Chinese
Moslems
Tibetans
Aborigines
Rivers
Provincial Boundaries
Motor Roads
Mule Tracks
0 10 20 30 40 50 60
Scale of Miles.
100°E
102°
104°E
36°N
34°N

Tibetans eat more meat than their Chinese neighbours, but are worse off as regards vegetables. In some of the districts at lower altitudes fruit is grown—apples, apricots, peaches and pears. Some of the fruit is good, but the majority is of poor quality. The bulk is sold into the cities.

Clothing is scanty, and the laws of hygiene quite unknown. Housing conditions are poor, and over-crowding is the rule. Syphilis is probably the commonest of all diseases met in this area. It is especially rife among the Tibetans, whose customs allow of a variety of "free love." Adenoma of the thyroid is also very common among the Tibetans, but it is, nevertheless, strictly confined by geographical boundaries. Tuberculosis is found in the cities, but acute specific fevers seem to take the heaviest toll of life.

These remarks apply generally to the tract of country lying south and west of Kaolan (Lanchow). All this is well above 5,000 feet. The grasslands begin between 10,000 and 12,000 feet above sea level. To the east and south of Kaolan, the standards of living are rather higher. There are no aborigines nor Tibetans here. The altitude is generally lower.

Two hundred and seventeen lepers have been treated at the China Inland Mission Hospital at Kaolan during the past fifteen years. The home location of about 200 of these is shown on the accompanying map. This Hospital is the only centre in Tsinghai and Kansu at which lepers receive care and treatment. For the most part of this period no other medical work of western standard was practised in these provinces. These lepers have mostly come from the Yellow River valley, and from the upper sources of the Yangtse, north and east of the Tasurkai mountains. Beyond this range the people are entirely nomad in their habits. No cases have come here, who have contracted the disease in the desert country north of Kaolan. Cases have been seen in Ningsia, about 700 miles north of Kaolan. This is a small area watered by the Yellow River and a system of canals, surrounded by deserts. From the south and east of the province no lepers have come here, but there is said to be a small group near Hweihsien on the Szechwan border.

There is practically no leprosy in the immediate vicinity of Kaolan. We know of one merchant in the city who has the disease, and refuses treatment! He carries on his business without hindrance. A second case has appeared in a labourer who has never been more than twenty miles up or down the river from Kaolan. He knows of no other lepers in his village, which is on an island in the river.

Travelling south from Kaolan, over a stretch of wild unfertile country, one reaches the Tao River, with Lintao (Titao) as the

chief town. We have received one leper whose home was in this city—the only case we have originating within a city. Ten miles further south from Lintao on the west bank of the river is a district peopled by Moslems, from which eight lepers have come to the Hospital. One of these lepers told us of thirty-eight other lepers known to him, living in his village. The upper part of this valley reaches into Tibetan country. The capital of the principality is Choni, where there is a large monastery, and a very small trading centre. From this place, one Tibetan and three Chinese lepers have come to us. Fifty miles further south, across the Min mountains live the Tebbu tribe of Tibetans. This tribe is partly settled and partly nomadic in its habits—mostly the former. We have four lepers from this very wild tribe, but we are told that leprosy is prevalent in the warmer parts of the valley, the water of which drains into the Yangtze.

Between the lower reaches of the Tao and the Hsia rivers, bounded on the north by the Yellow river, is a tract of loess mountain country inhabited by Moslems, known as the Tonghsiang Hueihuei. These people are of Mongolian descent, and speak a Mongolian dialect. They are farmers, but there are many little market towns. Five lepers have appeared from among this people.

The district surrounding Linhsia (Hochow) is very fertile, and is thickly populated by Moslems. Fifty lepers have come to the Hospital from this district, but only seven of these are Moslems—all from one village, about sixteen miles from the city. The remainder are Chinese farmers who live in the valleys leading out of the main Linhsia valley. Here there is less water, and the soil is less productive. Moslems are generally speaking more wealthy than the Chinese. They have bought out the Chinese from the better land nearer the city—and presumably insured themselves against contracting leprosy! Nearing the grasslands, the Hsia river divides into two, and two trading centres—Labrang and Hehtso—are situated at the river sources. From the valleys around eight Tibetan lepers have come to the Hospital.

Crossing the border into Tsinghai we find a much higher proportion of Tibetans in the population. Of the lepers coming here from Tsinghai, 75% have been Tibetan. On the border, just north of the Yellow river is a district inhabited by aborigines. These people have a Mongolian ancestry and speech. By religion they follow a lamaist type of Buddhism. They are extremely poor. Famine drove a proportion of them into the Sining district. From among these emigrants, and from the district of Si-ma-ying we have met nine lepers. No case has appeared which is native to Sining.

On the opposite side of the Yellow river is a Moslem popula-

tion having their centre at Shunhwa. These Moslems are not Chinese, but immigrants from Samarkand, and speak a corrupted form of Turkish. They are known as Salar Moslems. Five Salar Moslem lepers have been treated here. A little higher up the river, on the northern bank is another group of Salar Moslems. Racially they are descended from the Tibetans, but they have embraced the language, speech and customs of the true Salars. They are a very wild people. Eight lepers have come to the Hospital from this district. Other Chinese and Tibetan lepers have also come from this part of the country.

Between Shunhwa and Tongren the valleys support a population of sedentary Tibetans, from among whom we have met twenty lepers. Leprosy is said to be quite common in this area.

Forty miles higher up the river from Shunwa is a fertile stretch of valley known as Shui-ti-chüan. Many tributaries enter the river from the mountains on its south bank. This land is farmed by sedentary Tibetans of a very independent nature, over whom the Chinese have only nominal control. This district has sent us eleven Tibetan lepers.

The Yellow river leaves this region through a long and impassable gorge, and above is a similar gorge descending from another stretch of fertile valley having Kweiteh as its political and civic centre. Three Chinese lepers have homes in the immediate vicinity of Kweiteh, but from the surrounding valleys we have met twenty-five Tibetan lepers. Leprosy is well recognised, at least in its grosser forms by these people. In one of the valleys south of Kweiteh is a deserted village. A new village was built on the opposite bank of the river because of the prevalence of leprosy in the first village. Already two cases of leprosy have been recognised in the new village!

Above these valleys around Kweiteh the altitude forbids cultivation of the land, and the hills support only nomad Tibetans and their flocks. We have only one leper from a nomad tribe. He is a priest, and although his disease appeared while he was living as a nomad, it is possible that he contracted the infection in some monastery where he was studying.

Six other lepers have appeared here from other provinces—one from Anhwei, one from Hunan, who are both well educated men. There are two other ordinary Chinese from eastern Szechwan, one Tibetan from the Szechwan border, and two from an aboriginal tribe—the Kiarung—living in western Szechwan.

The clinical condition of 126 of these lepers is known. In the table following they are classified according to the nomenclature of the Cairo Conference of 1938. The figures in brackets are percentages.

District	Neural	Lepromatous	Total
Hochow (Linhsia) ...	11(30.4)	26(69.6)	37 (29.4)
Labrang	4(57.1)	3(42.9)	7 (5.6)
Titao (Lintao) ... and southwards	5(38.5)	8(61.5)	13 (10.3)
Si-Ma-Ying	3(50.0)	3(50.0)	6 (4.7)
Hualong	5(41.7)	7(58.3)	12 (9.9)
Hsuinhua	3(33.3)	6(66.7)	9 (7.1)
Paonan and Tongren	8(72.7)	3(27.3)	11 (8.7)
Kweiteh	16(51.6)	15(48.4)	31 (24.3)
Total	55(43.7)	71(56.3)	126(100.0)
Divided according to races the figures appear thus :—			
Chinese	15(44.1)	19(55.9)	34 (27.0)
Moslems	4(17.4)	19(82.6)	23 (18.2)
Aborigines	3(37.5)	5(62.5)	8 (6.4)
Tibetans	33(54.1)	28(45.9)	61 (48.4)
Total	55(43.7)	71(56.3)	126(100.0)

This distribution, expressed geographically or racially may not represent the true state of affairs. Generally speaking the Tibetans come greater distances. Lepromatous cases being more sick, are less likely to come. These figures confirm the general impression that leprosy is more severe among the Chinese and Moslems, than among the Tibetans.

We gladly acknowledge the help and advice that has been given to us, while writing this account, by our friends of the Christian and Missionary Alliance, who work among the people of whom we write.

FUNGUS INFECTIONS IN LEPROSY

E. MUIR.

Epidermophyton infection forms one of the most troublesome complications of leprosy in the tropics. It is found particularly in patients with widespread skin lesions of the neural type. These may have become more or less residual, leaving large flat hypo-pigmented anidrotic areas.

A relationship between anidrotic skin and fungus growth is suggested by a recent article by Peck and others on " Sweat as a

Fungicide ''.* They found that thermal sweat may have fungistatic and fungicidal properties at a pH below 7. Concentrated heat sweat is fungistatic, even when alkalinized. The fungicidal properties of sweat are due to its content of acetic, proprionic, caproic, caprylic, lactic and ascorbic acids. These substances must be present in the proper concentration to exert a fungistatic or fungicidal effect. There seems to be a relation between the localization of fungus infection and the distribution of sweat on the surface of the body. Areas which are exposed to the greatest concentration of sweat seem to have less tendency to fungus infection. Contrary to the usual opinion, there is comparatively little sweating between the toes, though moisture is retained and the skin is apt to become maserated. It may be on this account that epidermophyton so commonly begins between the toes. Topical application of ingredients of sweat, in proper concentrations, have proved valuable in the treatment of fungus infections.

It would seem likely, therefore, that the proneness of many neural type cases of leprosy in the tropics to epidermophytic disease may be due to the absence of sweat. The greater part of the trunk may be covered with tinea in a patient in whom the leprous infection has long since died out. The fungous growth is often mistaken for active leprous disease. Such cases are in marked contrast to those in whom hyperidrosis of the trunk compensates for anidrosis of the limbs; in these the trunk is remarkably free of fungus.

Widespread tinea infection in lepers, whether it cover the trunk or is confined to the limbs, is particularly difficult to treat by ordinary remedies, and Peck's paper suggests a new method of treatment that is perhaps worth trying. The sweat from other patients may be difficult to obtain in sufficient quantity, but some of the ingredients of sweat, such as acetic acid or lactic acid, might be applied in dilute form at frequent intervals. In South India, where hydnocarpus oil is stored in earthenware jars, I have been told that the old deposit of oil in the bottom of the jars is used for rubbing on the skin of lepers. As this substance is likely to be rancid oil, containing a large quantity of fatty acids, it is possible that its acidity renders it valuable in controlling the growth of fungus, while at the same time softening the hard epithelium.

A prescription that has been found very useful in treating discrete lesions of fungus (tinea versicolor, cruris, circinata, &c.)

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* Arch. of Derm. & Syph., Vol. 39, No. 1, Jan. 1939, p. 126. The Role of Sweat as a Fungicide, by S. M. Peck et al.

is as follows:—pure carbolic acid 2 parts, ichthyol 2 parts, tincture of iodine 2 parts, glycerine 4 parts, rectified spirit 4 parts. Painting on of this mixture is followed in a few days by exfoliation, after which it may be repeated if necessary.

Tinea shares with scabies the responsibility for being one of the chief localising factors of leprosy infection. Frequently lesions begin round old fungus patches, the irritation either aiding the entrance of bacilli through the skin, or fixing the infection circulating through the body.

*THE RECORD OF A LEPROUS VILLAGE

E. MUIR and K. R. CHATTERJI.

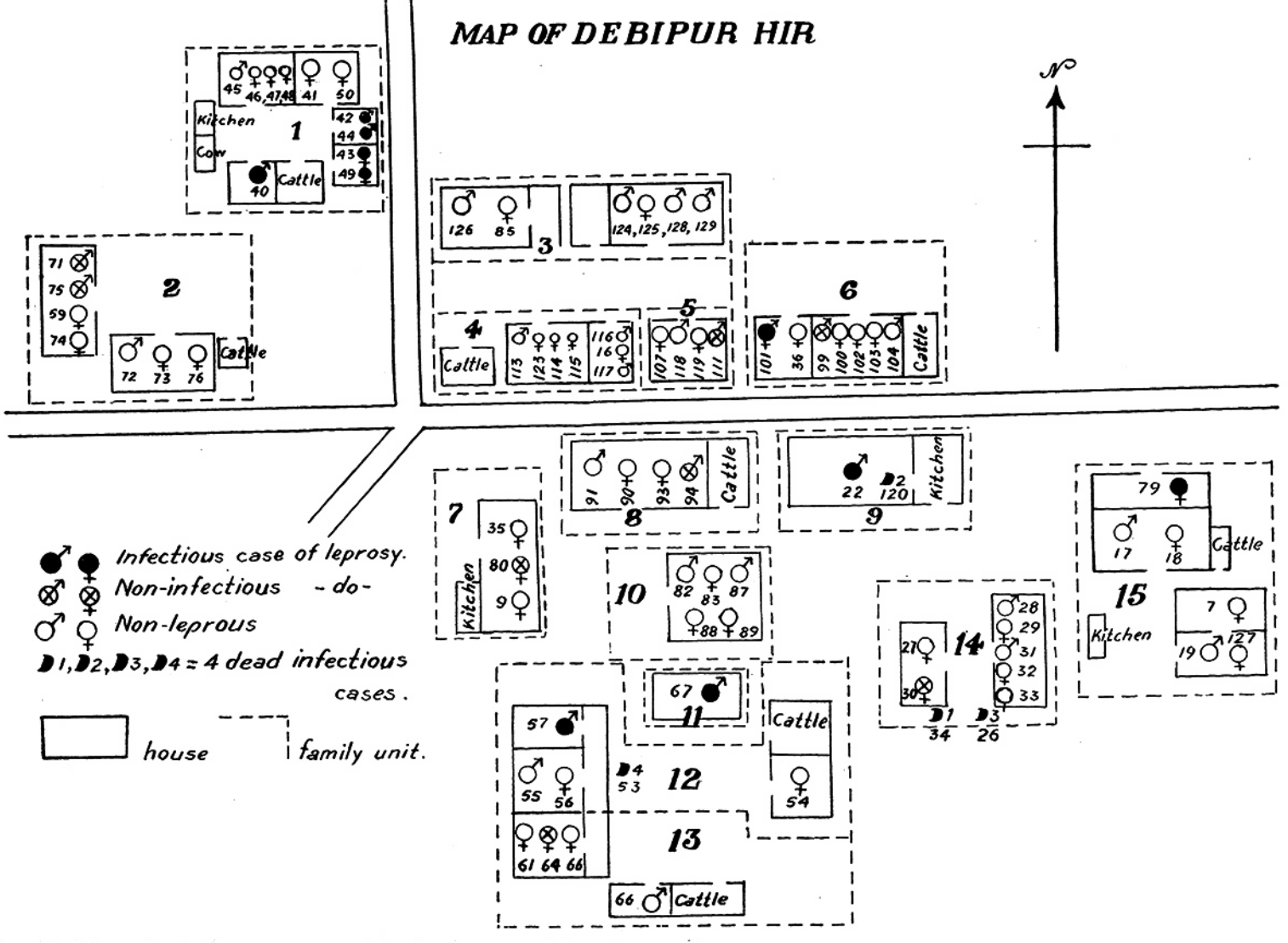
Before conducting a carefully planned and controlled statistical survey of the endemiology of a disease, it is necessary to make a preliminary enquiry in a carefully chosen and limited area, so as to arrive at a rough idea of the factors influencing the spread of the disease. This preliminary enquiry should furnish the data upon which the larger statistical survey may be based.

The village of Debipur Hir in the Bankura District of Bengal was founded about 74 years ago. It consists entirely of Mussalman families, most of which are inter-related. The records cover five generations. The first two generations were free from leprosy. The disease was apparently introduced by cases 26, 34 and 120 (see tables I and II), the first two of whom were infected by their maternal uncle in a neighbouring village, and the latter by the same individual, her husband's maternal uncle. Her husband (no. 21) probably escaped infection by being past the very susceptible age; he was 6 years older than his brother (no. 26). Case no. 120 was separated from her husband after leprosy developed, but she infected her daughter (no. 22). The latter (no. 22) however did not become infectious till her daughter (no. 15) had been married and left the village, thus escaping infection.

During the last 40 years the number of infectious cases present in the village varied as shown in the following list :—

	No. of infectious cases.		No. of infectious cases.
1894-1898	1	1912-1919	1
1898-1899	0	1919-1922	3
1899-1905	1	1922-1926	7
1905-1909	3	1926-1932	9
1909-1912	2	1932-1934	10

* The first part of a paper reprinted from *Leprosy in India*, VII-1, Jan. 1935.


MAP OF DEBIPUR HIR
N
Infectious case of leprosy.
Non-infectious -do-
Non-leprous
D1, D2, D3, D4 = 4 dead infectious cases.
house
family unit.
Kitchen
Cow
Cattle
Kitchen
Cattle
Kitchen
Cattle
Cattle
Kitchen
Cattle
Cattle
Kitchen
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15

The disease began in the third generation, and within the last 46 years 23 cases have developed in the village. Of these, 4 are dead. The remaining 19 cases may be classified as follows :—

N-1	... 3	9—Non-infectious.	
N-2	... 6		
C-1, N-2	... 1	3—Slightly infectious	10—all isolated now.
C-1, N-3	... 2		
C-2	... 4	7—Highly infectious	
C-2, N-2	... 2		
C-3	... 1		

At the present time there are 77 people in the village divided into 15 Mussalman families (see map), so that the present incidence of the disease is 24.6 per cent.

The village is a compact one and the houses are closely set together. The people are mostly day labourers, but a few cultivate their own land. The economic condition of the village is poor. There is a scarcity of drinking water and malaria is rife.

Two of the important factors in the spread of the disease are apparently family relationship and closeness of residence, both of these determining to a certain extent the degree of contact. To show the former a family tree of the two principal families has been prepared, while the latter is shown in the map of the village. The numbers in the tree and map correspond to the serial number in the general list of inhabitants (table I). This general list also gives various particulars regarding the villagers.

A second list (table II) deals with the leprous cases, and gives further details as to how and when the disease was acquired and the type of the disease at the time of examination.

Much of the historical information recorded is only approximate, as it was ascertained by cross examination of the villagers. This is particularly so with regard to the dates when cases of leprosy reached the infectious stage of the disease.

DISCUSSION.

In looking down the *source-of-infection* column in table II, one is struck by the number of cases infected by case no. 67, no less than 15 in all, though in two of these (nos. 53 and 71) case 34 was possibly a previous source of infection.

Of the 13 cases who were in continuous infectious contact from birth onwards, 6 have already become infectious cases, the estimated average age of their becoming infectious being 17½ years. The remaining 7 cases are still non-infectious, but their average age at present is only 12½; so that, giving them the same period of time

to become infectious, they have still 5 years. For the 6 infectious cases there was on an average a 6-year period between the first noticed lesions and their becoming infectious; but the 7 non-infectious cases only showed their lesions less than 5 years ago on an average; or leaving out case 93, 3½ years ago. There is thus a distinct tendency for those having infectious contact from birth to become infectious cases.

Cases 40, 53 and 71 apparently had their first infectious contacts after the age of 10, and yet they developed into infectious cases; but these 3 senior cases may quite possibly have come in contact with other sources of infection at earlier ages.

Of the non-infectious cases, no. 93 is exceptional in that he was in infectious contact from birth, but is still only an N-2 case at the age of 20.

There are, of course, other factors which determine the degree of infection which takes place, viz. the length, frequency and closeness of contact, and the degree of infectiousness of the source at the time of contact.

In studying the family tree one is at once struck with the frequency of infectious leprosy in the fourth generation in the families in which the infectious form of the disease was common in the third generation. In the fifth generation the children were still too young at the time of examination to show signs of the disease to any great extent.

Not only are the children of direct descent affected but also nephews and nieces who have come in contact.

The importance of early marriage both in spreading the disease and in preventing the spread is brought out by this survey. The former is shown in the case of no. 120 already remarked on. The latter is shown in nos. 63 and 92 who probably escaped infection by leaving the village at an early age to go to their husbands' homes.

In studying the map, the effect of close contact within the confined space of a small house is well shown.

There is reason to believe that the absence of sanitation, poor economic conditions, the frequency of such weakening diseases as malaria, dysentery and syphilis, and the general ignorance of the villagers have much to do with high incidence of leprosy in this village.

It should be added that a few weeks of intensive propaganda have been sufficient to secure voluntary effective isolation of the 10 infectious cases still alive in the village. Whether such isolation will be effectively maintained, and the extent to which the infection will be controlled thereby in future, must be a subject for future record. But the fact that a single infectious case, such as no. 67,

FAMILY TREE OF DEBIPUR HIR

can broadcast the disease throughout the community, shows the all-importance of a careful survey followed by persistent special propaganda with a view to securing the effective isolation of all infectious cases.

Above all, considering the frequency with which those in contact from birth or in early age develop the infectious form of the disease, we would emphasise the importance of separating young children from all chance of infection.

TABLE I

General List of Inhabitants of Debipur Hir.

Serial No.	Name	Sex	Born	Married	Died	Remarks
1	Momin ...	M.	...	...	?	Was one of the 2 founders of the village.
2	Wife of Momin ...	F.	...	...	?	
3	Pachai ...	M.	...	...	?	
4	W. of 3 ...	F.	?	...	?	
5	Sadulla ...	M.	1850	1862	1916	
6	Kahiton ...	F.	1855	1862	1897	Was the 1st wife.
7	Khenti ...	F.	1876	1900	...	Was the 2nd wife. Came at 24 years of age.
8	Harmuj ...	M.	1880	?	1931	No. 79 is his widow.
9	Lehara ...	F.	1930	...	...	Her mother is a leper but is separate from her.
10	Etimon ...	F.	1883	1894	...	Left the village after marriage.
11	Rohm ...	M.	1886	...	1896	
12	Letumon ...	F.	1889	1905	1928	Was the wife of Rastom no. 22 but left him when he became an infectious leper, in 1920.
13	Khasemon	F.	1901	1914	1915	
14	Aslemon ...	F.	1906	1918	...	Left the village after marriage.
15	Salemon ...	F.	1906	1918	...	Ditto.
16	Muslemon	F.	1909	1922	...	Wife of Samed no. 116.
17	Serajaddi ...	M.	1911	1928	...	
18	Mahijan ...	F.	1916	1928	...	Came to the village after marriage.
19	Mianur ...	M.	1912	1931	...	Married to no. 127.
20	Rahatulla	M.	1853	...	1894	
21	Khairat ...	M.	1860	1874	1914	He abandoned his wife no. 120 when she became infectious.
22	Rastom* ...	M.	1887	1905	...	Leprosy case.
23	Liash ...	F.	1916	1927	...	Was taken away by her mother no. 12 in 1920.
24	Malika ...	F.	1890	1902	...	Left village after marriage.
25	Totabibi ...	F.	1864	1878	1928	Ditto.
26	Earuddi* ...	M.	1866	1886	1912	Leprosy case.
27	Hablesh ...	F.	1870	1886	...	Came to village after marriage. Her husband used to remain separate from her and her son
28	Shishu ...	M.	1899	1919	...	Was separate from father.
29	Alebjan ...	F.	1903	1919	...	Came to village after marriage.
30	Fati† ...	F.	1926	...	...	Leprosy case. Pet of Rastom (22).
31	Isak ...	M.	1928	...	...	
32	Guleshi ...	F.	1930	...	...	
33	Aleshi ...	F.	1933	...	...	
34	Jiaruddi*	M.	1868	...	1898	First leper in village.
35	Ruta ...	F.	1870	...	...	Wife of Abid no. 77.
36	Uta ...	F.	1874	...	...	Wife of Misir no. 99.
37	Abbas ...	M.	...	...	?	
38	Ugan ...	F.	...	...	1914	

Serial No.	Name	Sex	Born	Married	Died	Remarks
39	Rohiton ...	F.	1858	1872	?	Left village after marriage.
40	Madhu* ...	M.	1864	1889	...	Leprosy case.
41	Koiton ...	F.	1872	1889	...	Came to village after marriage.
42	Surmail* ...	M.	1894	1914	...	Leprosy case. Wife left him when in infectious stage.
43	Mali* ...	F.	1899	...	...	Leprosy case.
44	Ismail* ...	M.	1901	1921	...	Leprosy case. His wife (122) left him.
45	Osman ...	M.	1904	1922	...	After birth he was reared at his maternal uncle's village till his marriage. Has returned and living in a separate house
46	Kasemon ...	F.	1908	1922	...	Came to village after marriage.
47	Nurjan ...	F.	1927	...	...	Lives separately from lepers.
48	Moharjan ...	F.	1933	...	...	Ditto.
49	Rupi* ...	F.	1907	1919	...	Leprosy case. Was married but husband divorced her at her infectious stage.
50	Nekjan ...	F.	1929	...	...	Living separate from mother (49).
51	Samsuddi ...	M.	1912	...	...	Born and brought up atmaternal uncle's village and still there.
52	Nuniton ...	F.	1870	1884	1926	Left village after marriage.
53	Ali* ...	M.	1880	1898	1926	Leprosy case.
54	Khurison ...	F.	1886	1898	...	Came to the village after marriage. Had no intimate contact with any infectious leper.
55	Asijaddi ...	M.	1905	1925	...	
56	Karimon ...	F.	1910	1925	...	Came to village after marriage.
57	Taimur* ...	M.	1910	...	...	Leprosy case.
58	Hamed ...	M.	1912	...	...	Was at another village from his infancy.
59	Aharan ...	F.	1882	1894	...	Wife of Ataulla no. 71.
60	Sadaraddi	M.	1884	1908	...	
61	Khusmon ...	F.	1893	1908	...	Came to village after marriage.
62	Hatem ...	M.	1912	...	1919	
63	Sabura ...	F.	1916	1928	...	Left the village after marriage.
64	Nebura† ...	F.	1920	...	...	Leprosy case.
65	Abjura ...	F.	1924	...	1927	
66	Guljura ...	F.	1928	...	...	Lives separately from uncle no. 67.
67	Badaraddi†	M.	1891	...	...	Leprosy case.
68	Saifu ...	M.	...	...	?	
69	Tarifan ...	F.	...	...	?	1st wife of 68.
70	Sairfan ...	F.	...	...	?	2nd wife of 68.
71	Ataulla* ...	M.	1865	...	...	Leprosy case. Wife is no. 59.
72	Abul ...	M.	1904	1920	...	
73	Aklimon ...	F.	1909	,,	...	Came to village after marriage.
74	Ayesha ...	F.	1923	...	...	
75	Mahamed†	M.	1926	...	...	Leprosy case.
76	Ramesha ...	F.	1931	...	...	
77	Abid ...	M.	1870	...	1934	Wife is no. 35.
78	Abson ...	F.	1909	...	1922	
79	Hati* ...	F.	1912	1924	...	Leprosy case. Widow of no. 8. Has a daughter, age 4 years, separated from mother.
80	Jati† ...	F.	1918	...	...	Leprosy case.
81	Mati ...	F.	1918	1929	...	Left village after marriage.
82	Nazimuddi	M.	1880	1898	...	
83	Afruja ...	F.	1886	1898	...	Came to village after marriage.
84	Khalil ...	M.	1917	...	...	Born and brought up at maternal uncle's house and still there.
85	Marufa ...	F.	1920	1934	...	Do. and came to village after marriage. Husband no. 126.
86	Jalil ...	M.	1923	,,	...	As no. 84.

Serial No.	Name	Sex	Born	Married	Died	Remarks
87	Khalek ...	M.	1926	...	...	
88	Kudbani ...	F.	1929	...	...	
89	Puti ...	F.	1932	...	...	
90	Kadu ...	F.	1896	1908	...	
91	Anus ...	M.	1884	1908	...	Came to village after marriage.
92	Kamini ...	F.	1909	1924	...	Left village after marriage.
93	Elijaddi* ...	M.	1914	...	...	Leprosy case.
94	Delera ...	F.	1920	...	...	
95	Amin ...	M.	...	...	?	Was one of 2 founders of the village.
96	W. of 95 ...	F.	...	...	?	
97	Dhiron ...	M.	...	...	?	
98	Moyna ...	F.	...	...	?	
99	Misir† ...	M.	1879	...	...	Leprosy case. 1st wife is no. 36.
100	Khusmon ...	F.	1908	1924	...	2nd wife of no. 99. Came to village after marriage.
101	Nathi* ...	F.	1914	...	...	Leprosy case.
102	Ashman ...	F.	1926	...	...	
103	Ajeda ...	F.	1929	...	...	
104	Sobhan ...	M.	1932	...	...	
105	Jigru ...	M.	...	...	?	
106	Lutfan ...	F.	...	...	?	
107	Duli ...	F.	1862	...	...	
108	Damu ...	M.	...	...	1928	Came to village after marriage.
109	Risol ...	M.	1900	1918	1926	
110	Khusmon ...	F.	1906	1918	...	Came to village after marriage and left it with younger son after death of husband.
111	Nursad† ...	M.	1920	..	...	Leprosy case.
112	Irsad ...	M.	1925	...	...	Went with the mother no. 110.
113	Sabed ...	M.	1904	1923	...	Wife is no. 123.
114	Sakemon ...	F.	1926	...	...	
115	Nahura ...	F.	1929	...	...	
116	Samed ...	M.	1908	1922	...	Wife is no. 16.
117	Mursad† ...	M.	1927	...	...	Leprosy case.
118	Jabed ...	M.	1912	1930	...	
119	Aslemon ...	F.	1916	1930	...	Came to village after marriage.
120	Durpadi* ...	F.	1872	...	1904	Leprosy case.
121	Bidu ...	F.	1882	1894	...	Left the village after marriage.
122	Hadi ...	F.	1905	1921	...	Came to the village after marriage and was married to no. 44 but left him at his infectious stage.
123	Udi ...	F.	1910	1923	...	Wife of no. 113.
124	Hauji ...	M.	1892	1915	...	
125	Nishada ...	F.	1902	1915	...	Came to village after marriage.
126	Johaque ...	M.	1916	...	...	Wife is no. 85.
127	Koimon ...	F.	1920	1930	...	Wife of no. 19.
128	Mialal ...	M.	1922	...	...	
129	Sadiman ...	M.	1927	...	...	

* Infectious case. † Non-infectious case.

[This same village of Debipur Hir has been re-surveyed five years later, and we abstract from *Leprosy in India*, July, 1939, an article by Dr. Dharmendra in which the results found are described. While it is still too soon to determine the effects of the survey, the educational campaign and the resulting partial segregation which resulted, the results are, at least, promising. —Editor].

TABLE II

List of Lepers in Debipur Hir.

Serial No.	Name	Sex	Source of infectious case number	Probable age first infectious contact year	Age and year when 1st lesion noticed	Age and year became infectious	Died	Present type	Remarks
22	Rastom*	M.	34 120	7 12	27—1914	33—1920	...	C-3	Syphilis.
26	Earuddi*	M.	Uncle	?	32—1898	41—1907	1912	...	Syphilis.
30	Fati†	F.	67	birth	6—1932	...	...	N-2	
34	Jiaruddi*	M.	Uncle	?	20—1888	26—1894	1898	...	
40	Madhu*	M.	67 34	41 30	50—1914	58—1922	...	C-1, N-3	Dysentery
42	Surmail*	M.	34 120	birth 5	20—1914	25—1919	...	C-2, N-2	Syphilis.
43	Mali*	F.	67	6	11—1910	28—1927	...	C-2, N-2	Malaria.
44	Ismail*	M.	120 67	birth 4	13—1914	22—1923	...	C-2	
49	Rupi*	F.	67	birth	8—1920	12—1924	...	C-2	
53	Ali*	M.	34 67	14 25	42—1922	44—1924	1926	...	Syph. and Gonhr.
57	Taimur*	M.	67	birth	11—1921	16—1926	...	C-1, N-2	
64	Nebura†	F.	67	birth	9—1929	...	...	N-2	
67	Badaraddi*	M.	34	3	7—1898	14—1905	...	C-1, N-3	
71	Ataulla*	M.	34 67	29 40	50—1915	...	...	N-2	
75	Mahamed†	M.	67	birth	5—1931	...	...	N-1	
79	Hati*	F.	67	,,	8—1920	12—1924	...	C-2	
80	Jati†	F.	67	,,	10—1928	...	...	N-2	
93	Elijaddi†	M.	67	,,	8—1922	...	...	N-2	
99	Misir†	M.	34 120	16 21	49—1928	...	...	N-2	
101	Nathi*	F.	67	birth	10—1924	18—1932	...	C-2	
111	Nursad†	M.	67	,,	10—1930	...	...	N-1	
117	Mursad†	M.	67	,,	6—1933	...	...	N-1	
120	Durpadi*	F.	Uncle of husband	?	22—1894	27—1899	1904	...	

* Infectious case. † Non-infectious-case.

The Re-Survey.

The population, change in.—The population recorded at the original survey was seventy-seven. Three persons at that time living in the village were missed. Of these one is still in the village while the other two have gone out in search of employment. During the period between the original survey and the present survey there have been fifteen additions—11 births, 2 migrated into the village, 2 girls came to the village after marriage. On the other hand, there has been a decrease of twelve—5 deaths, 3 girls left the village after marriage, 4 persons gone out in search of employment. Thus the net population of the village was eighty-one on 1st April, 1939.

Number of lepers, change in.—In 1934 there were recorded nineteen lepers. During the period from 1934 to 1939 there has been one addition—a case of leprosy coming to the village after marriage. On the other hand, there have been a decrease of four —2 deaths, one left the village after marriage and one gone out for employment elsewhere.

Fresh cases.—During this period no fresh case has arisen amongst the population examined in 1934. A case has been detected in one of the new comers to the village. Asleman Bibi came to the village after marriage from a neighbouring village (Badulara) which is known to have a high incidence of leprosy. Asleman's aunt was a lepromatous case who died about twelve years ago. Asleman's brother is a neural case with drop-foot and claw-hands. There are also two lepromatous cases in the neighbourhood—one on each side of her father's house at Badulara. Asleman is a lepromatous case.

Incidence of leprosy.—In 1934 there were 19 cases in a population of 77 giving an incidence of 24.6%. In 1939 there are 16 cases in a population of 81 giving an incidence of 19.7%.

Type distribution. In 1934 the nineteen cases were classified as follows:—

N1 ...	3	Non-infectious.
N2 ...	6	
C1N2 ...	1	Slightly infectious.
C1N3 ...	2	
C2 ...	4	
C2N2 ...	2	Highly infectious.
C3 ...	1	

Of the nine non-infectious cases one has since died, one has left the village after marriage, one is away on some employment and one has become infectious (a L2 case). The remaining five are still non-infectious.

Of the three cases recorded as slightly infectious one has since died and two show no signs of any lepromatous change. Slit smears from ears, forehead and back in these two cases were negative for leprosy bacilli, smears were taken on a second occasion but they were still negative. All the seven cases recorded as highly infectious are still in that condition. A fresh lepromatous case (Asleman Bibi) has come to the village. The sixteen cases found in the village at the re-survey can be classified as under:

Nm1	5	
Na2	1	7 non-infectious.
Na3	1	
L1	1	
L2	3	9 infectious.
L2Na2	4	
L3	1	

The accompanying table gives a comparative statement of the condition of the lepers in 1934 and in 1939. Only one of the cases (case No. 99) recorded as non-infectious in 1934 has become infectious. All the other neural cases reported improvement some of them quite a marked one. In all the neuromacular cases the macules were flat and showed no sign of activity. In some cases it was definitely stated that the patches had faded a good deal and in one case (117) there was no definite anæsthesia in the patch. The lepromatous cases also reported improvement in their condition since they were last examined.

In the original survey report it was recorded that of the thirteen cases who were in continuous contact from birth onwards six had already become infectious cases, the remaining seven being still non-infectious. At the re-survey after a period of five years those seven were still found to be non-infectious. One of the six cases reported to be infectious in 1934 was found to be non-infectious at the re-survey. So that of the thirteen cases who had infectious contact since birth only five have so far become infectious. One of the neural cases (case No. 99) has in the meantime become infectious, this case probably had his first infectious contact at the age of 16. This brings the number of cases who had their first infectious contact after the age of 10 and yet developed into infectious cases to four.

Isolation.—In the report of the original survey it is noted that through propaganda it has been possible to secure voluntary and effective isolation of the infectious cases in the village. One of the purposes of the present survey was to find out whether such isolation has been effectively maintained and if so what has been its effect on the spread of the disease in the village.

The isolation consists in the infectious patients not sharing the

same room with the healthy persons in the family. It was also stated that their food was served in their own rooms. There is no isolation beyond this as the infectious patients were seen moving about freely in the compound of the houses and round about the village. In the centre of the village there is a shady tree. This provides a common meeting place for the villagers including the lepers and the children. The isolation thus appears to be only partial. It is too early to say to what extent will the spread of infection be controlled by this partial isolation but the findings at the re-survey appear to be rather encouraging.

In 1934 there were twenty-three persons of susceptible ages that is of ages below 15 (5 under 3, 13 from 4 to 10, and 5 from 11 to 15) who were free from any signs of leprosy at that time. In 1939 only twenty of these cases could be examined as two girls had left the village after marriage and one boy was away at his maternal uncle's village. None of the twenty persons examined showed any signs of leprosy and the villagers said that the three persons who had left the village had no sign. Eleven children were born in the village after the survey, none of them showed any sign of the disease but that does not mean anything as it is too early to expect anything in them. Two girls have come to the village after marriage, one of these is a case of leprosy. The present age of this girl is about 20 years, she came to the village after the 1934 survey, i.e., after she was 15 years old. She has come from a neighbouring village Badulara by name which has a high incidence of leprosy. In this case there is a definite history of contact, her aunt was a lepromatous case, her brother is a neural case and there were two lepromatous cases in the neighbourhood. It is very probable that this girl was infected in her father's house before she came to Debipur Hir. Thus if this case be excluded there has not been a single fresh case during these five years although persons of susceptible age were present in the village.

REFERENCE

Muir, E., and Chatterji, K. R. 1935 ... Leprosy in India, Vol. VIII, No. 4.

Note.—We are using the symbol L (lepromatous) in place of symbol C (cutaneous) according to the Cairo Leprosy Congress classification.

There are three cases which were classified as N2 in 1934 but are being classified as N1 now. According to the Cairo classification a case is not necessarily to be classified as N2 simply on account of presence of lesions on more than one part of the body. All these three cases had two macules each, the macules being on the different parts of the body. The extent of the lesions, however, does not justify these cases being classified as N2.

Table showing the condition of patients in 1934 and 1939.

Serial No. according to original survey	Name.	Sex.	Born.	Probable age at first infectious contact (years).	Condition in 1934.	Condition in 1939.	Remarks.
22	Rustom	M	1887	7	C3	L3	
30	Fati ...	F	1926	Birth	N2	NM1	
40	Madhu ...	M	1864	30	C1N3	Dead	Dead.
42	Surmail	M	1894	Birth	C2N2	L2Na2	
43	Mali ...	F	1899	6	C2N2	L2Na2	
44	Ismail ...	M	1901	Birth	C2	L2Na2	
49	Rupi ...	F	1907	Birth	C2	L2Na2	
57	Taimur	M	1910	Birth	C1-Na2	Na2	
64	Nebura	F	1920	Birth	N2	Nm1	Has since been married. The patches faded to a great extent.
67	Badaraddi	M	1891	3	C1N3	Na3	
71	Attaulla	M	1865	29	N2	Dead	Dead.
75	Mahamed	M	1936	Birth	N1	Nm1	Patches faded to some extent.
79	Hati ...	F	1912	Birth	C2	L2	
80	Jati ...	F	1912	Birth	C2	L2	Left the village after marriage. Said to be much improved.
93	Elijadi ...	M	1914	Birth	N2	Nm1	Much improved. The patches which were raised previously are now flat.
99	Misir ...	M	1879	16	N2	L2	
101	Nathi ...	F	1914	Birth	C2	L2	
111	Nursad	M	1920	Birth	N1	...	Away, said to be much improved.
117	Mursad	M	1927	Birth	N1	Nm1	The patch much faded. Anæsthesia on patch not definite.
	Asleman	F	1920	Birth	...	L1	Came from another village after marriage and after the original survey.

RESISTANCE AND THE TYPING OF LEPROSY SKIN LESIONS

BY E. MUIR.

In recent tours through Africa I found in most of the leprosy institutions visited difficulty and confusion existed in distinguishing the various types of skin lesions and in classifying cases. Yet this classification is most important for prognosis, treatment and control.

Much has been written on this subject, but it is a difficult subject to make lucid. The object of this note is to give a short and simple guide to the recognition of the various types of lesions and their significance.

Resistance.

The variation in the types of leprosy and in the nature of the lesions may depend to a certain extent on the intensity, duration and nature of the infection, and on the duration of the disease. But the chief factor is not in the seed or in the sowing, but in the soil; it is the resistance or immunity of the patient that is mainly responsible for the nature of the lesion.

Immunity to leprosy may be divided under three main groups: natural, general and acquired.

1. *Natural Immunity.*—There seems strong evidence that the great majority of people are born with strong natural resistance to leprosy. If infected, they generally either fail to develop any recognised lesion or such lesions as do develop are abortive; or, if progressive disease does occur, it is of the milder neural type.

A small minority of people, perhaps one out of ten, seem to be born with low natural resistance. Even a slight contact may be sufficient to produce the disease, and it tends to be of the more severe lepromatous type.

The evidence that this difference in resistance exists at birth depends on observations such as the following. Two members of one family have been subjected to equal chances of infection; the one acquires the severe lepromatous form of the disease, while the other who is seemingly in poorer general health either escapes altogether or develops only a mild neural lesion. The lepromin test, which is one of the best indexes of resistance to leprosy, shows differences in resistance among children irrespective of their general health.

There is as yet little evidence as to what extent natural resistance varies in its distribution in families or in races. This is a

matter in urgent need of investigation with the aid of the lepromin test.

2. *General Resistance.*—As in other diseases, the general health of the patient plays an important part in determining the type of leprosy, the progress of the disease and the nature of the lesion. This factor is the more important because of the long duration of leprosy, during which there is abundant time for complicating conditions to affect the health, and because leprosy itself lowers the health only to a comparatively slight extent.

The chief things which affect the general resistance are complicating diseases, malnutrition, lack of physical exercise, climate and the mental condition. Age also seems to be of importance, young children being less resistant than adults.

Under this heading should also be mentioned localised lowered resistance. Leprous lesions frequently originate in areas of skin, the resistance of which has been impaired by previous injuries such as scars of scabies and septic infections. Also skin lesions tend to be severer and to develop nodules in parts exposed to injury, such as the face and hands.

3. *Acquired Resistance.*—This may be systemic or local. Even in the most severe lepromatous cases the disease frequently becomes spontaneously eliminated and the infection may entirely die out, leaving only residual deformities. This is apparently the result of acquired immunity.

In tuberculoid skin lesions of the neural type it is common to find the disease spreading at the margin and simultaneously dying out in the centre. This central healing is obviously the result of local acquired immunity.

Types of Leprosy.

Leprosy is divided into lepromatous and neural types. The former is associated with high, and the latter with low, resistance. While the degree of natural immunity may be the main determinant of the type of disease, the general resistance has an important bearing on its extent and progress. Between the naturally susceptible and the naturally immune patient, there appears to be a threshold, as is shown by the clear cut differences between the nature of the skin lesions found in each. Thus the typically lepromatous lesion is very distinct from the typically tuberculoid lesion found in the neural type. This threshold is probably higher in some communities than in others.

Before discussing further the significance of leprous skin lesions it will be well to enumerate them in tabular form, placing the most typically lepromatous to the left and the most typically resistant to the right.

Lepromatous.				*Neural.*		
Nodule	Diffuse	Macule	Residual	Simple or Pale Flat Macule	Minor Tuberculoid	Major Tuberculoid

Under *lepromatous* we have three forms of lesions—nodular, diffuse and mascular; under *neural* we have major and minor tuberculoid and the simple macule; while in the centre are the residuals of either category. It is generally the two forms on either side of the "residual," namely the lepromatous macule and the simple or pale flat macule, that are difficult to distinguish from each other; but it is not uncommon to confuse even the nodular and the major tuberculoid. The following table gives the main clinical and bacteriological distinctions between the two groups.

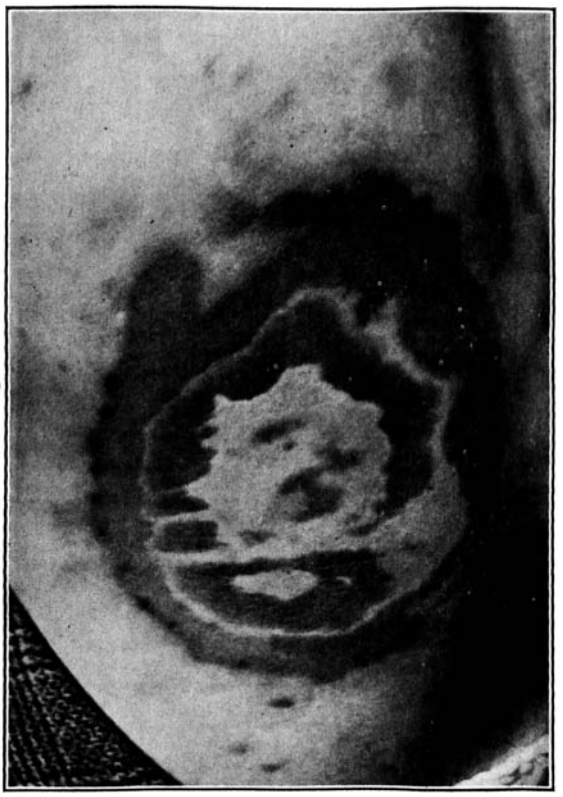
Lepromatous.	*Neural.*
1. Many bacilli especially in thickened and nodular lesions. A fair number of bacilli even in the less conspicuous macules.	1. No bacilli found as a rule except in the reactionary phase of the major tuberculoid; in these the number of bacilli is small in comparison with the degree of swelling.
2. Lesions tend to be symmetrical.	2. Lesions tend as a rule to be asymmetrical.
3. Macules are flat and smooth and tend to merge at the margin with the surrounding skin. They have no ringed margin.	3. Macules (leprides) are raised above the surrounding skin both visibly and palpably. As they increase in size they have a tendency to become flat in the centre and form a ringed margin, the width of which varies greatly in different cases.
4. Macules are numerous and closely placed; they soon coalesce leaving a mottled appearance.	4. Macules are, as a rule, few and far apart and may reach a large size.
5. Changes of sensation are slight and difficult to elicit; no thickening or tenderness of supplying nerve branches or anidrosis.	5. Changes in sensation are more marked as a rule, and there may be thickening and tenderness of supplying nerve branches and anidrosis.
6. The lepromin test is negative.	6. The lepromin test is usually positive.

In lepromatous cases in proportion to the number of bacilli there is very little thickening of the skin. Diffuse lesions covering almost the whole body may be almost unnoticeable, especially in dark skins, and yet show many bacilli.

The major tuberculoid is the most conspicuous lesion of the neural type. When activated it is red and swollen and often shows desquamation or scaliness. In severe cases there may be sloughing of the epithelium and ulceration. In such cases a fair number of bacilli may be found, but there is a tendency to spontaneous resolution, the bacilli disappearing and the lesions healing with a greater or less amount of scar formation.

The minor tuberculoid is similar but less conspicuous.

The simple macule is still less conspicuous. It may spread widely with a fine thin red margin, like a grassland fire in Africa when the grass is short, and a thin line of fire circles the dead, burnt-out area. This type may spread over the face, scarcely being noticed and leaving behind in its scar anaesthesia of the skin, which may be followed by paresis of the underlying facial muscles; this is a common cause of paresis of the eyelids and lips, and of the



TUBERCULOID LEPRIDE REPLACED BY LESIONS OF THE LEPROMATOUS TYPE.

mask-like face, though these may also be caused by the other subtypes of neural lesions.

Residual lesions are of two kinds. (1) In both lepromatous and neural leprosy there are sensory and trophic lesions of the extremities and especially of the hands and feet.

(2) The residual lesions of the skin in the lepromatous type are conspicuous in proportion to the thickness and fibrous tissue formation of the previous lesion. The crushed parchment appearance is common as severe lepromatous lesions resolve.

The major tuberculoid, if deep and inflamed, leaves a conspicuous scar like that of a burn. When less severe, this and the minor tuberculoid may resolve into a pale white macule, similar to the simple macule described above, but without the thin red margin.

We have mentioned above the threshold between lepromatous and neural leprosy resulting, as a rule, in a clear distinction between the lesions characteristic of each type. Apparently this threshold varies in height in different communities. In some communities the threshold is high, lepromatous and neural lesions contrast strongly, and the one form seldom passes into the other. In other communities, especially where climatic and other general conditions are unfavourable and depressing, the threshold may be low, the distinction between the lesions of lepromatous and neural cases may be difficult to discern in a larger proportion of cases, and the neural type may frequently give place to the more severe form. Relics of old tuberculoid lesions may be found in cases which have become distinctly lepromatous; or reference to previous records may show that this change has taken place. In the photo attached a lepromatous lesion has developed in the flattened centre of a residual tuberculoid lepride, and numerous lepromatous macules are seen all over the previously unaffected skin.

REVIEWS

Lecture Notes on Leprosy by John Lowe, M.B., Ch.B. (British Empire Leprosy Relief Association (Indian Council), Delhi, India.

This booklet supplies the need for a small, concise and cheap publication on leprosy. It is based on lecture notes which the author has used for several years as the basis of a series of six lecture-demonstrations delivered as part of the D.T.M. and D.P.H. course in Calcutta. It is intended for non-specialists who wish to grasp the most important practical points regarding leprosy, also for those who have to teach others about leprosy in medical colleges and schools.

The book is clearly and concisely written, and contains 46 excellent illustrations. It concentrates within its 56 pages a wealth of information in an easily assimilable form, at the same time referring readers to larger works for further details when desired. While written primarily for doctors and students in India, it should prove useful also in other countries where leprosy is a problem.

Short Notes on Leprosy by D. N. Mukerjee, M.B., Leprosy Specialist to the Government of Central Provinces and Berar, India.

This booklet condenses into the small space of 25 pages much of the most recent teaching with regard to leprosy. It is not sufficient for anyone who wishes to specialise, but sufficient for anyone who wishes to obtain a working knowledge of leprosy. It does, however, fulfil its object of "acquainting anti-leprosy workers with the up-to-date advances or modifications in the classification, treatment and various other aspects of the disease," and for those who require it, introduces them to other literature. It should serve a useful purpose, especially in India.

Through the Leper Squint by Anthony Weymouth.

This is a study of leprosy from pre-Christian times to the present day. On the whole the facts recorded are correct, though there is a certain number of inaccuracies. The book is attractively written and is intended for the layman as much as, or perhaps more than, the medical reader. It contains an account of the disease in Biblical times, with special reference to the descriptions in Leviticus, Kings, and Job; classical antiquity, in which it is mentioned by Aretaeus of Cappadocia, Celsus, Pliny, and Lucretius; the Middle Ages, and modern times. A special chapter is devoted to the Knights of St. Lazurus, whose order was founded by St. Basil in the fourth century for the care of the sick, and lepers in particular, and was dissolved in 1830. Other chapters deal with leprosy in legend and literature, the story of Father Damien, leper colonies of to-day, and the modern treatment of leprosy. The text is liberally interspersed with portraits of Hansen, Danielssen, Sir Leonard Rogers, and Father Damien, photographs of various types of the disease, and other illustrations. Tables dealing with the chronology of leprosy and its incidence throughout the world, and a bibliography of English and French works, are appended. The book is published by Selwyn and Blount, price 12s. 6d.

La Lèpre, Diagnostic—Traitement—Prophylaxie, by A. Dubois, Bruxelles, Imprimerie Industrielle et Financière, 47 Rue du Houblon.

This little book is based largely upon the writer's experience of leprosy in the Belgian Congo and has special reference, in the section on prophylaxis, to conditions in that country. There are some excellent photomicrographs in the clinical and clinical anatomy section. There is a preface by Prof. Marchoux. The book is condensed and practical and follows, to a large extent, lines

similar to the book recently published by the Indian Council of B.E.L.R.A. It should be of great value to leprosy workers, especially those in Belgian and French Colonies.

International Journal of Leprosy, Vol. 7, No. 1. Jan.-Mar. 1939.

The first half of a Review of *The Bacteriology of Leprosy* is given by E. B. McKinley. After reviewing all the claims that have been made to cultivate the bacillus artificially up to 1918, he says:

"It seems necessary to admit that none of these organisms was established beyond question as the true leprosy germ. We therefore pass to what is here designated as the period of our newer knowledge of the bacteriology of leprosy, with apologies for determining in an arbitrary fashion where one period should leave off and another begin, or indeed that two periods should be recognised."

[The second half of this review appears in the April-June, 1939, number of the Int. Jl. of Lep.].

K. O. Courtney writes on *Leprosy in Panama.*

"At the time this study was begun there were 109 lepers in the colony, 32 of whom were females and 77 were males. Of the total no less than 78, or 71 per cent, were of Spanish-Indian origin (mestizos). The remaining 31 were of foreign origin. . . . . Leprosy in Panama appears to be a familial disease, which the writer interprets not as an example of biologic inheritance of the bacterial invasion, but as evidence of inherited predisposition to leprosy, coupled with prolonged intimate contact with the disease."

S. S. Juschko writes on *Hydrophylia of the Tissues in Leprosy.* This is tested by McClure and Aldrich's "Wheal test" (QRZ) which is carried out as follows:

"On the inner surface of the forearm 0.2 cc. of physiologic salt solution is injected intracutaneously, and the time required for the resulting wheal to disappear is registered. The reaction is considered ended when the wheal can no longer be seen or palpated."

His conclusions are:

"In leprosy, disturbance of the water exchange, as demonstrated by means of the McClure-Aldrich wheal test, is very frequent—95% of cases. The acceleration of the QRZ parallels the extent of the specific leprous processes. The QRZ is accelerated in lepra reaction. Specific leprous changes of the hands and feet (edema leprosum of Jadassohn) are accompanied by acceleration of the QRZ. The skin phenomena of leprosy (namely the chronic inflammation processes), influence the acceleration of the QRZ. In leprosy the QRZ is of as much importance in the determination of the general condition of the patient as, for example, the red-cell sedimentation test."

H. Ross writes on *Blood Glutathione in Leprosy.*

M. Suwo and S. Kin write on *Culture of Leprous Tissues in Vitro Using as a Medium Tissue-Culture of Skin Nodules from Human Leprosy.* The method used and the results obtained are abstracted as follows:

"Small pieces of skin nodules of human leprosy grow well in tissue culture. The growing tissue is composed chiefly of fibroblast-like elements and epithelioid cells. Both of these elements always contain leprosy bacilli in their protoplasm in much greater numbers in the epithelioid cells

than in the others. Epithelioid cells take up vital stains actively, while on the other hand the fibroblast-like cells take few or none of the pigment granules. Under the influence of the multiplying leprosy bacilli the fibroblast-like cells are converted into pigment-storing epithelioid cells, both in the explant and the growing zone. Lepra cells develop in vitro from the epithelioid cells. Leprosy bacilli multiply, not only in the body of the cell but also in the nutrient medium, after the culture is gone, without forming colonies visible with the naked eye."

R. O. Prudhomme reports on *Weakening of the Virulence of Stefansky's Bacillus with Ultra-violet Rays.*

"Irradiation of from 2 to 5 minutes causes attenuation of the virulence and a change of character in the evolution of the disease: absence of local lesions and limitation of the infection to the superficial ganglions, which are not enlarged. Bacilli irradiated for 10 minutes show themselves to be avirulent and are destroyed by the organism of the rat."

J. Lowe reports on *Tuberculoid Changes in Lymph Nodes in Leprosy.* Definite changes of this nature were found in six out of eleven cases examined.

In an Editorial, Prof. Marchoux puts forward three suggestions:

"That infectious material from the greatest possible number of lepers be inoculated into rats to determine if different germs can cause leprosy. That the Hansen bacillus be inoculated in all the animals existing in the vicinity of the investigator, for the purpose of discovering a receptive one. That leprologists find out what becomes of the bacillus found in the superficial lymphatic ganglia of apparently uninfected persons."

In answer to a question as to why it is difficult to retain the acid-fastness of lepra bacilli in sections, Dr. Wade points out that after treating the section with xylol or other oily substance changes take place which facilitate the removal of fuchsin by alcohol. Lowe avoids the use of alcohol by drying the sections between xylol and water. Wade used certain essential oils and thus avoids both alcohol and desiccation. Dr. Wade hopes to publish his technique in the near future.

International Journal of Leprosy, Vol. 7, No. 2, Apr.-June, 1939.

This number opens with an article by J. Lowe and S. N. Chatterji on *Seasonal Variation in Leprosy in Calcutta.* Analysis of records of new cases at the Calcutta leprosy clinic over two years showed a marked increase in the attendance of neuromacular cases from March to October, as compared with the rest of the year. Also during March, April and May the percentage of these cases showing positive bacteriological findings rose from 5 to 26 or 27. The cause of this is discussed and the authors connect it with the high temperature and low humidity prevailing during these months. Oberdoerffer's theory that this seasonal variation is due to eating of colocasia is rejected. [It is a well-known fact among the natives of Bengal that leprosy and tuberculosis become worse in the three hot months; also at the end of this season people are affected with

boils. Possibly the condition in all three diseases is dependent on adrenal exhaustion owing to preoccupation of these glands with the more urgent task of regulating the body temperature.]

Von A. A. Stein writes on the *Morphology of Lepra Reaction.* In a previous article the author described the clinical manifestations of lepra reaction, divided into three types: (1) with the new eruption of lesions, (2) with only reactivation of old lesions, and (3) mixed, with both. From his new observations and those of other observers it is concluded that the supposedly new reaction lesions studied were not new at all, but were old ones that were not evident until they were activated by the reaction. This and other features are discussed in some detail. It is pointed out that one cannot really speak of two types of reaction because the apparently new lesions are actually old ones made evident, the pathological process being the same in both. The difference between the two clinical forms is only quantitative, not qualitative.

A. Rotberg writes on *The Reading of the Lepromin Test.* A former article by this author dealing with a similar subject was reviewed on page 130 of the April number of *Leprosy Review.*

The *Irritant Action of some Drugs Derived from Hydnocarpus Oil* by H. Paget, J. W. Trevan and A. M. P. Atwood.

"It has been shown beyond question that the principal cause of excessive irritation by hydnocarpus preparations is the presence of oxidation products of the unsaturated and unstable acids, chaulmoogric, hydnocarpic and dehydrochaulmoogric. Their presence is due either to the use of deteriorated seed as a source of the oil, or to exposure to light and air of the oil or of the esters or salts of the acids. Once formed, these products cannot be completely removed by washing or by distillation, and they give rise to volatile and irritant products in the course of manufacture of the esters." "Iodised ethyl esters of the crystalline acids are unstable on exposure to air, but oxidation is inhibited by addition of 0.1 per cent of catechol or pyrogallol."

M. Kervingant and L. Baré write on *La Lepre en Nouvelle-Calédonie en 1936.* Leprosy is stationary among the indigenous population and is diminishing among the Europeans and immigrants as the result of the work of the medical specialists. Contagious European patients are hospitalized or isolated in their domiciles, while contagious natives are segregated in agricultural colonies: non-contagious patients are kept under observation and treatment. Suspects are carefully examined. This method of dealing with leprosy is popular among the people, and they come readily to the doctors.

Leprosy in Queensland, by R. Cilento. The quinquenial number of lepers detected rose from twelve in 1905 to 65 in 1935. The average ages at time of notification among whites is 41 and among coloured 33.9.

"The numbers are too small to establish an incidence in the various age groups. The youngest leper was seven and the oldest ninety-two. It is to be remembered, of course, that cases are usually found after the disease has existed for some years, and, in many instances, for many years. The outstanding fact of importance is that an increasing number of children show infection, indicating the established endemicity of the disease."

A. P. Davis writes on *Leprosy in Western Australia.* Leprosy was introduced by the indentured labour of the pearling industry as leprosy is fairly common in the Asiatic countries from which the crews were drawn.

"The problem of leprosy is therefore for all practical purposes confined to the Kimberleys. Careful supervision of, and, where practicable, absolute prohibition of, the southern migration of northern natives into 'clean' parts will tend to delimit the disease and will enhance the prospect of ultimately stamping it out. . . . The situation, therefore, while serious enough to contemplate, is not in my opinion as desperate as has been suggested, and I believe it is now well under control. There is no doubt that cases of leprosy will continue to crop up for some time to come, but they will be found, I think, in gradually decreasing numbers. Nevertheless, there should not be any relaxation of vigilance in the inspection of those parts from which in the past the great majority of cases have been drawn."

The conclusion of the Review on *Bacteriology of Leprosy* by E. B. McKinley, is given. Among the concluding remarks is stated:

"Considering all of the facts it must be said that there does not exist today *any absolute proof* that any investigator has actually succeeded in the artificial cultivation of the leprosy bacillus. We are aware that there are investigators who will not agree with this statement, who probably feel that organisms cultivated by them from the tissues of lepers represent the true *M.leprae*. We can appreciate this point of view. Yet the author with his colleagues, who have also secured cultures which they feel are probably of that organism, are of the opinion that that statement is the only fair one that can be made at this time. Nevertheless, we feel definitely that our organism has more in its favour than any other one which has been submitted as *M.leprae*, though it is grown only with great difficulty and is very sparse in growth. Obviously further advances as regards cultural methods are required. Meanwhile, no doubt, other investigators will be critical of the rather feeble results which we are able to obtain, but we hope that at least serious efforts will be made to confirm our findings up to this point.

As for animal experimentation, we feel again that the only fair statement which can be made at the present time is that no investigator has yet succeeded in producing in any experimental animal the counterpart of human leprosy as it is known in man. Naturally, in this statement we include our own attempts in this direction, though we feel that we have perhaps gone somewhat farther than others in establishing our organism as *M.leprae* through animal experimentation. There is hope that new approaches to this problem may eventually lead to success in producing progressive lesions of the disease in lower animals. If this can be accomplished in a satisfactory manner, then there is also hope of eventually determining beyond doubt whether or not a given culture, suspected of being the leprosy bacillus, is really that organism."

A comprehensive and valuable bibliography is given with 296 references.

Leprosy in India, Vol. II, No. 2, Apr. 1939.

There is an article by J. Lowe and S. N. Chatterji on *Surgical Removal of the Sheath of the Ulnar Nerve in Severe Leprous Neuritis.* They consider that cases suitable for this " operation are those in which thickening and inflammation of the ulnar nerve is marked, but is of relatively short duration, and in which the deformity of the hand has either not yet developed, or else is slight. The operation must be done at the right time before the nerves have been permanently damaged. We think the operation is possibly most beneficial in the type of case in which caseation is likely to take place in the nerve, but in which nerve abscess has not yet occurred. This is the type of case which shows marked tuberculoid lesions in the skin or nerves. In cases in which the nerves are already caseous, the benefit from the operation in the prevention of deformities is slight, because the nerve damage has already been done. In suitable cases the operation if properly performed will, in our opinion, do no harm and will often do good. The operation does not cure the inflammation of the nerve and it was never expected that it would, but it is believed that the operation often prevents the inflammation of the nerve from producing destruction of the nerve fibres."

The following is a description of the operation.

" An incision from four to six inches long is made through the skin over the thickened inflamed portion of the nerve. The deep fascia over the nerve is then incised and also the intermusular septum covering the nerve. Sometimes the nerve has to be separated by blunt dissection from an adherent triceps muscle. Small vessels may be encountered. The thick inflamed nerve is thus freely exposed. With a sharp scalpel a small longitudinal incision is made in the middle of the anterior surface of the nerve sheath, and this incision is very carefully deepened until the nerve bundles are clearly seen. This small incision is then enlarged up and down the thickened part of the nerve by the use of a blunt instrument. The next step is to free the nerve bundles from the sheath. The cut edge of the sheath is held in forceps while a blunt instrument is used for separating it from the nerve bundles, working first on one side and then on the other and finally round the back of the nerve until in the centre of the affected part of the nerve, the sheath is free all round. When this has been done, a curved blunt instrument is inserted behind the nerve and in front of the separated sheath, and with the aid of the finger covered with gauze, the nerve sheath may be gently stripped upwards and downwards until the sheath is free over the whole length of the thick inflamed portion of nerve. The sheath is then cut through at the top and the bottom. It is much easier to remove the sheath in one piece than to remove it piecemeal. Not infrequently when the nerve sheath is first incised, the tension inside is shown by bulging of the contents; foci of caseous material may be found lying usually between the nerve bundles. Sometimes small abscesses are exposed.

Sometimes because of inflammation, caseation and adhesions, it is impossible to strip off the nerve sheath in the manner described, and in such cases three or four longitudinal incisions are made through the nerve sheath on different aspects of the nerve.

All cut vessels are ligatured and the deep fascia may be loosely sewn over the nerve, but if so, care must be used to prevent pressure on the nerve. The skin should be carefully sutured to produce a neat scar and quick healing. The whole operation should be done with great care and precision to avoid all damage to nerve fibres.

The operation may be done under local anaesthesia, regional anaesthesia or general anaesthesia. The actual operative procedure should take only a few minutes. On the evening following the operation it is advisable to give analgesics by the mouth or by injection."

An article by U. P. Basu and A. Mazumder on *The Keeping Properties of Hydnocarpus Oil* shows that early addition of creosote (4%) or hydroquinone (0.02%) inhibits auto-oxidation and markedly improves its keeping qualities. This should be added before boiling.

Leprosy in India, Vol. II, No. 3, July, 1939.

An article by R. G. Cochrane, N. P. Raj and E. Roy, describes *Treatment of Acute Lepra Reaction.* After recounting the signs and symptoms, the treatment by laxatives, alkalis and anodynes is described. Fouadin, a pentavalent antimony product in 2 cc. doses is preferred to potassium antimony tartrate. It can be given intramuscularly whereas the latter must be given intravenously. The importance of distinguishing lepra reaction from other febrile complicating diseases is emphasised.

J. Low writes on the *Indian Leprosy Commission of 1890.* Some of the inconsistences surrounding this commission are summarised in the last paragraph:

"To one unfamiliar with the ways of governments it is difficult to understand how within a few years the Government of India could solemnly declare, first that the only measure to control leprosy in India is the isolation of life of all persons with the disease, later that as a public health measure they propose to legislate for the isolation of vagrant lepers, and later still, that they believe that leprosy is rarely spread by contagion but nevertheless they propose to legislate (and actually do legislate) for the isolation of vagrant lepers and for the limitation of contact between healthy persons, and lepers, and articles handled by lepers."

Dr. Dharmendra writes on a *Re-Survey of the Village of Debipur Hir.* This note has been added to a reprint of the note on the original survey to be found on p. 208 of this number.

Blood Cholesterol in Cases of Leprosy, by Dharmendra and N. K. De, is summarised as follows:

"Blood cholesterol was estimated in 23 non-lepers and 250 patients suffering from leprosy. The mean average value in non-lepers was 163.44 mg. per 100 c.c. of plasma with a standard deviation of 48.34. In the untreated cases of lepromatous leprosy the mean average value was 129.09 mg. per 100 c.c. of plasma with a standard deviation of 34.24. In the untreated cases of neural leprosy the mean average value was 143.08 mg. with a standard deviation of 38.01. The average values in the treated lepromatous and neural cases did not differ significantly from the average values in the untreated cases in the same groups. There was no significant difference in the blood cholesterol values of Nm1 cases as compared with those of Nm2 cases. The administration of hydnocarpus oil was attended with an increase in the blood cholesterol in those cases which showed some improvement after the treatment. It is difficult to say whether or not the increase in blood cholesterol is the direct result of hydnocarpus oil administration."

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