# THE LEPROSY ENDEMIC IN NORTHERN RHODESIA WITH SPECIAL REFERENCE TO SEX INCIDENCE

DR. J. T. WORSFOLD

*Chitokoloki, Balovale, Northern Rhodesia*

The author's recent survey, 1957, in 'Lovaleland', which is the Balovale District of Northern Rhodesia,[1] revealed the progress of the leprosy endemic and some interesting data of sex incidence. This area long has been considered as of high leprosy prevalence. By analogy with other areas, this is not surprising, for it is an undeveloped area in the Zambesi basin and public health measures have not developed far: its general leprosy incidence is 11.85 per thousand. This was the first intensive leprosy survey in Northern Rhodesia. In 1950, Ross Innes did a sampling survey of certain areas which did not include the Western Province nor Balovale.[2] In 1932, Cochrane[3] had stated that the country had 10 per thousand of leprosy in parts of it. The Northern Rhodesian Medical Report for 1934 stated that 169 cases of leprosy were notified during the year and that Balovale was the most infected area, with 82 cases notified out of the 10 places mentioned. Muir[4] visited the country in 1939 and singled out Barotseland and Balovale, in particular, as having the highest incidence, and commented that the work of the small leprosy institutions which existed were of little value for the ultimate control of the disease, though noble relief work was done in them. In the 19 years elapsed since this opinion was given, its accuracy does not appear to have changed, for in 1949, the medical officer at Balovale described the very high incidence in that district (personal communication). The survey by Ross Innes[2] showed an average prevalence of 12.6 per thousand for the areas he visited in the Eastern, Southern, and Northern Provinces: the greatest prevalence was 25.6 per thousand on the Luapula River in the Northern Province. In comparison with the present author's findings in the Balovale District, he found a sex rate of 62.2, a childhood rate of 18.4, and a lepromatous case rate of 20.4 per cent (Balovale figures were 44.6, 2.5, and 23.75 per cent respectively). He concluded that: "Northern Rhodesian Leprosy is very much alive, and is slowly and surely increasing."

The present author's Balovale survey of 1957 indicates that a decline in prevalence may have set in, and he agrees with the opinion of Cochrane[5] that predisposing diseases and a lowered state of health have been overrated as epidemiological factors. There was also nothing to suggest any high contagion in leprosy and there was only one case of possible conjugal contagion. It was found that out of 234 adult leprosy cases, 119 were living with an uninfected

husband or wife, even though 21 cases were lepromatous, which hints at low grade infectivity even of lepromatous leprosy and brings in the importance of infection in childhood. The familial source of the infection was indicated in that 66.25 per cent of all cases admitted having a near relative with the disease; prolonged and close contact was probable and most acquired the infection before puberty. After puberty, every person marries and leaves the original house and often the village. Though a case presenting with leprosy may have had no contact with the infected relative for 10 to 20 years, there is a strong presumption that the relative was the essential source of the infection. Each successive age group shows a steadily rising prevalence. Therefore, it does seem as if in many cases leprosy is acquired in early life but does not become clinical until later life, i.e., there is a long *latent period*. The form of resistance in the patient which keeps the disease subclinical for years may be part of the immunological state of the population, but we do not know what causes the breakdown in resistance which leads to the clinical appearance of the disease. There is markedly less leprosy in the lower age groups, parallel with the absence of any decline in population in those age groups, which further suggests that the general incidence of leprosy may be tending to fall in this region. Further support for this is the finding in the Balovale survey of an increase in the average age of onset, which as Doull[6] pointed out occurs when the disease declines, even though leprosy is commonly contracted in childhood.

The *sex prevalence* of leprosy is accepted in most text books as 2 : 1 in favour of males. In 1934, Lowe ([7, 8]) made an exhaustive survey of the literature and found that so it was reported for most countries of high prevalence, though in countries of low prevalence, the females exceeded the males. Since Lowe made this review, Doull,[6] Hopkins,[9] Chaussinand,[10] Strong,[11] Romero,[12] Gehr,[13] and Arnold,[14] and others arrived at the same broad conclusion and most of them attributed the sex difference to inherent female characteristics rather than to environmental influences.

Institutional sex rate figures for neighbouring territories are 63.19 for *South Africa*, based on 1,020 males and 594 females in the four institutions, as given by Davison, A. R. in a personal communication; 64.40 for *Southern Rhodesia*, based on 713 males and 393 females in Ngomahuru, as given by Mostert in a personal communication; 58.60 for *Nyasaland*, based on 2,029 males and 1,433 females in five institutions, as given by Currie in a personal communication. For *Northern Rhodesia*, Ross Innes gave 62.2 sex rate for the three provinces of the East, South, and North: this rate does not have an institutional basis. The present author's Balovale survey in the Western section of Northern Rhodesia revealed the low sex rate of 44.6.

In this region it is likely that we see a fairly true picture of the prevalence of the disease, as leprosy work has been going on a long time and there is no evidence of a tendency to concealment of cases. Certain special factors operate in the other areas, such as compulsory segregation in South Africa, migration of males for labour from the north to Southern Rhodesia, and to the south from Northern Rhodesia and Nyasaland. There may not be much significance in this migration of males as a medical certificate must be obtained before they leave for work.

Cochrane[3] on the basis of the observations of Sharma thought that sex incidence may be more important than any other factor for the purpose of estimating the state of a leprosy endemic, but said that the endemic may be on the increase when the preponderance of males is reduced. Our findings suggest that the endemic may be declining when females preponderate. Both these propositions may have validity in the sense that an early reduction in the preponderance of males may indicate a temporary increase in the endemic which ushers in a general decline. This may be just what we are observing here in this region. Our figures from Chitokoloki Leprosarium are also of interest since it receives patients from a wider field than Lovaleland, and some patients come from over 1,000 miles away.

SEX PREVALENCE OF ALL RECORDED PATIENTS 1930–1958 AT CHITOKOLOKI LEPROSARIUM

| | *Males* | *Females* |
|---|---|---|
| Present inpatients ... ... | 86 | 98 |
| Present outpatients ... ... | 104 | 150 |
| Former patients ... ... | 1,189 | 1,325 |
| Rehabilitation villages ... | 128 | 143 |
| Outpatients in districts ... | 238 | 565 |
| | 1,745 | 2,281 |
| Total patients: 4,026. | Sex Rate: 43.3 | |

The high female leprosy prevalence rate, therefore, damages the concept of a universal male preponderance, though it may be true for most countries. We think that in Africa the movement of peoples from primitive conditions towards civilization brings them through a stage of susceptibility to leprosy and possibly an early male preponderance in incidence because the males make the first steps. With the later inevitable dissolution of tribal disciplines, the more conservative females enter more into the prevalence of leprosy, and changes in domestic and economic conditions are more potent in this respect than any postulated sex differences in susceptibility. In this part of Northern Rhodesia, there is not the Islamic influence on the degree and rate of the emancipation of women. Their pro-

gressive freedom in social contacts goes on steadily, together with participation in all the civilized benefits of health, hygiene, and housing, and leads to the paradox, in one stage at least, of participation in a heavier leprosy incidence. Their growing freedom includes a freedom to acquire leprosy. Later, there should follow a fall in the total incidence, in which females would share, and final eradication would become possible. The Lovaleland findings do suggest that the idea of an inherent lesser susceptibility of the female requires some modification. Any protection against other diseases by the female endocrine functions is also disputable: in these diseases also social and environmental influences are probably much more important.

Kerr as quoted by Tolentino[15] said that the women and men are equally affected by leprosy, provided the chances are equal. Muir ([16, 17]) and several other authors record areas with an excess of females in leprosy. Davey also from his experiences in Nigeria affirmed in a personal communication that the sexes are affected about equally and that he had never found anything comparable to the 2 : 1 sex ratio. Manson Bahr[18] stated that sex seemed to have little bearing on the liability to leprosy.

Reverting to the Chitokoloki figures previously given in this paper, the preponderance of females is clear in all the groups given, but there is evidence that it co-exists with a decline in the incidence of leprosy, such as the very low childhood rate and clinical features which include lesser incidence of lepra fever, nerve abscesses, and acute leprous eye conditions. Furthermore, there is a high percentage of residual cases, which strongly suggests a previous rise in total prevalence followed now by a decline.

The decline of the leprosy endemics in Norway and England has been ascribed to effective segregation and improved hygienic conditions, one or both with differing emphasis, and both these factors have been in operation in Lovaleland, and it seems possible that the next few years will witness a rapid decline in the prevalence of leprosy. If this proves to be true, there will be a temptation to give the whole credit to the sulphones but there is much evidence that the decline had set in before sulphone therapy was introduced in Northern Rhodesia in 1950. There is no doubt that this therapy is hastening greatly the complete control and final extinction of leprosy.

**Summary**

The author in 1957 completed the first intensive survey in Northern Rhodesia, in the Balovale District which lies in the Zambesi basin. He found an incidence of 11.85 per thousand. Previous wider but more cursive surveys were those of Cochrane in 1932, who estimated 10 per thousand for parts of the country and Ross Innes

in 1950, who sampled the Eastern, Northern, and Southern Provinces and found an average of 12.6 per thousand. The recent Balovale survey of the author indicated that a decline in prevalence may have set in, and showed a clear preponderance of female sex incidence, which the author thinks is more rooted in social and environmental factors than anything inherent in the female, and may be a stage leading to a decline in the endemic. The stage of an early male preponderance may have been passed through already. Other authors are quoted in support of the view that the male sex preponderance is by no means universal, and there is no essential inherent sex difference in the reaction to leprosy. The author describes a decline in leprosy prevalence in his area which began before the sulphone therapy and which is likely to go on and be greatly hastened by it.

REFERENCES

1. WORSFOLD, J. T. Central African J. of Med., **3**, Nos. 9 and 10, Sept. and Oct. 1957, pp. 359–365 and 401–406 resp.
2. ROSS INNES, J. Leprosy Survey in Northern Rhodesia, 1950. East Africa Med. J., **28**, Jan. 1951, pp. 21–28.
3. COCHRANE, R. G. Leprosy in the Rhodesias. Leprosy Review **3, 1**, Jan. 1932, pp. 25–28.
4. MUIR, E. Leprosy in N. Rhodesia. Leprosy Review **11**, 1: 1940, pp. 18–24.
5. COCHRANE, R. G. Practical Textbook of Leprosy, 1949, p. 20.
6. DOULL, J. A. Salient Features in the Epidemiology of Leprosy. The American Assoc. for the Advancement of Science, 1938, p. 108.
7. LOWE, J. Sex Incidence in Leprosy. Internat. J. of Leprosy **2**, 1934, p. 58.
8. *Ibid.* Napier's "The Principles and Practice of Tropical Medicine", p. 492.
9. HOPKINS, R. Heredity in Leprosy. The Amer. Assoc. for the Advancement of Science, 1938, p. 116.
10. CHAUSSINAND, R. Internat. J. of Leprosy, **15**, 4, Oct.–Dec. 1947, pp. 406–416.
11. STRONG, R. P. Stitt's Tropical Diseases, 7th Edn. p. 822.
12. ROMERO, A. and BRENES IBARRA, A. A. Revista Med., Costa Rica, **8**, 1948, pp. 294–297.
13. GEHR, E. Zeits. Tropenmed. v. Parasit. **2**, 1950, pp. 193–198.
14. ARNOLD, H. L. "Modern Concepts of Leprosy" 1953, p. 9.
15. TOLENTINO, J. G. Journal of Phil. Is. Med. Assoc. **15**, 1935, pp. 374–377 quoting KERR, I. Leprosy Review, **4**, 1932, p. 165.
16. MUIR, E. Leprosy Review, **9**, 1: Jan. 1940, p. 40 and p. 51.
17. *Ibid.* Manual of Leprosy, p. 16.
18. MANSON BAHR, P. Manson's Tropical Diseases, p. 555.