The Evolution of Tuberculosis in Southern Rhodesia

BY

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PART I

HISTORY: THE INTRODUCTION AND SPREAD OF INFECTION

Before 1890

A good account of the migrations of the Bantu tribes, as far as they are known, from the north-west corner of Africa to their present habitat throughout East, Central and South Africa is given by Theal (1919).

From a study of their history some idea of the people with whom the Bantu had come in contact before 1890 may be gained. There is the remote possibility that they carried tuberculous disease with them when they first entered the continent, though this is unlikely in view of the acute form which it takes when it attacks them. It is most improbable that they ever met the ancient miners of Rhodesia, nor are the Bushmen likely to have been a source. From the estimated course taken by Theal's Group II (including the Zulus and their offshoots the Matabele and the Shangaans), which was westwards to the Guinea coast and then south-east to reach Natal, it may be conjectured that they probably at no time came in contact with infection. The Matabele in their passage to the west and north after the wars of Chaka had only the distant contact of hostile neighbours with the advancing Boer trekkers, whereas it is essentially close and intimate contact that spreads tuberculosis; thus the Matabele were probably free from the disease. The tribes which advanced down the east coast (which include the MaShona group) are much more likely to have had intimate contact with Arab traders and slave raiders; especially does this apply to the VaKaranga, in whom, according to Theal, there is a strong admixture of Arab and Indian blood.

The Tuberculosis Research Committee of the South African Institute for Medical Research (1932) went into this question. They gave the opinion that in the widely scattered condition of the tribes, "the passing on of bacterial infection from one person to another is limited to a far greater extent than in more sophisticated communities: and more especially, in diseases of slow development like tuberculosis, sporadic cases, if they occur, tend to die out with individual or family." Those who had had opportunities for studying such communities before tribal conditions were unduly disturbed had almost always found tuberculosis conspicuous by its absence both in humans and in their cattle.

Livingstone, in his letters, often referred to the rarity of tuberculosis and scrofula among the indigenous population (*Central African Journal of Medicine*, 1955).

An important point against the previous existence of tuberculosis is the fact that the less contact an African group has had with immigrant races, the lower is the degree of tuberculisation of that group.

Again, strong evidence of its absence is the difference in response to infection displayed by the Bantu to that of Europeans. They suffer severe, acute and highly fatal disease, suggesting a community which is ill-adjusted to tuberculosis through lack of experience of it; or more specifically, one which has not been made resistant through many generations of natural selection, and in which most individuals have not had a primary protective infection such as occurs in communities which have been harbouring the disease for a long time. This view is further supported by the increasing chronicity of tuberculosis in the Bantu, which has been noted in all parts of Southern Africa, indicating that the people's resistance is increasing simultaneously with their experience of the infection.

Since 1890

After colonisation, one of the first official recorded utterances on tuberculosis was an extraordinarily perceptive one by Dr. Fleming, the first Medical Director. He said in his report for 1906: "Phthisis (in the Natives) shows a marked increase . . . and this is a matter of primary importance, bearing as it does on the dissemination of it among the Native population and its early introduction into their kraals. . . . The segregation of all affected is a possibility if controlled by legislation and could easily be undertaken before the disease spreads to Native districts."

It soon became evident that Natives employed in the mines had much greater sickness and mortality rates than those in the kraals or in domestic service, and that tuberculosis contributed substantially to these. From 1907 the Government adopted the principle in use in South Africa of sending back to their own homes

all cases of tuberculosis discovered on mines who were fit to travel.

From 1912 it was clear that tuberculosis had spread to the kraals to a small extent, carried there by returning mine workers. In 1913 the first rural medical unit was started at Ndanga, but it was not until 1933, with the expansion of services for the treatment of Natives in the reserves, that an estimate of the extent of the disease in these areas could be made. At the Ndanga unit (1933): "Pulmonary tuberculosis was found to be by no means uncommon, and there were typical advanced cases in women and children who had never left the reserve."

Since 1949 various tuberculin surveys have been carried out to determine the distribution of infection in different parts of the country. In 1950 two surveys were carried out on school children: one urban and suburban, in Bulawayo, and the other rural, in Mashonaland. In the former group the sensitivity rates varied from 10.5 per cent. at ages 4-7 to 40.8 per cent. at ages 16-19; in the latter group from 10 per cent. at ages 1-5 to 20.6 per cent. at ages 11-16. The urban group thus had higher rates than the rural, but even more interesting is the fact that both are considerably lower than the rates found in the present survey in Manicaland, to be described later. (The method of testing was the same except that (a) the Old Tuberculin was diluted 1/1,000 instead of 1/2,000, from which one would expect, if anything, more and not less positive reactors; (b) the criterion of a positive reaction was 5 mm. instead of 6 mm., but few reactions of these sizes occur, as is shown in Fig. II later in this paper.) It may be that even the past eight years have seen a considerable extension of tuberculous infection in the Colony.

Table I

Tuberculosis Cases and Deaths Notified in Southern Rhodesia, 1945-57

Year	Cases per 100,000	Deaths per 100,000	Case Mortality %
1945	18.6	4.4	23.4
1946	19.1	5.3	27.6
1947	14.4	3.1	21.6
1948	19.8	4.1	20.5
1949	22.8	4.3	19.0
1950	36.0	7.7	21.3
1951	36.0	10.2	28.3
1952	46.3	8.7	18.9
1953	61.3	7.8	12.8
1954	67.7	7.0	10.3
1955	82.2	6.8	8.3
1956	95.8	8.8	8.0
1957	106.9	10.6	9.9

Morbidity and Mortality Trends Since 1945

Morbidity rates, obtained from notifications, are no guide to the actual incidence of tuberculosis, since a great many cases never come to hospital. The notifications between 1945 and 1957, however, are interesting in the trend they show (Table I and Fig. I). There is a rapid and consistent increase, with a more than five-fold rise in the 13 years. Though part of this is undoubtedly due to better notification and a greater tendency to come to hospital, there is little room for doubt that there has been a real and substantial increase in cases.

Table I and Figure I also show the deaths reported over these years in the form of case mortality and of deaths per 100,000. As these deaths are only in those occurring in hospital, they are too low in the same way as the morbidity rates, but more so as the death rate is higher in those not receiving treatment.

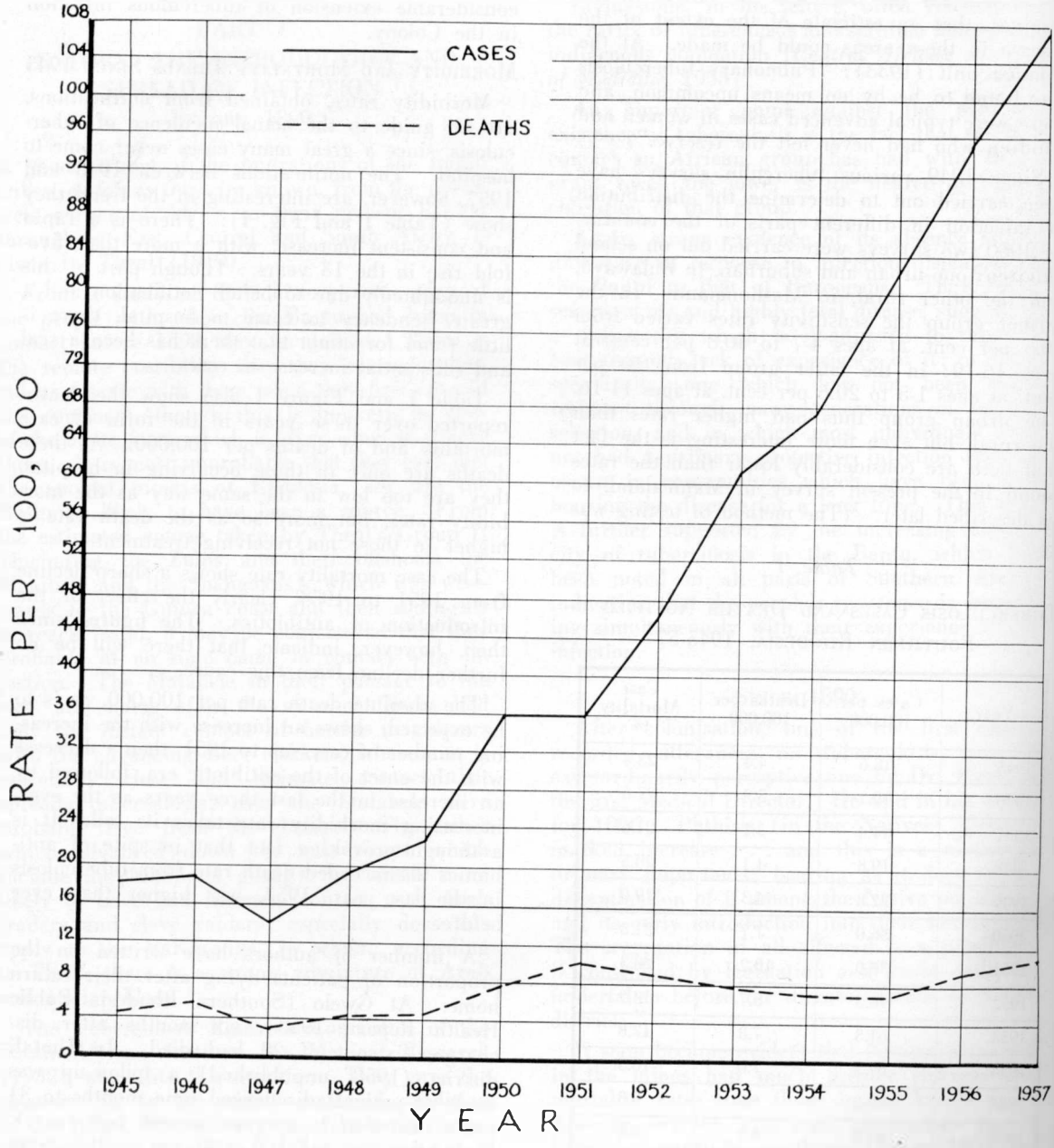
The case mortality rate shows a sharp decline from 1951 to 1954, clearly the effect of the introduction of antibiotics. The figures since then, however, indicate that there will be no further decline from this cause.

The absolute death rate per 100,000, as is to be expected, shows an increase with the increasing number of cases up to 1951, then a decrease with the onset of the antibiotic era, followed by an increase in the last three years as the ever-increasing morbidity rate takes its toll. It is a thought-provoking fact that in spite of antibiotics the recorded death rate from tuberculosis in the last year, 1957, was higher than ever before.

A number of authors have written on the proportion of patients dying after their return home. At Gwelo (Southern Rhodesia Public Health Report, 1953), six months after discharge, six out of 28 had died. In Umtali district (1958, unpublished) a follow-up was made of patients discharged nine months to 3¼ years beforehand (average period, two years). Eighty-five out of 106 (80 per cent.) were found, and of these 17 (20 per cent.) had died.

Figure 1

T.B. Cases and Deaths Notified in S. Rhodesia, 1945-57.



Of all cases *notified* in Umtali, Inyanga and Makoni districts and followed up after the same period, 306 out of 485 (63 per cent.) were traced, and of these 72 (24 per cent.) had died. Of 204 notified cases followed up on the Gold Coast by Eddey in 1943-44, 88 per cent. had died after two years (Singleton, 1957). Seventy per cent. of cases followed up in Tanganyika one to two years after discharge were known to have died (Clark, 1951). The mortality after discharge in Southern Rhodesia thus seems to be considerably less than in territories further north. The case mortality in hospital over the same period has been about 9 per cent.; and since about 20 per cent. have been found to have died two years after discharge, one may safely multiply the reported mortality rate by three. Allan (1932) did a two-year follow-up on 691 miners "repatriated" from the Rand to the Transkei and Ciskei Native territories during the years 1926-29. Of 475 whose fate was ascertained, 60 per cent. had died. In the same period (Tuberculosis Research Committee, 1932), 722 cases died in the mine hospitals, while 3,389 cases were repatriated. This means that if 60 per cent. of these 3,389 died within two years of repatriation, there were 2,033 such deaths for 722 on the mines, or 2.8 deaths for every one recorded on the mines. This figure is thus reasonably close to that found for discharged hospital patients in the Eastern Province of Southern Rhodesia, where the ratio is 2.2/1 (above).

If, on this basis, the notified mortality rates are multiplied by three, the rate at present is about 30/100,000, not including patients who have never been treated in hospital.

It is extremely difficult to analyse the distribution of disease within the Province by studying the notifications, and hence to derive the effects of urban life, industry, farming and climatic conditions on tuberculosis. The only information of value that can be obtained is a minimum morbidity rate: for 1958 this was about 108/100,000. The total populations of farm labourers and rural industrial workers are not known; these may be more carefully supervised and therefore come more often to hospital; addresses given by patients are often not their permanent address, and hospitals often give a kraal name which cannot be identified as being in a particular area. The greatest difficulty, however, is that the distribution of notifications is clearly related to the distribution of the hospitals, and fewer cases are reported from areas less accessible to a hospital or clinic.

Though it seems paradoxical, more accurate information about morbidity in different areas can be derived from the infection rates than from a study of notifications. In the Manicaland survey an effort has been made to evolve a picture of disease distribution from the reactor rates discovered.