A STUDY OF LEPROSY IN FIJI

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INTRODUCTION

The present study is based on the records of the Central Leper Hospital, Makogai, from its inception by the Government of Fiji in 1911 to the end of 1933. There have been more than sixteen hundred admissions to Makogai during that time, but a number of these cases were admitted only for short periods prior to repatriation to India. Of the cases admitted only those whose notes were sufficiently full to be of statistical value have been included in this survey. The total of these is 1,378, but 13 of them were discharged at various times as nonlepers, which leaves 1,365 cases for analysis.

Unfortunately, it has proved impossible to do more than indicate the possibility of racial differences in relation to leprotic infection, owing to the multiplicity of factors involved. For this reason too much stress must not be laid on the comparative statistics, for example regarding the Fijians and Indians, for as indicated in the text repatriations have rendered the Indian data unsatisfactory from this point of view.

HISTORICAL

As is the case with other South Sea Islands it is impossible to be definite with regard to the duration of leprosy in Fiji. To the Chinese is commonly ascribed the introduction of the disease into the South Seas, but there are indications that must have been present in Fiji for a much longer period than is generally believed.

The earliest known mention of the disease here was made by Lyth of the Methodist Mission, who in 1837 (7) briefly recorded that cases were being treated by himself and his ministerial colleagues. However, the Fijian language has at least two definite names for leprosy: *sakuka* in the Western dialects, and *vukavuka* in the now more generally accepted Bauan dialect. Neither of these terms has any other meaning and, moreover, there are a number of subsidiary

names for the various symptoms. Fijian mythology furnishes additional evidence of prolonged acquaintance with the disease, one of their gods being a reputed sufferer. Furthermore, as reported by Corney (3), there are a number of "leprosy stones" (*vatu ni sakuka*) scattered throughout Viti Levu. These were the property of certain families which were infected to a greater or less extent, and by their aid the heads of the families could hand on the infection to anyone obnoxious to them.

The same writer records a complaint of the Fijians that leprosy was increasing as the result of the action of the Government prohibiting the destruction of lepers. It would appear, however, that the only lepers put to death were very advanced cases with ulcerating nodules, or those helpless from crippling deformities. This suggests that they were clubbed more to put them out of their misery than because they were sufferers from leprosy; those sick from any disease were dealt with more or less similarly.

A story is told regarding a small island to the north of Vanua Levu in Macuata Bay, close to the island of Kia. It is little more than a stony ridge which the inhabitants of Kia used as a dumping ground for rubbish of all kinds, giving it the name of *de ni Kia,* "the refuse of Kia." On this salubrious spot were isolated a number of advanced cases of leprosy, in order that the local chief should never be without a *bokola* (body prepared for the oven) with which to welcome visitors or celebrate any important event!

There appear to have been, from very early times, Fijians who claimed to be able to cure leprosy, but as more recently cases that had been treated by such people have been admitted to Makogai the cures were evidently more apparent than real. However, a case is reported by Moore (9) of which he claims to have personal knowledge, that of a young Fijian suffering from leprosy who submitted to being "smoked" over burning *sinu qaqa* (*Excoecaria agallocha* Linn.) and so recovered his health. The sinu tree referred to is well known in Fiji and is intensely poisonous, although some Fijians appear to be immune to its action. Cases have occurred in which the eating of prawns from a fresh water creek into which sinu leaves had fallen have provoked in both Europeans and natives the most extreme diarrhoea and collapse, which was later followed by an intensely irritating eruption.

The first direct administrative step taken by the Government of Fiji concerning lepers was the passage of "The Lepers Ordinance, 1899." This ordinance forbade certain callings to the lepers, and gave the Governor power to establish leper asylums and to consign thereto any leper in the gaol or lunatic asylum or any leper guilty of an offense against the ordinance.

As a result of this ordinance a leprosy station was established on the island of Beqa, to the south of Suva. It is remarkable that the chief medical officer was able to report in 1910 that 90 per cent of the inmates of Beqa had come there voluntarily. However, it was impossible to acquire the whole island, and it gradually became evident that this was essential if isolation was to be absolute. In 1909 the island of Makogai was bought for the purpose by the Government, and after certain delays due to financial stringency forty patients were transferred from Beqa in 1911.

THE PRESENT LEPROSARIUM

Makogai, which was a coconut plantation before its purchase by the Government, is a reef-encircled island about two and a half miles long and one and a half miles broad. It lies in the Koro Sea between the two main islands of the group—Viti Levu and Vanua Levu—and is about seventeen miles from Levuka, the former capital of the Colony. Its flat land is limited to an irregular area around the sea coast, bounded inland by rocky ridges leading up to numerous peaks the highest of which are more than 850 feet above sea level. The climate is agreeable, being considerably modified by the sea breezes.

The hospital proper is situated at Dalice, on the northwest coast. It is divided into two compounds, the two sexes being kept strictly separate. The women's compound has accommodations for all the female patients, but the men's compound is used only for the helpless, the acutely ill, and those requiring special attention. In the hospital area, also, are the administrative block, pharmacy, operating theatre, dressing rooms, laboratory, etc. Most of the buildings are of wood, raised on concrete piles and with corrugated iron roofs, ceiled on the inside. Dressing-rooms and baths are floored with concrete. Two Delco engines provide power for light in the hospital buildings, as well as for the cinema and two refrigerators.

Men able to look after themselves are housed in five outlying villages. So far as possible the men are assigned to these according

to race, each village having its own headman. There is a dressing-room in each village where dressings are made daily, but patients come to hospital for injections or other special treatment.

The gradual increase in numbers of inmates at Makogai since its inauguration will be seen from Table 1, which gives the total number of patients at the end of each year.

TABLE 1.—*Numbers of inmates at Makogai, by years.*

1911	40	1923	289
1912	154	1924	272
1913	177	1925	297
1914	279	1926	302
1915	249	1927	438
1916	287	1928	409
1917	314	1929	442
1918	327	1930	433
1919	352	1931	494
1920	243	1932	476
1921	216	1933	427
1922	266		

TYPE OF THE DISEASE

The classification utilized for the purpose of this study is, in the main, that recommended by the Manila Conference (8). The "mixed" group, however, has been omitted on the ground that, as shown below, every cutaneous case is regarded as suffering from some degree of nerve involvement. Our experience at Makogai also renders the classification into "cutaneous" and "neural" practically identical with that of "open" and "closed," a recent survey having failed to reveal a single case in which the nasal mucosa was positive that did not also show positive skin smears. It may therefore be taken that the cases recorded as neural have been bacteriologically negative, or closed, cases.

Of the 1,365 cases available for analysis, 746 were neural in type and 619 were cutaneous, as is seen from Table 2. This gives percentages of 54.7 for the neural type and 46.6 for the cutaneous type over the whole period. Hopkins and Denney (6), reporting on 718 cases in the U. S. Federal leprosarium, at Carville, Louisiana, record only 11 per cent as neural, 39.1 per cent "skin" and 49.9 per cent "mixed," which under the scheme of classification here used would give 89 per cent cutaneous.

In a previous study of 100 cases of leprosy in children (1) the writer found 54.3 per cent to be neural, but of 20 cases under the age of ten years 16 (80 per cent) were so classed. This was taken as an indication of an earlier stage of infection, and it was argued that every case of leprosy passes through a neural stage, none of the cases failing to show some degree of interference with sensation. As a test of the truth of that argument every patient at Makogai was recently examined for evidence of nerve involvement, and the only instance in which this could not be demonstrated was that of a European admitted in 1926 as a C2 case and now quiescent. However, it was found from his records that for some time after admission he had required periodical treatment for ulnar neuritis. It may therefore be assumed that so far as leprosy as met with in Fiji is concerned there is no such condition as pure cutaneous leprosy without some degree of nerve involvement.

TABLE 2.—*Relation of race and type of disease.*[a]

Race	Number of cases	Neural					Cutaneous				
		N1	N2	N3	Total	Per cent	C1	C2	C3	Total	Per cent
Fijian	444	44	163	81	288	65.0	11	83	62	156	35.0
Solomon	130	15	39	16	70	53.8	4	31	25	60	46.2
Rotuman	26	—	7	4	11	(42.3)	—	6	9	15	(57.7)
Samoan	34	5	5	2	12	(35.3)	4	11	7	22	(64.7)
Tongan	13	1	3	3	7	(53.8)	2	2	2	6	(46.2)
Cook	80	15	28	9	52	65.0	8	14	6	28	35.0
Maori	4	1	—	1	2	(50.0)	—	1	1	2	(50.0)
Niue	3	—	—	—	—	—	—	3	—	3	(100.0)
Indian	595	74	164	53	291	48.9	34	180	90	304	51.1
Chinese	18	—	3	2	5	(27.8)	1	6	6	13	(72.2)
European	18	3	3	2	8	(44.4)	—	6	4	10	(55.6)
Total	1,365	158	415	173	746	54.7	64	343	212	619	45.3

[a] Percentages in parentheses pertain to groups of less than 50.

Race and type of disease.—Data on the type distribution of the different races are given in Table 2. Analysis of these figures indicates that the gross percentages given above represent real averages and do not serve to cloak any racial extreme. Thus the percentages of neural cases are: for 444 Fijians, 65.0; for 130 Solomon Islanders, 53.8; for 80 Cook Islanders, 65.0; and for 595 Indians, 48.9. Figures for 1933 show that of 510 inmates of Makogai during the year, 44.1 per cent were neural, and that of 34 fresh admissions 67.6 per cent were of that type (4).

SEX

With regard to sex, the well-known but so far unexplained large male majority is further exemplified by our figures. Thus 1,049 (76 per cent) were males and 329 (24 per cent) were females, a ratio of 3.2 : 1. It must be borne in mind, however, that the Indian population of the Colony of Fiji still shows a preponderance of about 14,000 males over females, and that during the earlier part of the period concerned in these statistics the discrepancy was much greater, so that the Indian male percentage of 81 per cent is certainly too high. However, the Fijians, whose male and female populations are more nearly comparable, and have been more or less stable over the period concerned, are not far behind with 73 per cent males.

TABLE 3.—*Relation of sex and type of disease.*

Sex	Number of cases	Neural					Cutaneous				
		N1	N2	N3	Total	Per cent	C1	C2	C3	Total	Per cent
Male	1,039	101	312	135	548	52.7	49	277	165	491	47.3
Female	326	57	103	38	198	60.7	15	66	47	128	39.3
Total	1,365	158	415	173	746	53.7	64	343	212	619	45.3

In the case of children, however, the difference between the sexes is much less striking, the percentages being 57.2 for males and 42.8 for females, while of twenty cases under ten years of age the number in each sex was the same (1). This is very different from the figures given by Rogers (12) as taken from the data of five Indian censuses, according to which there was an average of 92 females as against 45 males in the 0-5 year age period, and 200 females as against only 103 males in the 5-10 year period. Rogers, however, does not lay much stress on these figures, and indeed remarks that:

> ...it is difficult to suppose that there can be much actual difference in the proportion of boys and girls infected and developing the disease in the first five years of life, as sex distinctions develop long after that age.

This statement is supported by the Makogai figures.

The suggestion of Cooke (2) attributing the sex and age distribution of the disease to the factor of "domesticity" is an attractive one to explain this similarity of the sexes in the lower age periods, but to be adequate it would need to be correlated with a higher adult female incidence, which is not the case. It may be, however, that there is a double explanation in this country. The adult male Fijians,

most of whom leave their homes for longer or shorter periods of indenture in other districts, are still in Cooke's "pioneering" stage. This fact provides more numerous opportunities of infection among men, who live together in "lines" at their places of indenture, with less likelihood of infection being carried to their women folk.

Sex and type of the disease.—There is little evidence in our data that sex plays any role of importance in determining the type or course of leprosy. As is shown in Table 3, the neural type was found in 52.7 per cent of the males (548 cases) and in 60.7 per cent of the females (198) cases, though in the early nerve class there is a 2:1 male-female ratio instead of the general 3:1 ratio. On the whole, then, the females show a slight advantage as regards the stage of the disease. Data on the relation of sex and response to treatment are given later in this report.

TABLE 4.—*Causes of death.*

DIAGNOSIS	CASES	
Leprosy and exhaustion from leprosy	102	
Leprotic exhaustion and senility	28	130
Pulmonary tuberculosis	71	
Generalized tuberculosis	38	
Other forms of tuberculosis	8	117
Septic absorption		44
Nephritis		35
Pulmonary conditions other than tuberculosis		24
Circulatory conditions		28
Alimentary conditions		14
Malignant disease		8
Tetanus		6
Unclassified		11
Total		417

MORTALITY AND CAUSES OF DEATH

A total of 417 deaths have occurred at Makogai, as shown in Table 8, but any attempt to work out mortality rates for leprosy from these figures would be futile. Of the 1,365 cases dealt with in this report, 298 Indians were repatriated in fairly good physical condition. Of the 1,067 remaining cases analyzed with respect to results of treatment, 260 have been conditionally discharged. Thus the patients least likely to contribute to the mortality rate are continually leaving the hospital, which makes the actual death rate disproportionately high. The highest rate recorded was 116.6 per thousand in

1930, but this was followed in 1931 by 84.2, in 1932 by 36.1 and in 1933 by 66.6. Such extreme variations without any very obvious reason indicate the small value to be placed on any such calculations, although the 1930 rate does to a certain extent reflect the results of severe epidemics of dysentery and influenza, whereas the 1932 rate results from a year remarkably free from epidemic disease. The death rate is therefore decided by side issues and not by leprosy.

The main causes of death recorded are shown in Table 4. The diagnoses recorded should not, however, be allowed to obscure the fact that it is the leprous process that, by lowering the resistance, underlies many of these other conditions. It is no more correct, for example, to regard sepsis as the cause of death in leprosy than in a transverse section of the cord.

It may be of interest to compare this summary with Harper's analysis of the causes of the first 157 deaths at Makogai, up to 1921 (5). The first four causes were: tuberculosis 42 cases, septic infection 31 cases, nephritis 18 cases, leprosy 14 cases. If, however, exhaustion and senility be added to his leprosy figure in order to render the lists comparable, leprosy comes third with 22 cases. A comparison of Harper's data thus corrected, with our own total to the end of 1933, is given in Table 5.

TABLE 5.—*Comparison of principal causes of deaths, Harper's data (to 1921) and present total (to 1933), in percentages.*

To 1921		To 1933	
Tuberculosis	26.7	Leprosy	31.1
Septic infection	19.7	Tuberculosis	28.0
Leprosy	14.0	Septic absorption	10.5
Nephritis	11.4	Nephritis	8.3

Mortality and type of case.—An analysis of the deaths with reference to the type of the disease (Table 6) shows, as one would anticipate, a more or less regular gradation in mortality from the less to the more advanced cases. In the same table is an analysis of the duration of the disease at the time of death with respect to type. This presents certain features that are somewhat surprising. According to our data the average duration in those who died has been only 5.9 years, with wide variations in the different stages of the disease. To find the longest duration in the most advanced stage of each type obviously requires some explanation. It should first of all be pointed out that the figures are admittedly too low. In the

great majority of cases, even among the least advanced, no reliable history as to duration could be obtained. Therefore, rather than base this analysis on mere estimates, which could only be approximate and might be far from correct, the date of admission to Makogai has been used instead of the date of the first appearance of symptoms.

Referring again to the causes of death, it may probably be taken for granted that the deaths more directly attributable to leprosy or its effects are those certified as due to leprosy, exhaustion and senility, septic absorption and nephritis. A tabulation of these deaths according to type (Table 7) offers the gradation one would expect.

The analysis shows, in the first place, the great disproportion between the two types in this respect, 63.3 per cent of deaths in cutaneous cases being attributed to leprosy and only 30.8 per cent of

TABLE 6.—*Relation of type of case to (a) number of deaths and (b) average duration at the time of death.*

Type of case	Number of cases	Deaths		Average duration years.[a]
		Number	Per cent	
Neural, slight (N1)	158	13	8.2	2.4
Neural, moderate (N2)	415	95	22.8	5.4
Neural, advanced (N3)	173	61	35.2	7.9
Cutaneous, slight (C1)	64	16	25.0	3.4
Cutaneous, moderate (C2)	343	115	33.5	4.8
Cutaneous, advanced (C3)	212	117	55.1	7.0
Total	1,365	417	30.5	5.9

[a] For explanation of these figures see text.

neural deaths. It is of interest that there were no deaths directly attributable to leprosy in the early neural stage, so that the duration of 2.4 years given for this stage in the gross analysis (Table 6) is very misleading. It further indicates that only about 50 per cent of the total deaths in this institution can fairly be ascribed to leprosy, and that most of these deaths occurred in the more advanced stages. As for duration, considering only those cases indicated in Table 7, an average duration of 7.2 years has been obtained, as against the general average of 5.9 years. Of these 209 cases, 70 had lived more than ten years after admission to Makogai.

Using the same assumption (that deaths certified as leprosy, exhaustion, senility, sepsis and nephritis are all actually due to leprosy)

the comparative percentages for the periods to 1921 and to 1933 are 40.1 and 50.1, respectively. This increased proportion is to be expected with the passage of time, advanced cases of leprosy being left behind as a result of the sifting processes of repatriation and discharge.

RESULTS OF TREATMENT

An analysis of the results of treatment in this hospital since its inception is shown in Table 8. This pertains to the 1,067 cases that remain after deducting those repatriated to India (298) and the few (13) that have been released unconditionally as non-lepers. In it are given (a) the total figures relating to improvement, etc., and details of the relation of such changes to (b) race, (c) sex, and (d) type of case.

TABLE 7.—*Relation of type of case to deaths attributable to leprosy.*

Diagnosis	Number of cases	Neural					Cutaneous				
		N1	N2	N3	Total	Per cent	C1	C2	C3	Total	Per cent
Leprosy and exhaustion with senility	130	—	12	17	29	22.3	4	37	60	101	77.7
Septic absorption .	44	—	9	5	14	31.8	2	14	14	30	68.2
Nephritis ..	35	—	3	6	9	25.7	—	10	16	26	74.3
Total	209	—	24	28	52	24.9	6	61	90	157	75.1
Percentages of total deaths of each type.	50.1 [a]	0.0	25.2	45.9			37.5	53	76.9		

[a] Percentage of all deaths.

A total of 260 cases, or 24.4 per cent of all of the cases here considered, have been liberated under the regulations governing conditional discharge as having been free from all signs of leprous activity over a period of two years. A total of 33 of these cases were readmitted later, and 8 of them have been redischarged.

With regard to the results of treatment in the different races, a comparative assessment as between Indians and Fijians, which might well have been instructive, is unfortunately impossible because of the repatriation of so many of the former. With regard to these repatriates, it may be taken as a fact that all of them had improved up to a point before discharge but that many would probably have

retrogressed later, for 138 of them (46.3 per cent) were cutaneous in type. Nevertheless, there is a general impression at Makogai that the East Indian does better under treatment than the South Sea Islander. This fact, however, is probably due not so much to any inherent resistance or difference in reaction to the disease as to the Indian's anxiety to improve, which results in better care of himself and greater willingness to submit to dietary and other restrictions than is the case with the careless, carefree native of the group.

TABLE 8.—*Results of treatment (a) in total, and in relation to (b) race, (c) sex, and (d) type of case.*[a]

Group	Number of cases	Discharged	Quiescent	Improved	Stationary	Worse	Dead	Percentages	
								Discharged [b]	Improved [c]
A. TOTAL	1,067	260	79	189	71	51	417	24.4	49.5
B. RACE									
Fijian	443	146	23	46	25	22	181	33.0	48.6
Solomon	130	26	16	14	5	7	62	20.0	43.1
Rotuman	26	5	—	5	2	1	13	(19.3)	(38.5)
Samoan	34	7	3	8	1	2	13	(20.6)	(52.9)
Tongan	13	1	—	2	4	2	4	(8.5)	(23.1)
Cook	80	23	4	14	6	3	30	28.8	51.2
Maori	4	1	—	—	—	1	2	(25.0)	(25.0)
Niue	3	—	—	1	—	—	2	(0.0)	(33.3)
Indian	299	43	30	95	27	8	96	14.4	56.2
Chinese	18	4	—	2	1	4	7	(22.2)	(33.3)
European	17	4	3	2	—	1	7	(23.6)	(53.0)
C. SEX									
Male	776	198	53	126	54	40	305	23.1	48.7
Female	291	62	26	63	17	11	112	21.3	51.9
D. TYPE									
N1	121	58	22	19	9	—	13	48.0	81.9
N2	325	146	25	24	9	26	95	44.9	60.0
N3	140	41	25	6	5	2	61	29.3	51.4
TOTAL	586	245	72	49	23	28	169	41.8	62.4
C1	51	3	3	19	5	5	16	5.9	49.1
C2	257	12	1	80	32	17	115	4.7	36.3
C3	173	—	3	41	11	1	117	0.0	25.4
TOTAL	481	15	7	140	48	23	248	3.1	33.6

[a] Not including 13 nonlepers released unconditionally, and 298 cases repatriated to India.
[b] Patients discharged conditionally, disease arrested.
[c] Total cases improved, including those discharged conditionally.

Type and result of treatment.—Some of the data on the relation of the results of treatment and the type and stage of the disease are sufficiently interesting to be set forth separately. Outstanding is the difference as regard the conditional discharges, with 41.8 per

cent for the neural cases and only 3.1 per cent for the cutaneous. Even considering all degrees of improvement, the total figure for the former is nearly twice that for the latter. Details for the different degrees of advancement with regard to (1) discharged, (2) quiescent, (3) improved, and (4) total improved are as follows:

(1) DISCHARGED (Total 260 cases).

N1, 48.0 per cent (58 cases).
N2, 44.9 per cent (146 cases).
N3, 29.3 per cent (41 cases).
C1, 5.9 per cent (3 cases).
C2, 4.7 per cent (12 cases).
C3, no cases.

(2) QUIESCENT (Total 79 cases).

N1, 18.2 per cent (22 cases).
N2, 7.7 per cent (25 cases).
N3, 17.8 per cent (25 cases).
C1, 5.9 per cent (3 cases).
C2, 0.4 per cent (1 case).
C3, 1.7 per cent (3 cases).

(3) IMPROVED (Total 189 cases).

N1, 15.7 per cent (19 cases).
N2, 7.4 per cent (24 cases).
N3, 4.3 per cent (6 cases).
C1, 37.3 per cent (19 cases).
C2, 31.2 per cent (80 cases).
C3, 23.7 per cent (41 cases).

(4) TOTAL IMPROVED (528 cases).

N1, 81.8 per cent (99 cases).
N2, 60.0 per cent (195 cases).
N3, 51.5 per cent (72 cases).
C3, 49.0 per cent (25 cases).
C1, 36.2 per cent (93 cases).
C2, 25.4 per cent (44 cases).

The rates, on the whole, show the differences that would be expected between the different types of cases and the different degrees of advancement. Because of the contrast between the numbers of neural and cutaneous cases discharged, that in the quiescent list is consequently less. The reverse contrast in the proportions that were merely improved naturally follows from the preceding. It may be remarked that of the 15 cutaneous cases that were discharged 7 were readmitted on account of reactivation of the disease, so that only 8 cutaneous cases have remained free from activity.

With regard to results of treatment in the different sexes, it is found that the total of males improved, including those classified as "conditionally discharged," "quiescent," and "improved," amounted to 377, or 48.5 per cent, while the total of females improved came to 151, or 51.9 per cent. If the Indian repatriates, most of whom were greatly improved before leaving Makogai, were included, the males would have shown an improvement rate of 61.6 per cent and the females of 56.7 per cent.

It is evident that while cases in all stages of the disease may be improved under treatment, the prognosis as regards "cure" and ultimate discharge is very poor in the cutaneous type. These findings

enable us to corroborate the opinion of Harper (5)—difference in terminology being noted—who in a paragraph on prognosis says:

> . . . that all cases of purely skin leprosy (i.e., such as are characterized only by macules without demonstrable nerve lesion or nodule formation and without acid-fast bacilli in the nasal mucus) can be cured by modern methods, that many cases of nerve leprosy can be cured, and that some nodular cases can be cured. Cure or failure will thus largely depend on early diagnosis [and] treatment.

This is a truism, but one which needs to be stressed periodically. From the same viewpoint Neff (10) offers a word of warning regarding cutaneous cases:

> I am now strongly of opinion that while our Ordinance serves splendidly for maculo-anaesthetics and the more developed nerve cases, two years of inactivity is not sufficient for the few individuals, with true leprous infiltration of the skin, who become negative. Treatment abruptly ceases on their discharge and they return to their village life, rather outcasts usually, where they never take up their life as they left it and soon, through neglect, are covered with scabies and some form of ringworm probably. Under the circumstances, a fair percentage are almost bound to become, in a disease such as this, active again.

TABLE 9.—*Results of the follow-up of patients conditionally discharged.*

Race	Discharged	Readmitted	Died	No record
Fijian	146	25	25	19
Solomon	26	5	4	1
Cook	23	—	—	18
Samoan	7	—	—	—
Rotuman	5	1	2	—
Tongan	1	—	—	—
Maori	1	—	1	—
Indian	43	6	1	12
Chinese	4	1	—	—
European	2	—	—	—
Halfcaste	2	—	—	—
Total	260	38	33	50

As a result of the general tightening up of examinations, in particular of "quiescent" cases, it is hoped that the required two years of observation will prove sufficient to eliminate those who might run the risk of reactivation. The resolution of "true infiltrations of the skin" takes place very gradually, so that the process of healing has been going on for a considerable time, in the majority of cases, before any question of their admission to the "survey list" arises.

With regard to the follow-up of discharged patients, 210 out of the 260 cases have been examined periodically and reported on within at most a year of the time of writing. The data on these cases are given in Table 9.

Of the 50 cases that were not traced, 18 have returned to the Cook Islands, leaving 32 untraced in Fiji. There have been 38 readmissions, 5 of which were only for treatment of trophic lesions, so that there remain 33 reactivations; eight of these have been discharged. Of the 139 remaining after the deduction of those readmitted, untraced and dead, those remaining well are as follows:

Well for 16 years	1	Well for 8 years	10
Well for 14 years	3	Well for 7 years	5
Well for 13 years	2	Well for 6 years	12
Well for 12 years	4	Well for 5 years	12
Well for 11 years	13	Well for 4 years	17
Well for 10 years	3	Well for 3 years	8
Well for 9 years	5	Well for 2 years	17
		Well for 1 year	27

DISCUSSION

In an interesting paper recently published Strachan, of South Africa (13), appears somewhat sceptical with regard to the beneficial effect of chaulmoogra oil and its derivatives in the specific treatment of leprosy, and this scepticism appears justified by the results reported by him as occurring with little or no treatment. He claims that "more than fifty per cent of N1 patients undergo arrest spontaneously when placed under proper conditions." This is greatly at variance with the results given herein. At Makogai intensive treatment with chaulmoogra derivatives has produced only 48 per cent of arrests in early nerve cases, while with "treatment approaching adequacy" Strachan has had 79 per cent of arrests. With cutaneous cases our arrests amount to 3.1 per cent as compared with the African percentages of 3.8 for untreated and 13.2 for treated cases. Our numbers of early cutaneous cases are undoubtedly small, but we can only claim 5.8 per cent of arrest in such cases, as against Strachan's 21.4 per cent. One is almost tempted to echo, for these cases, the question of Burnet regarding the discharges from Culion which Strachan quotes: "What becomes of the liberated?" But this would be unfair.

There must, however, be some factor entering into the assessment of results that renders these two sets of records incomparable. It is admitted that among contacts of lepers there are probably numbers of people infected who, owing to a high degree of resistance, never show signs of the disease. This explains the long incubation period in some cases, and the incidence in such of our own cases as give a history of freedom from symptoms until the advent of pregnancy or of some debilitating condition such as dysentery. A better prognosis does appear to be justified in such cases, provided the antecedent condition is one that can be removed.

It may well be that the average native in the district dealt with by Strachan is habitually below par as the result of nutritive or infective factors. Such cases would naturally exhibit a low threshold to leprosy, and it is quite probable that the removal to "proper conditions," with good food and treatment of intercurrent diseases, would suffice to raise the resistance level and so produce a large proportion of spontaneous cures. In Fiji, on the other hand, the average native is comparatively well-fed, and he is accustomed to seek out the medical officer or native medical practitioner of his district for treatment of his ailments. His general resistance is therefore high, and when he does exhibit signs of leprosy general hygienic and other treatment are insufficient to produce spontaneous cure, and recourse must be had to the chaulmoogra preparations. This is, of course, pure speculation regarding the condition of the African native dealt with, but it is difficult to conceive of such gross differences in results without some such explanation.

Another recent paper, by Rodriguez, of the Philippines (11), works up to the conclusion that "the chaulmoogra oil derivatives do not seem to be as effective in incipient leprosy as in the more advanced cases with lesions showing acid-fast bacilli." If that be accepted at its face value it would surely be more logical to let our incipient cases develop into the more advanced grades in order to gain the benefit of chaulmoogra oil treatment. This conclusion, however, is not in harmony with our Makogai figures, which are based mainly on the use of chaulmoogra derivatives. It may, of course, be objected that our early nerve cases would have become arrested by the "proper conditions" referred to above, but this is purely hypothetical, and judged by our own experience of early cases that have neglected chaulmoogra treatment it cannot be accepted.

SUMMARY

Leprosy has been in existence in Fiji from a very early date.

A leprosy station was established by the Government of Fiji on the island of Beqa in 1900, and the present hospital at Makogai was opened in 1911. The number of patients has increased from 40 in 1911 to 427 in 1933.

Of the 1,365 cases recorded, 54.7 per cent were neural in type and 45.3 per cent were cutaneous. Investigation serves to indicate that there is no such condition as pure cutaneous leprosy without some degree of nerve involvement.

The sex ratio of the patients was 3.2 : 1, with 76 per cent males and 24 per cent females. No evidence appears that sex played any part in determining the type or course of the disease in these patients.

There have been 417 deaths in the series, of which about 50 per cent could fairly be attributed to leprosy.

Of the cases dealt with, 24 per cent have been conditionally discharged. There were 33 readmissions, of which 8 have been redischarged to date.

The most satisfactory improvement occurred in the earlier nerve cases, and it is found that while all types can be improved the prognosis of cutaneous cases as regards cure is comparatively poor.

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