

TUBERCULOSIS

IN THE

BANTU

of

SOUTHERN RHODESIA.

4/1/52

by

D. H. Shennan, M.B., D.C.H., D.P.H., D. T. M. & H.

A Thesis

Submitted to the University of Cape Town for the degree of
Doctor of Medicine.

1955.

The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

ACKNOWLEDGEMENTS.

Warm thanks are extended to the following people whose willing assistance with work not normally within their sphere made this study, not only possible, but pleasant to carry out:

Dr.J.Montgomery, Senior Government Medical Officer, Umtali, for access to radiological facilities and for kind advice and encouragement.

Dr.J.Addison, Honorary Radiologist to the Umtali Hospital who reported on the 49 X-rays.

Mr.K.Dunn, headmaster of Mutanda African School, for willing cooperation in the blood sedimentation rate and B.C.G.-life experiments.

Mr.D.Black, Government Health Inspector, who put in many hours on the arduous clerical side of the jute mill investigation.

Miss J.Bain, Senior Technician at Umtali Public Health Laboratory, who set up the 268 blood sedimentation rate tests.

Mrs.G.Sanders, radiographer at Umtali Hospital, who took and developed the X-rays.

My wife, who did the sterilizing in the early part of the survey, gave much assistance with the clerical work, and drew the many copies of diagrams that were required.

Native Lay Vaccinator Reuben Dube, who worked willingly as assistant in the tests at all times of the

night and day, and has been an invaluable standby in the minutiae of boiling-up, remembering details, etc.

All the mission superintendents concerned who gave hospitality and ready cooperation in the carrying out of the tests, and all the African school teachers who placed their pupils at our disposal to the derangement of their school time-tables.

INDEX.

	Page.
Introduction.	1.
Part I. Review of the relevant literature	3.
I. The home life and social background of the Bantu	3.
The Keynote	3.
The social order	4.
Religion and its counterpart	5.
The agencies of change in the social structure.	7.
II. The special nature of tuberculosis in Africans	14.
A. The disease process in individuals	14.
B. Reasons for racial differences in reaction to tuberculous infection	17.
C. The increasing chronicity of Bantu tuberculosis.	20.
Summary.	21.
III. The history of tuberculosis in Southern Rhodesia	22.
A. The question of the existence of tuberculosis in the Bantu before their contact with Europeans	22.
B. The history of tuberculosis in Southern Rhodesia since colonization, 1890	24.
IV. Tuberculin sensitivity	27.
A. Prevalence of tuberculin sensitivity in Southern Rhod. Tuberculin test surveys of Africans, 1949-54.	27.
B. Prevalence of tuberculin sensitivity among The Bantu of other countries.	27.
1. Union of South Africa.	27.
2. Kenya.	31.
3. Tanganyika	32.

Index - continued.

	page.
4. Uganda	32.
Conclusion	33.
C. The meaning of a positive reaction	33.
Summary	35.
D. The nature of the reaction	35.
Summary	38.
V. Morbidity and mortality rates	38.
A. Morbidity and mortality rates in Southern Rhodesia	38.
B. Morbidity and mortality rates in the Bantu of other countries	42.
1. Union of South Africa	42.
2. Kenya	44.
3. Tanganyika	44.
Conclusions	44.
VI. The epidemiology of tuberculosis	46.
A. Spread in isolated communities	46.
B. The epidemiology of tuberculosis in certain Native areas	47.
C. General rules governing the spread of tuberculosis in African territories	50.
D. The epidemiological situation in Southern Rhodesia as far as we know it	52.
VII. Non-pulmonary tuberculosis	53.
A. Incidence of non-pulmonary tuberculosis in Southern Rhodesia	53.
B. Non-pulmonary tuberculosis among the Bantu of other countries	54.

Index - continued.

	Page.
C. Infection with the bovine bacillus	56.
Summary	57.
VIII. Tuberculosis on mines	57.
A. In Southern Rhodesia	57.
1. Morbidity and mortality statistics for phthisis on mines	57.
2. Report of the Silicosis Commission, 1938	62
The causation of silicosis	63.
General characteristics of silicosis	64.
The relation of silicosis to tuberculosis.	64
The prevalence of silicosis and tuberculosis on the gold mines of Southern Rhodesia	65.
Prevalence of silicosis and tuberculosis on 13 selected gold mines	65
Asbestos mines	69.
Wankie Coal Mine	70.
B. Tuberculosis on mines in the Union of South Africa.	70.
Possible factors in the production of tuberculosis on the mines	72.
Conclusions	73.
<u>PART II. Field work.</u>	76.
I. Survey of tuberculin-sensitivity rates in the Eastern Region, Southern Rhodesia	76.
A. Tests on school children and teachers	77.
(I) Tests with 2 T.U.	77.
1. Crude sensitivity rate	77

Index - continued.

	Page.
2. Sensitivity rates at ages	77.
3. Degrees of positivity	78.
4. Sex and tuberculin sensitivity	79.
5. Comparison of different schools	80.
6. Family spread of tuberculin sensitivity	82.
(II). Tests with 5 T.U.	84.
1. Crude sensitivity rate	84.
2. Sensitivity rates by age and sex	84.
3. Comparison of the different schools involved.	85.
B. Tests on industrial employees	88.
1. Tuberculin sensitivity rates	88.
2. Tuberculin sensitivity and duration of employment	89.
3. Tuberculin sensitivity and nature of employment	90.
4. Tuberculin sensitivity and tribes and countries of origin	90.
Summary and conclusions from sensitivity rate surveys	93.
II. The criterion of a positive reaction in the Bantu	95.
Preliminary: study of reaction sizes by W.H.O.	95.
A. Measurement of reaction sizes in Eastern Region school children	96.
B. Tuberculin tests with control injections using heated tuberculin	98.
III. The significance of differences in the nature of positive reactions	100.

Index - continued.

	Page.
IV. B.C.G. conversion rates and complications to B.C.G.	109.
A. The use of tuberculin-allergy as a guide to B.C.G.-induced immunity	109.
B. Nett conversion rate	110.
C. Conversion rates by level of schools and by interval after giving of B.C.G.	111.
D. Complications to B.C.G.	112.
E. 6-month reversion rates	113.
F. Size of B.C.G.-induced positive reactions	114.
V. Field methods to be adopted	116.
A. Technique of giving injections	116.
B. Organization within a school	119.
C. Organization - general	122.
<u>PART III</u> General conclusions	127.
Prevention - general principles	128.
General consideration of the prevention of tuberculosis	131.
A. The mines	131.
B. Urban and rural	133.
Role of the African in a tuberculosis service	138.
Vital statistics	139.
The corner-stone - individual resistance	139.
Programme of immediate preventive measures to be carried out by an individual regional health department	140.

Index - continued.

	Page.
The use of B.C.G. vaccination	141.
Use of strongly positive reactions for case-finding	147.
Other aspects.	148.
Appendix I: History of the African people before 1890.	
Appendix II: Map of the Federation of Central Africa showing tribes etc.	
Appendix III: Map showing schools tuberculin tested	
Appendix IV: Tables.	
References.	

TUBERCULOSIS IN THE BANTU

OF

SOUTHERN RHODESIA.

INTRODUCTION.

In many African territories tuberculosis is an increasing problem. Southern Rhodesia is no exception to this rule. The great difficulty is the very wide front on which it needs to be faced; the claims of industry on the African, overcrowding in towns, inadequate nutrition, lack of education and tribal breakdown are all great problems if taken separately, but to make an effective attack on tuberculosis they have to be dealt with together.

Tribal nomenclature is very variable, and in the following pages the convention has been used of putting a capital letter at the beginning of the name, and again at the beginning of the stem; if the latter is not done the name is often difficult to recognize. For instance, the Shangaan tribe may be

referred to as AmaShangana, or the Ngoni as ANgoni.

In Part I, as there are not many long tables, and those used are mainly of immediate relevance, they have been included in the text instead of as an appendix. The tables for Part II are to be found in Appendix IV.

The references are in the form: "(Haynes, 1951)", and are given in full, in alphabetical order, at the end.

Places in Southern Rhodesia and its neighbourhood referred to in the text have been included in the map, Appendix II.

PART I.

REVIEW OF THE RELEVANT LITERATURE.

1. THE HOME LIFE AND SOCIAL BACKGROUND OF THE BANTU.

There is not room in this text to include an account of the history of the Bantu, which is related to their present way of life, but it is given briefly in Appendix I.

In the consideration of any public health measures it is necessary to consider the nature of the society into which they are to be introduced. Tuberculosis in particular requires a thorough knowledge of it, so that one can study its epidemiology in the community, understand the people's conception of the cause of the disease, discover the measures for its elimination that will be acceptable, and decide through what channels these may be most effectively applied. The effects of kraal society are also of great importance in considering the urban African and the control of tuberculosis in towns and on mines.

If we consider the MaShona, this will cover the great majority of the people, and where there are marked differences from the other tribes these will be mentioned.

The Keynote.

Charles Bullock (1950) emphasizes that the key to the understanding of the MaShona, and the basis of their whole tribal organization, religion and tradition, is the belief that a man's immortality depends on his having a son, in whom

his spirit will continue to dwell when he dies. His son will become him when he sits in his hut, bears his name and takes his place. He will also have another part, which will join the unified ancestral spirit, which watches over the welfare of the family; but his real hope of immortality is continued earthly existence in the person of his son.

The Social Order.

Considering this, it is not surprising that the unit of society is the family, usually enlarged as polygyny is customary. The head of the family is the father. He receives and distributes all the cattle, allocates the land, and governs his family with a benign hand. Everyone in the family shows him respect. The family is closely bound together by ties of religion (the Mudzimu or familial ancestral spirit), by the ever-pervading desire to have descendants, by superstitions, and by the resulting system of common family property. On the other hand, there is relatively little connection with other members of the tribe, though families are loosely knit into subtribes under headmen or princes, and the subtribes are in turn governed by the tribal chief. The power of all the rulers, down to the father, or baba, of the family, is limited, and emphasis is laid on their responsibility for the welfare of the group as well as on privilege.

Bearing in mind this type of tribal structure it is clear that any approach to be made to the people with a

view to sociological study, rehabilitation, contact-
traeing or tuberculin testing must be done through the
father of the enlarged family living in the kraal. His
approval will be the password to success.

Though this form of society seemed stable and was not
open to political abuse, advancement and the development of
culture were hindered by two main features: the lack of contact
between the families in the society, and the belief in magic
and evil spirits. The witch-doctor or nganga was a powerful
member of the tribe, and independent of the Chief, who could
not deal with witches, magic and spirits. The witch-doctor
was quick to find an evil spirit in any social inventor, and
death was the usual punishment for thinking or acting differently

The Matebele nation, on the other hand, was organized on
a rigid military basis, under the strict and cruel despotism,
first of Mzilikaze, then of his son Lobengula. It was already
showing signs of weakness when Rhodes made his first approach
to Lobengula. It was finally crushed in the Matebele War of
1893, when Lobengula, disheartened, died while retreating.
It has nothing to teach us about a social order for present-
day Africans.

Religion and its counterpart.

Mwari is the God of the Cosmos, not interested in
individuals except in so far as they commit an act contrary
to natural law (as the MaShona know it) such as incest. Then
he may send pestilence or famine, and appeasement will be
necessary.

Mudzimu is the family spirit, and is more important.

It consists of the unified spirits of all the ancestors, and guards the family. It is jealous, and if offended inflicts punishment on the family, perhaps by causing the illness of a child.

Then there are the debasing spirits. Ngozi is the spirit of a wronged person such as a murdered man, which kills another in vengeance. Still "more degrading is the black incubus of magic, superstition and anti-religion that lies on the real religion and throttles any good that there may be in it".

(Bullock, 1950). These have no social or retributive function. The main medium of magic is the nganga. He also, however, cures by means of medicines, which still bring him a large clientele among the MaShona, though in recent years more and more people have been going to the Government clinics instead.

As a result of these beliefs, sickness and death are looked upon as the work of supernatural forces, either a punishment from the Mudzimu or the work of a witch. No facts are available as to the attitude towards tuberculosis in particular. Because of these beliefs, the African is a fatalist. He does not struggle against disease, for he knows that if he is to die, he will die; and if he is to live, he will live. Many an African with pneumonia has died because he would not muster up the will to fight it. Tuberculosis, above all diseases, requires a high morale and the desire to get better. It is therefore important from this point of view to rid the MaShona of the idea of evil spirits as quickly as possible, and to remove the idea of ancestral punishment. This will, of course, be part of a large-scale education programme, and will have more important indications than tuberculosis. Perhaps

unfortunately, it would involve destroying the socializing idea of the Mudzimu; but Christianity seems to be able to supercede this, and should be acceptable by a people with a great demand for emotional expression, and whose religious belief has been that of the Mudzimu.

The many details of kraal life, including birth, upbringing, marriage and everyday life, are very interesting and instructive when one is seeking to understand the African's background. This also applies to the practice of totemism and the kinship system. The "cattle complex" or the religious attitude held towards cattle, is of importance in that it inhibits satisfactory cattle farming and meat production in a country where first-class protein is scarce. All these aspects are dealt with in full by Bullock (1950) The agencies of change in the social structure.

The largest change has been brought about by the introduction of the system of wage-labour with the white man. From the beginning the chief industry was mining, principally gold, and also asbestos, chrome, coal and other minerals. Farming also became an important occupation, and of this cattle ranching, and the growing of tobacco and maize, were the main strings. There is much coming and going between the reserves and the areas of employment, and the majority of mine employees stay only for a few months. Many go home at the time of the maize harvest, when their help is most needed. The result has been on the one hand, a weakening of the man-power in the kraals, and on the other hand, what is today Southern Rhodesia's biggest problem, the growth of large urban African populations housed in locations and compounds without any

established social structure. Thus we have two groups of people to think of:

(1) The kraals: These are situated in the Native Reserves, where the advent of the European has had the least effect on the African. None the less, the changes have been fundamental and have had far-reaching effects on the social structure.

The fact that taxes are now paid to the Native Commissioners has given the feeling that there is a different authority receiving allegiance, and thus a fundamental change in the tribal structure.

The attraction of young men to industrial centres has had an important economical and social effect on the African populations all over South and East Africa. Dr. Margaret Read (1942) studied the effect of the excessive migration of young men that takes place from Nyasaland to the Union of South Africa, the Rhodesias, the Belgian Congo, and Tanganyika. She states in her conclusions that "in the last fifty years the labour demands of an alien economic enterprise have completely undermined the old economic life of the country, and are going to undermine its social life as well". She found village life becoming strained and tense, especially between in-laws, as a result of the absence of the men. Many men did not return and came to be called "the lost ones". She emphasises that "in all East and South Africa the economic future of the Africans in their villages depends upon the policy followed for meeting the European demand for labour". In addition, "the future of indirect rule, which can only be built on stable social life in the villages, is at stake". Wilcocks (1953) emphasises the disequilibrium caused in South

Central and East Africa by the migration of young men to and from the industrial centres. He quotes sources from the Transkei, Nyasaland, Kenya and the Belgian Congo, all expressing deep apprehension about the effects of industrial attraction of men away from their homes.

Under the Land Apportionment Act of Southern Rhodesia many of the Native Reserves allotted were mountainous and in parts arid. This led to ploughing of sloping land which should not have been ploughed, and soil erosion. Elsewhere pasture land has been turned into desert by over-stocking. Much, however, has been achieved by the Native Affairs Department by a programme of education, intensive soil conservation and husbandry.

The Act also set aside 800,000 acres as Native Purchase Areas to provide farms for individual Natives. This meant a considerable change in outlook towards land, as previously all land rights were centred in the Chief and the princes, and no individual owned land.

It is now intended by the Government ultimately to convert the whole of the Native Reserves to farms on the lines of the present Native Purchase Areas. This is a logical step now that European immigration has given the country a basically cash economy, and only those who contribute to this can thrive. In the Reserves at present there is very little incentive to development of the land, since it belongs to no particular individual but only to the people in general.

The Missions have had the effect of weakening the tribal religion, and this of course is basic in breaking up the patriarchal clan. Education, largely done by the Missions, has altered the attitude of the women, so that they will not

tolerate polygyny. They also demand European-style clothes, and men tend to find that one woman is all they can support.

The moral standards in the kraals are suffering due to the men who go to town for a short time and contract temporary unions with women there. They also bring back European finery to the women in the kraals.

The introduction of Native buses has been basic in bringing about the free coming and going between the kraals and the towns, and hence the changes in the kraals.

The "kaffir store" or small isolated store in the reserve, usually run by an Asiatic, buys surplus crops from Africans; thus the price factor alone tends to influence their farming, and the fertility of the soil is not conserved.

(2) The towns: The development of industry has led to the growth of large locations and compounds, inhabited mainly by males. These have lost the restraint and security of their home life, and in the towns there are no settled standards of conduct to take their place. The very isolation of the kraals makes it difficult for Africans from different parts of the country, and even different countries, to find common standards which can be an established code between them, even if it were natural for young people to make such a code.

The results have been as might be expected. There is much crime in urban areas. The breaker of a European-made regulation is put in prison with lawbreakers of all kinds, and he receives a severe jolt to find that he has been classed with what is in his eyes the worst sort of criminal, the

breaker of an established Native social law. It 11.
does not help his morale, he loses self-respect, and soon
he may become one of those whom he used to despise.

Urbanization has tended to degrade the lobolo system,
or the exchange of cattle between families at the time when
a bride passes the other way. A young man meets a quite
willing girl in the town, and they decide to get married. Her
father, who probably lives many hundreds of miles from his,
demands lobolo; thus lobolo, instead of being a bond between
two adjacent families, with a religious significance and of
moderate amount, becomes merely a bride-price, which was often
exorbitant until recent legislation limited the amount to £25.
In many cases it is not paid, and divorce follows.

A very serious defect in urban and farm life is the re-
lationship between African and European. There is a strong
tendency for the European to regard the Native as inferior, and
a resulting loss of dignity in employer-employee relationships.
Understanding between the two races is deplorably lacking. The
health worker in any field will achieve very much more if he
seeks to establish it.

It would be out of place here to suggest an answer to these
social ills, yet when approaching tuberculosis we can scarcely
be independent of them. There is a need for a sound organiza-
tion, both urban and rural, through which the Government can
operate to bring about social, agricultural, and public health
changes. Under the Native Councils Act, some areas have
councils consisting of the Chiefs and headmen, commoners
nominated by the people, and the Native Commissioner as guide,
leader and executive. Yet on the whole, more steps are needed

to encourage the initiative of the people themselves to adopt European schemes for the conservation and development of land, improvement of health, etc. Perhaps the best line is to encourage an increasing degree of local government in the reserves, along with improvement of education. The family is the natural unit of government. The family heads would form a sub-tribal committee, and the Native Council would receive by degrees more autonomy, the Native Commissioner (and perhaps other specialists) becoming less a director and more an advisor of the Council.

In the towns, there is an urgent need for an established society. So far, there are only Native Advisory Boards without executive functions. The people are not interested in these; in one election less than one-eightieth voted. A stable society is hard to create with the population moving constantly to and from the towns, and so lacking in the old and the young. The present locations are too big, and it has been suggested that small units might be formed, having a core of families who are permanent residents in each. To quote Bullock again: "Some sort of civic pride - some aspiration towards citizenship - might form the unifying spirit of a social group, which would probably benefit from a residential rearrangement. There might then be management boards with executive functions in subdivisions of the locations; and ultimately there might be representation on the city council". Though in certain places a fair amount of civic spirit has been developed, there is in general a need

^{more} for/recreations and social activities: churches, schools, libraries, recreation grounds, garden allotments, streets, shops, restaurants, clubs cinemas etc., Thus we would have formed small towns each generating its own local spirit with its own amenities, sports, and organized entertainments. An increased responsibility for their own affairs is a sound base from which a people may build their society, with its moral code, its unspoken taboos, and its own special character and customs. Responsibility will provide an incentive to social growth which previously was lacking.

II. THE SPECIAL NATURE OF TUBERCULOSIS IN AFRICANS.

A. The disease process in individuals:

S.P.I.M.R.

The Tuberculosis Research Committee, in their 1932 investigation of South African Natives, studied the literature on the effect of tuberculosis on isolated communities. The following is a review of some of their findings.

The effect of tuberculosis when introduced into a previously unexposed community - "the tuberculosis of virgin soil" - was studied by Borrel in Senegalese troops called to Europe for military service in the 1914-18 war. He found that "these practically uninfected persons - only 4-5% gave a positive tuberculin test on arrival - were exceedingly susceptible to tuberculosis and tended to develop the disease in a severe and generalised form. He found that the disease presented two stages - an initial glandular phase with no fever and no definite change in the general state, tending to last, under army conditions, for one, two, or three months; and a subsequent phase characterised by fever, emaciation and generalised lesions such as caseous lobar or lobular pneumonia, affections of the serous membranes, pleurisy and military tuberculosis, either primary or secondary. This latter phase was usually short, and death often ensued within a fortnight to two months after its inception.

It will be noted, however, that although cases which reached the second stage were almost invariably fatal, those detected early and while still in the 'glandular' phase, if placed at rest and given suitable food, tended to do well.

Borrel reports that 50% of such cases regained their health and were fit to be repatriated".

The Committee drew attention to the similarity of the disease in isolated communities to that in children. Each has the glandular phase, normally running a benign course but progressing to severe disease if the subject is under the conditions of stress. The analogy has been discussed by Prof. Lyle Cummins (1929).

Gilmour, (1952), observed that in Uganda, where tuberculin sensitivity rates were relatively low, most of the cases occurred in young adults. The history of onset was usually recent, and the disease characterised by gross, almost suppurative disease in the lung, associated with little evidence of repair, and gross enlargement of the mediastinal glands.

Wood (1948) studied glandular and serious tuberculosis in East African Natives, all men between 18 and 30 years of age.

36 cases presenting as primary glandular tuberculosis developed as follows :

<u>Course of illness</u>	<u>No.</u>	<u>Deaths.</u>
Confined to glands	16	0
Developed unilateral pleural effusion	16	0
General military spread with polyserositis	3	3
General military spread	1	1
<u>Total</u>	<u>36</u>	<u>4</u>

150 cases presenting as primary pleural effusion developed as follows:

<u>Course of illness</u>	<u>No.</u>	<u>Deaths.</u>
Unilateral pleural effusion	125	0
Consecutive effusions of polyserositis type	11	1
Polyserositis	<u>14</u>	<u>1</u>
<u>Total</u>	150	2

The work of Oswald is quoted to show that almost all Africans (96%) dying of tuberculosis have enlarged mediastinal, cervical or abdominal glands.

The author explains the relation of pleural effusion to the progress of the disease by saying the glandular enlargement may:

- (1) Remain as it is and subside.
- (2) Be associated with a pleural effusion.
- (3) If the spread in the glands is extensive, other serous membranes may be involved, and sometimes death may follow.
- (4) Breakdown of the glands will lead to generalised military spread.

Prognosis was shown to depend not so much on the extent of the infection as on the unpredictable liability at any stage to acute military dissemination.

Apart from their review of the work of Borrel, the Tuberculosis Research Committee (1932) recorded observations on cases seen by themselves on the Rand mines. They found that in the African the disease tends to run a much more rapid course than in the European, with a marked tendency to a

fatal termination.

Clark (1951) relates that of 300 patients with decisive clinical criteria of tuberculosis from the Fort Hall Reserve in Kenya, who were admitted to hospital between July 1948 and September 1950, 182 had died at some time subsequently. (The article was published in September 1951.)

B. Reasons for racial differences in reaction to tuberculosis infection.

The Committee concluded that the differences in susceptibility to tuberculosis between individuals, and races, appear to be due mainly to the acquisition of resistance by exposure to a small infection. This suggests that the African will become, and can be made, as resistant to tuberculosis as the European. There also seems to operate, as a minor factor, an hereditary ability to form immune bodies to combat the disease, and in this respect the African is probably at a disadvantage, as he has not had the natural selection which the disease has wrought among Europeans. (The Committee's later finding, that the tuberculin-positive mine recruits were more likely to develop tuberculosis while in employment than the tuberculin-negative, shows that for individuals the tuberculin test is no indication that immunity has been acquired).

Dorner, Friedlander and Wiles (1943) noted that in certain Native areas of the Union there was a large amount of infection and very little disease, whereas in urban areas, with a similar high rate of infection, there was a large amount of killing disease. They postulated that the disease was aided

in the case of the town Native by malnutrition and physical effort. They suggested that Europeans under the same conditions would react the same way. However, Prof. Lyle Cummins, reviewing their report in the Bulletin of Hygiene, upholds the more usual view, that the African reacts differently to the primary infection, displaying a high degree of allergy, which perhaps makes him particularly sensitive to physical strain.

The Tuberculosis Research Committee also relate the severity of the disease to the concentration of Native industrial recruits under the conditions inseparable from practically all commercial developments in Africa, together with the unaccustomed hard work and the unfamiliar housing and diet conditions of a new environment.

(In this connection, it may be noted that the Bills of Mortality, which have been estimated to give at least a fairly accurate idea of the proportion of deaths due to tuberculosis, showed that in London there was an increase in mortality from pulmonary tuberculosis between 1700 (12% of all deaths) and 1800 (26%). After this a drop began, pulmonary tuberculosis representing 12% of deaths in 1850, and 9 % in 1910 (Kayne, 1937). This "epidemic wave" coincided with the Industrial Revolution, when the sedentary village life of the people was largely exchanged for a factory existence. These changes are in many ways analagous to those occurring to the African who enters industry. It may be that the factors of overcrowding, hard work and a radical change of environment play a large part in the increase in tuberculosis amongst Bantu who have

entered industry, and that we should guard against laying too much stress on immunity due to previous infection, or on racial immunity or lack of natural selection.)

The Committee differentiate two types of pulmonary tuberculosis in the African mine worker:

- (1) Tuberculosis of the new recruit, which tends to generalization of the lesions, and which is acute and rapidly fatal.
- (2) Tuberculosis of the long-service worker with fibrosis which is in part due to silicosis, much more chronic and more akin to the European type of tuberculosis, with a lower mortality rate than type (1).

In the case of the acute type, it was considered, in the light of the tuberculin tests, (see page) that it occurred mainly in those who had received a primary infection at home, and in whom the primary focus had lighted up again under the stress of mine work. There were, however, a proportion of sufferers who were tuberculin-negative on commencing work, and who were, therefore, true primary cases of the type described by Borrel. These were probably much more common in the early days of mining.

In the case of the chronic type, the Committee felt that the higher resistance shown by this group was due: (a) mainly to the cumulative development of resistance resulting from constant small re-infections while at work. (b) to a less extent, to the development of the fibrotic limiting process of silicosis.

C. The increasing chronicity of Bantu tuberculosis.

Although the disease still tends to take a virulent form in the African, there have been several reports of a tendency to increasing chronicity in recent years. Allen, (1927), in a study of tuberculosis in Native colliery workers, noticed that there had been an increase in the number of slowly developing and recovering cases in the previous twelve years. He observed that African women tend to get a chronic form of the disease, similar to that of Europeans. He considered that the change had been apparent, not real, and was due to the greatly improved conditions on the mine. The acute fulminating cases that had occurred previously, and some of which still were occurring, he put down to stress and not to racial susceptibility. Allan (1932) found many chronic patients in the Transkei often with a course of three years or more. Dormer (1948) observed that "disease in the lungs in Native areas shows a tendency to fibrosis and healing". Haynes (1951) quotes several sources to support this, and gives his own reasons for believing it of Kenya. The Southern Rhodesia Public Health Report, 1951, says: "It is impossible to substantiate by statistics the view very generally held by clinicians who see most of the patients, that there is a decrease in the numbers of the really acute types of tuberculosis infections, with a corresponding increase in the more chronic types approximating more closely to those seen in Europe. This feature raises many problems, in that it is still a matter of great difficulty and rarity to render those patients bacteriologically negative, with all the risks of

further spread which are entailed in having large numbers of patients with moderately good general health, but still able to carry the infection to others. It would be rash to be dogmatic in this matter, but there is nevertheless a strong impression among clinicians that the Bantu, especially in urban surroundings, are slowly acquiring a far greater degree of resistance to this infection." The development of silicosis in long-service mine workers is another factor causing much greater chronicity of tuberculosis when it occurs.

SUMMARY:

There is a wealth of evidence to show that tuberculosis in the Bantu tends to take an acute, severe, readily generalized and rapidly fatal form, and that given rest and good food in the early stages, resolution often takes place. On the other hand, evidence has been obtained in the Union that this form does not occur under the African's natural conditions (an exception to this, however, being Uganda) and has led to a greater emphasis on the environmental changes associated with industrial life. Tuberculin testing of recruits to the Rand mines, showed, rather surprisingly, that more than twice as large a proportion of positive than negative reactors from the Native Areas developed tuberculosis while in employment, (see page), and this led the Tuberculosis Research Committee to lay even more stress on the environmental changes associated with industry, and moreover to postulate that most infections are a re-awakening of a previously-acquired infection. Haynes, of Kenya, disagreed with this, saying that such disease is caused by a fresh

infection in a highly allergic person, and Prof. Lyle Cummins also set much importance on allergy. This is one point which still requires clearing up.

Allan noticed that many of the pulmonary cases in the Transkei had a long course. Here we have a community living in its natural surroundings but subject to overcrowding and malnutrition. Under these circumstances it appears to react to tuberculosis just the same as a European community does - by a high endemicity, high prevalence, and chronic form of disease. It is only in the artificial circumstances of unaccustomed environment and hard work that the acute form of the disease occurs.

A point of general agreement is that recently tuberculosis has tended to become more chronic in the African. This has been observed in Southern Rhodesia. Of particular interest are the chronic forms noted on the Rand mines, with or without silicosis, and the explanation offered by the Tuberculosis Research Committee, that the relative immunity of such cases is due to repeated small infections.

III. THE HISTORY OF TUBERCULOSIS IN SOUTHERN RHODESIA.

A. The question of the existence of tuberculosis in the Bantu before their contact with Europeans.

The history and present distribution of the Bantu are summarized in Appendix I.

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 the disease came with them into the north-east part of the continent,

though this is unlikely in view of the acute form which it takes when it attacks them. They did not, of course, ever meet the ancient miners of Rhodesia, and the Bushmen are unlikely to have been a source. From the estimated course taken by Group II of the tribes, to the Guinea coast and then south-east, 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 Tchaka had only the distant contact of hostile neighbours with the advancing farmers, and it is essentially close and intimate contact that spreads tuberculosis; thus the Matabele were probably free from the disease. The tribes that advanced down the east coast are much more likely to have had intimate contact with Arab traders and slave-raiders; especially does this apply to the VaKaranga, in whom there is a strong admixture of Arab and Indian blood.

The Tuberculosis Research Committee in the Union, 1932, went into this question. They gave the opinion that in the widely spread 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 of studying such communities before tribal conditions were unduly disturbed, had almost always found tuberculosis conspicuous by its absence both in the humans and also in their cattle.

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

Again, strong evidence of its absence is the difference in reaction displayed by the Bantu from that of Europeans, as has been described above. Their reaction is very similar to that of childhood, and is strongly suggestive that they have not had a primary protective infection such as occurs in communities which have been harbouring the disease for a long time.

B. The history of tuberculosis in Southern Rhodesia since colonization, 1890.

The little band of pioneers was fully occupied in establishing itself in the first few years. However, the medical department was soon set up, and from the turn of the century annual public health reports were issued. Great credit is due to the Medical Director, Dr. Fleming, who by 1906 was writing, in the Public Health Report: "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 to 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".

Mining was from the beginning the Colony's main

industry, and it soon became evident that Natives employed in the mines had much higher sickness and mortality rates than those in the kraals or in domestic service, and that tuberculosis played an important part in these. From 1907 the Government adopted the principle in use in the Union of sending all cases of tuberculosis discovered on mines who were fit to travel back to their own home.

In 1911 the first case of "miner's phthisis" or silicosis was reported in an African, and subsequently it became increasingly common and developed into a serious problem.

From 1923 the influx of European sufferers from the disease who came to the country for their health was becoming serious, and the immigration of such people was forbidden. In spite of this a considerable number continued to arrive, and only a proportion of these were discovered and sent home. In 1951 a regulation was made under the Immigration Laws requiring all new permanent residents to submit a radiologist's report of freedom from active tuberculosis; this resulted in a marked decrease in the number of European cases in the following year.

From 1912 it was evident that tuberculosis had spread to the kraals to a small extent through the medium of the returning mine employees. In 1913 the first rural medical unit was started at Ndanga, but it was not till 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: "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". (Public Health Report, 1933)

In 1937 a large-scale investigation was undertaken by a Silicosis Commission of the Miner's Phthisis Bureau of the Union of South Africa, into the problem of silicosis and tuberculosis on the mines of Southern Rhodesia. Their report is considered later in this enquiry. The mines have always played the major part in the exchange and dissemination of the disease amongst the Africans.

In 1946 a sanatorium for Africans in the Chindamora Reserve near Salisbury was opened. In the same year, however, the new Medical Director, Dr. Morris, expressed the view that more important than this was the provision of acute isolation hospitals for the segregation and early treatment of infected cases. He also felt the need of a contact finding and observation service.

In 1947 the Nutrition Council was founded.

From the beginning there appears to have been no appreciable infection of cattle with tuberculosis. Of recent years, however, there has been an increase of non-pulmonary tuberculosis in Africans, and the question of bovine disease deserves review.

In 1949 - 51 a survey to determine the distribution of tuberculin-positivity and the effect on negatively-reacting persons of B.C.G. was commenced. It is discussed later.

Certain missions about the country have beds reserved for treatment of cases of tuberculosis.

IV. TUBERCULIN SENSITIVITY.

A. Prevalence of tuberculin sensitivity in Southern Rhodesia.

There was little definite information available on this subject, until 1949 when the tuberculin-testing ^{mentioned} ~~described~~ below was commenced.

~~2.~~ Tuberculin test surveys of Africans 1949-54.

The results of these have yet to be published, but they have shown that in the Northern, Western and Midlands Regions of Southern Rhodesia, the proportion of children who are Mantoux positive increases from birth until at the age of 20 it is, on the average, 50% or more.

No tests have previously been performed in the fourth or Eastern, Region.

B. Prevalence of tuberculin sensitivity among the Bantu of other countries.

1. Union of South Africa. The Report of the Tuberculosis Research Committee of the South African Institute for Medical Research, 1932, on "Tuberculosis in South African Natives", includes a survey done by Dr. Peter Allan of the Native Territories of the Transkei and Ciskei, and to a less extent of Basutoland, Natal and Zululand. Tuberculin tests were done in four areas: the southern and north-eastern coastal parts of the Transkei; and inland from these, the mountainous Northern Transkei and Basutoland, both over 5,000 feet.

The tests were done with 0.1 cc. of Koch's Old Tuberculin, 1/5,000.

In the southerly part of the Transkei, of 2281 rural Africans tested, 74% were positive.

In the eastern coastal part of the Transkei of 183 tests, there were 78.5% positives.

In the northern part of the Transkei, of 1211 tested there were only 40% positives, and in Basutoland, of 414, 50% positives.

(The population examined in the Southern Transkei was made up as follows :

Under 10:	651	15 - 19:	278
10 - 14:	610	Over 20:	742.

If the rates in each area are standardized to this population, they are as follows:

Southern Transkei:	74%
Eastern Transkei:	81%
Northern Transkei:	40%
Basutoland:	40%)

Similar investigations done in institutions and locations in these areas showed a slightly higher proportion of positives.

There was a steady increase through the age-groups up to 20 - 25 years, at about which age the maximum percentage of positives was reached.

Following up these observations, Dr.Allan found that the incidence of tuberculosis amongst mine boys coming from the latter two areas was higher than amongst those from the two more heavily tuberculized areas.

The authors conclude that the southern group shows quite a high degree of tuberculization as judged by the tuberculin test, and going with this a fair amount of endemic tuberculosis,

some of it "chronic". Boys from this area going to work on the Witwatersrand goldfield have a comparatively low tuberculosis prevalence.

The northern group shows a much lower degree of tuberculization as judged by the tuberculin test, and going with this, tuberculosis was uncommon, and the cases seen were of a mild type. (However, as appears later, the occurrence and severity of active cases is probably not related to the endemicity so much as to the local living conditions, as indicated by the Committee's next remark). The mountainous nature of the country and the scattering of the population are probably important factors here. Boys from this area going to work on the Witwatersrand goldfield had a high tuberculosis prevalence.

As between the sexes, tuberculization was even, taking all ages, but the girls ran ahead of the boys between the ages of 5 - 15 years. This may be because young girls are kept together in the huts while the boys go out to work. The men catch up again in early adult life when they return after a period of work away from home. (Perhaps, also, the less fit men, including the tuberculous, tend to stay at home while the fit go away to work).

As judged by the intradermal tuberculin test with 1/5,000 strength, the men of over 20 years of the coastal district are 88% tuberculin-positive, while those of the northern district are 69% tuberculin-positive.

Two reports deal with the incidence of tuberculin-sensitivity among children in the Union of South Africa.

Gampel (1952) did a study of urban Bantu infants up to one year of age born in Durban. Of 209 babies born in one district in 1949, 143 were tuberculin-tested, at approximately weekly intervals, by means of the Lederle patch-test, which was removed after 48 hours, and the result read 48 - 72 hours later.

There were altogether 24.5% reactors in the first year of life. After a statistical correction had been applied by the authors, this figure became 32.3%. The earliest occurrence of a positive test was at six weeks and subsequently the number of conversions was as follows:

	<u>No. positive</u>	<u>% of all those becoming positive in the 1st. year</u>
First 12 weeks	7	20%
First 24 weeks	17	49%
First 36 weeks	26	74.3%
First year	35	100%

Griffiths and Kreher (1953) gave the Mantoux test to 1,145 nursery school children between 1 and 7 years of age in the Johannesburg district. They used Old Tuberculin, 0.1 mg., then if negative 1 mg., then 10 mg. (that is 1/10 cc of 1/10,000, 1/1,000 and 1/100). 26% of the total gave positive reactions. There was an obviously rising incidence of positive reactors with increasing age.

In addition, 345 European children from poorer parts were tested, giving 10% positive results.

The results are given in Table V.

Table V.Results of tuberculin testing of African and European children.

<u>Age.</u> <u>years.</u>	<u>Number</u>	<u>Africans.</u> <u>Positive</u>	<u>% Positive.</u>	<u>No.</u>	<u>Europeans.</u> <u>Positive.</u>	<u>% Positive.</u>
1 -	29	3	10	3	-	-
2 -	124	19	15	54	1	2
3 -	264	49	19	95	16	17
4 -	298	78	26	121	13	11
5 -	279	96	34	68	5	7
6 - 7	151	51	34	4	-	-
Total	1,145	396	26	345	35	10

As far as mine workers are concerned, the Tuberculosis Research Committee, 1932, found from their tests that over 72% of the Native labour force apply for engagement already invaded by the virus of tuberculosis, as judged from their response to a single intradermal test with 1/10 cc. of 1/5,000 Old Tuberculin. Tests done by others with stronger solutions have shown the figure to be 90%

2. Kenya. In a comprehensive survey of tuberculosis in Kenya, Haynes (1951) found that African male urban residents aged 26 - 40 gave 90% reactors. The lowest rate found in a total of 15 districts was in a rural district, where it was 55.5%.

It was found that there were higher reactor rates near the coast; the same finding by Allan in the Native Territories in South Africa is noted, and the suggestion put forward that this is because this was the route of ingress of disease-bearing newcomers.

Certain areas, particularly the coastal, were found with a high reactor rate and low disease incidence, and others with a low sensitivity rate but high disease incidence. It is suggested that the former occur in places where the disease has been endemic for long periods, and the latter imply a more recent introduction of the disease. (It will be remembered that the situation in the Transkei is the reverse, and immediate environmental factors probably play a more important part in the determination of active disease than the lack of endemicity).

A comparison of urban and rural populations shows that urban populations have higher sensitivity rates. Also, a period of urban residence raises the incidence of tuberculin reactors in males tested in the Native Reserves in comparison with those who have never left the Reserves.

3. Tanganyika. In the Mbulu district in Tanganyika, Davies (1952) found that the incidence of tuberculin positivity at all ages up to 50, in 5,006 people living in 10 villages, varied from 30% to 69%, with a total rate of 47%.
4. Uganda. According to Gilmour (1952), Uganda's population includes Nilotes and Hamites as well as Bantu, and is still scattered in family groups, with few villages or towns. The people appear to be still "virgin soil" for tuberculous infection. Tuberculosis is, however, on the increase.

Of 4,403 persons tested with Old Tuberculin 1/10,000, 35.7% were positive. The children showed 16.7% positives and the adults 45.8%. The steepest rise in sensitivity was at 20 years of age.

Conclusion:

From these figures it will be seen that tuberculosis has become endemic in widely scattered areas of Africa, and in almost every region that has been tested.

The significance of "tuberculization" and its relationship to manifest disease touched upon in the foregoing section, will be discussed after a review of the morbidity and mortality figures available (page 50).

C. The meaning of a positive reaction.

A positive tuberculin reaction is accepted as denoting allergy to the products of the bacillus, though not necessarily immunity to the organism itself. It indicates that there has been an infection with tuberculosis in the past (provided that no B.C.G. or other anti-tuberculosis inoculation has been done). How long bacilli continue to live in the body, and how long tuberculo-allergy continues after they are all dead, are questionable matters. In Great Britain, where there is high endemicity and constant reinfection, it is generally accepted that once the tuberculin reaction has become positive, it remains so. However in areas of low endemicity, such as are common in Africa, we must consider the possibility of reversion of the reaction to negative some years after the subject has overcome an infection. In such a case negative reactions might be obtained in those who have had an infection, and the tuberculin sensitivity rate would give too low an estimate of the endemicity.

The follow-up done by the Tuberculosis Research Committee after tuberculin tests of Rand mine recruits showed that

Africans yielding a positive reaction to this test were more likely to develop a recognizable tuberculosis than were those yielding no response to the test. In those with a positive reaction, 855 per 100,000 developed the disease, whereas in the negative reactors the rate was 405 per 100,000. Thus in these subjects the possession of tuberculo-allergy was associated with lowered resistance against infection.

In those in whom disease developed later, the common form was a generalized or "natural" tuberculosis, whether the response to the test was originally positive or negative. The septicaemic type of tuberculosis arose most frequently in the negative reactors, so the possession of tuberculo-allergy was associated with some resistance to virulence, but not, as a rule, with sufficient resistance to localize tuberculosis. Such localized tuberculosis as did occur was most common in subjects showing tuberculo-allergy.

The possession of allergy was not associated to any useful extent with the ability to modify an after-coming tuberculosis. The Committee stated that the common form of generalized tuberculosis is the metastasizing type, involving lymphatic glands and producing caseating granulomata in the abdominal organs in addition to the pulmonary lesions. Chronic localized tuberculosis occurs in a minority of cases and is met with in two forms. One is chronic pulmonary tuberculosis, which is nearly always associated with silicosis. The other is local glandular tuberculosis, e.g. glands of the neck. In none of these types was the incidence related to the presence

or absence of tuberculin-allergy.

Summary:

It is possible that in communities less "salted" with tuberculosis, positive reactions may revert to negative in the absence of re-infection. This deserves note in conducting epidemiological surveys.

To the individual African a positive reaction is not the unmixed blessing that it is assumed to be for Europeans. It may mean an increased danger of developing tuberculosis at some future stage when the subject's resistance is lowered.

Practical work having a bearing on the significance of the positive tuberculin test is recorded on page 95.

D. The nature of the reaction.

The Tuberculosis Research Committee found that in Rand mine recruits, the greater the degree of tuberculo-allergy, as shown by the reaction to the tuberculin test, the less was the resistance against infection. The reactions were classed as "negative", "positive", or "strongly positive". Of 3,879 returned as "strongly positive", the incidence of tuberculosis was 15.47 per 1,000; of 57,236 returned as "positive", 6.83 per 1,000 developed tuberculosis.

Those cases of positive reactors who later developed tuberculosis, and who on admission to the mines had been passed as clinically and radiologically free from the disease, were regarded as having a "larval form" of tuberculosis, developing in the mediastinal part of the radiographic lung field. The degree of activity of this focus was related to

the degree of tuberculin-sensitivity, and would later determine whether the infection was likely to be overcome, or would break out into acute tuberculous disease.

Haynes (1951), after quoting this report, considers this likely to be true also of Kenya. He suggests, however, that reinfection in a hypersensitive reactor, rather than a breakdown of a pre-existing lesion, may determine the onset of tuberculosis. The fact that a "primary" type of tuberculosis is so often seen in adult Africans, is, he says, explained by their lack of ability to form antibodies to the original infection.

Davies (1952) in Tanganyika also made a distinction between low-grade and high-grade reactions to tuberculin. All marked reactors to the test and all blister reactors were put into the high grade. All less marked reactors and all slightly positive reactors were put into the low grade. A low-grade reaction in a healthy-looking person might then, he said, be taken as an indication of that person either having a very slight tuberculous infection or being on the way to recovery from a more intense infection. A high-grade positive reaction, while it might be compatible with good general health, was an indication that somewhere in the body there existed a tuberculous lesion that was still at least potentially active. It was to be taken as a sign of instability.

Altogether 61% of the positive reactors were high-grade and 31% low-grade, but the proportion of low-grade reactors

increased as the age of 50 was approached. The article points out the danger of introducing the 20 - 39 age-group to industrial labour, and warns that a shift of all phases for the worse would be caused, with the precipitation of many more actual cases. The 61% of high-grade cases includes 4.5% of the population who were found to be diseased.

(It will be seen that Davies had a far larger proportion of his positive reactors in the "high-grade" class than the Tuberculosis Research Committee in South Africa, hence either his criteria were very different or his population had a much higher degree of allergy: the latter possibility may be to some extent true judging by his high morbidity figures.)

Griffiths and Kreher (1953), in Johannesburg, also found a qualitative difference in the reaction between their Bantu and European groups of children. The Bantu showed vesiculation in 10% of their positive reactors, and phlyctenular conjunctivitis occurred in one child. There is no vesiculation in any of the European children, and their reactions were uniformly smaller than those of the Bantu. These results show that even as children the Bantu react more strongly to tuberculin than Europeans. (Griffiths and Kreher also found a higher morbidity rate among the Bantu as compared with the Europeans, but made no effort to relate these two findings. It may, however, be noted that though some of the Europeans had active disease, not one gave a marked reaction to tuberculin, suggesting that the Bantu have the inherent tendency to develop a high degree of allergy when infected with the bacillus.)

On the other hand, Thompson (1955) working at Broadmoor Institution in England, reports that those who reacted severely had neither clinical nor radiological evidence of active tuberculosis.

Summary:

Though Thompson's findings may hold good for European peoples and perhaps others who have had contact with tuberculosis for a long time, several studies have indicated that in the African qualitative differences in the tuberculin reaction are significant. There is evidence that a higher degree of allergy is displayed by Africans than by Europeans indicating an inherent difference between the races. There is also evidence that Africans possessing this allergy in high degree are more likely to suffer later from tuberculosis than those having it only in low degree. There is disagreement as to whether disease developing later in highly allergic individuals is due to later reinfection, or whether the allergic state is due to the presence of latent tuberculous disease which later will break down into activity.

A field study on the significance of the nature of the reaction is described on page 100 .

V. MORBIDITY AND MORTALITY RATES.

A. Morbidity and mortality rates in Southern Rhodesia.

It will be recalled from an earlier section that as early as 1906 tuberculosis was looked upon as a serious problem among employed Africans.

The mortality from pulmonary tuberculosis for Africans and Coloured people together were recorded in the Public Health Reports from 1907 to 1929. There are in the population about 1,000 Africans to one Coloured (persons of mixed descent) These figures are shown in Table VI. They are exclusive of mine employees. They are not reliable, as many Africans die at home and the cause is not known. Another source of

difficulty is that as the years went on, probably more was known about cases occurring on the reserves, and this would account for an increase in the recorded deaths.

Table VI.

Average annual Non-mining African Death-Rates from Tuberculosis

<u>Years.</u>	<u>Deaths.</u>	<u>Deaths per 100,000 of estimated pop</u>
1907 -	19	2.7
1911 -	39	4.9
1916 -	58	7.0
1921 -	64	7.1
1926 - 9	79	8.0

It was thought that urban mortality statistics would be more reliable than those for the whole country, and in Table VII have therefore been shown the average annual cases and deaths recorded, for four-yearly periods, in Bulawayo from 1936-7 to 1951-2. (No figures were registered in 1939-40, 1940-41, or 1941-42, thus these three years have been left out of this calculation).

Table VII

Average annual tuberculosis cases and deaths in Bulawayo.
All types of tuberculosis. Residents and non-residents.

<u>Years.</u>	<u>Cases.</u>	<u>Deaths.</u>
1935-38	0.5	32
1942-45	11	46
1946-48	11	45
1949-51	84	40

The morbidity rates are completely unreliable. Table VIII shows that of recent years the notifications have

increased very rapidly, probably showing an increased efficiency of notification rather than an increase of the disease.

Table VIII.

Notifications of tuberculosis among Africans in Bulawayo.

<u>Year.</u>	<u>Notifications.</u>
1947-48	1
1948-49	23
1949-50	69
1950-51	85
1951-52	99

The mortality rates are probably fairly reliable in an urban area of this kind, and this is borne out by their relative constancy.

The population of Bulawayo is increasing rapidly. In 1950-51 the African population was 68,000. If we apply the 1949-51 cases and deaths to this, we obtain a mortality-rate of 0.59/1,000. The same process applied to the notifications gives a minimum morbidity-rate of 123,5/100,000.

As regards morbidity rates for the whole country, these are so unreliable as to be of no value. However, in his Public Health Report for 1953, Dr. Graham, the Regional M.O.H. of the Midland Region, Southern Rhodesia, reported that the notification rate here was 141 per 100,000, as compared with 212 per 100,000 in 1952 for Gateshead-on-Tyne, one of the poorest industrial areas in England. Allowing for the many cases that must escape

notification, it is plain that the disease must be well-established in the Midlands. There has also been a rapid increase of recent years, notifications for 1952 having been only 50 per 100,000.

Age and sex distribution: From 1921 to 1929 records were kept in the Public Health Reports of the sex of persons dying from disease, and whether they were over or under 5 years of age. The summated results for these 9 years in respect of tuberculosis are given in Table IX. African mine workers are excluded from the table. Again it must be borne in mind that many African children under 5, and women, may not have been notified as dying from tuberculosis, since they are more likely to stay in the kraal than the men.

Table IX.

Age and sex distribution of deaths from tuberculosis, 1921 to 1929. Totals as for these years.

Type of disease	African.						European.					
	Under 5		5 & over		Total.		Under 5		5 +		Total.	
	M	F	M	F	M	F	M	F	M	F	M	F
Pulmonary Tb	-	1	617	18	617	19	1	1	107	21	108	22
Miliary Tb	-	-	4	2	4	2	-	-	4	-	4	-
Tb.Meningitis	2	-	8	1	10	1	1	1	3	1	4	2
Abdominal Tb.	1	-	26	1	27	1	-	-	2	2	2	2
Pott's Disease	-	-	11	2	11	2	-	-	-	1	-	1
Tb.of other organs	-	-	12	-	12	-	-	-	2	-	2	-
Disseminated Tb.	1	-	9	1	10	1	-	-	1	1	1	1
Total	4	1	687	25	691	26	2	2	119	26	121	28

As might be expected, females have considerably lower rates than males. With the African, this is much more so: ratio of males: females : for Europeans, 81% : 19%; for Africans, 96% : 4%.

B. Morbidity and mortality rates in the Bantu of other countries

1. Union of South Africa. Allan (1932) observed the following rates in the Native Areas:

In the Southern Transkei (with a high tuberculin sensitivity rate), although no statistics were available, the local medical practitioners were agreed that about 9% of Natives consulting them had tuberculosis. Allan estimated that there were 450 cases diagnosed in a year in a population of 23,000 (2.0% of the population annually).

In the Northern Transkei, which had a relatively low sensitivity rate, tuberculosis did not occur frequently, and the cases seen were of a mild type.

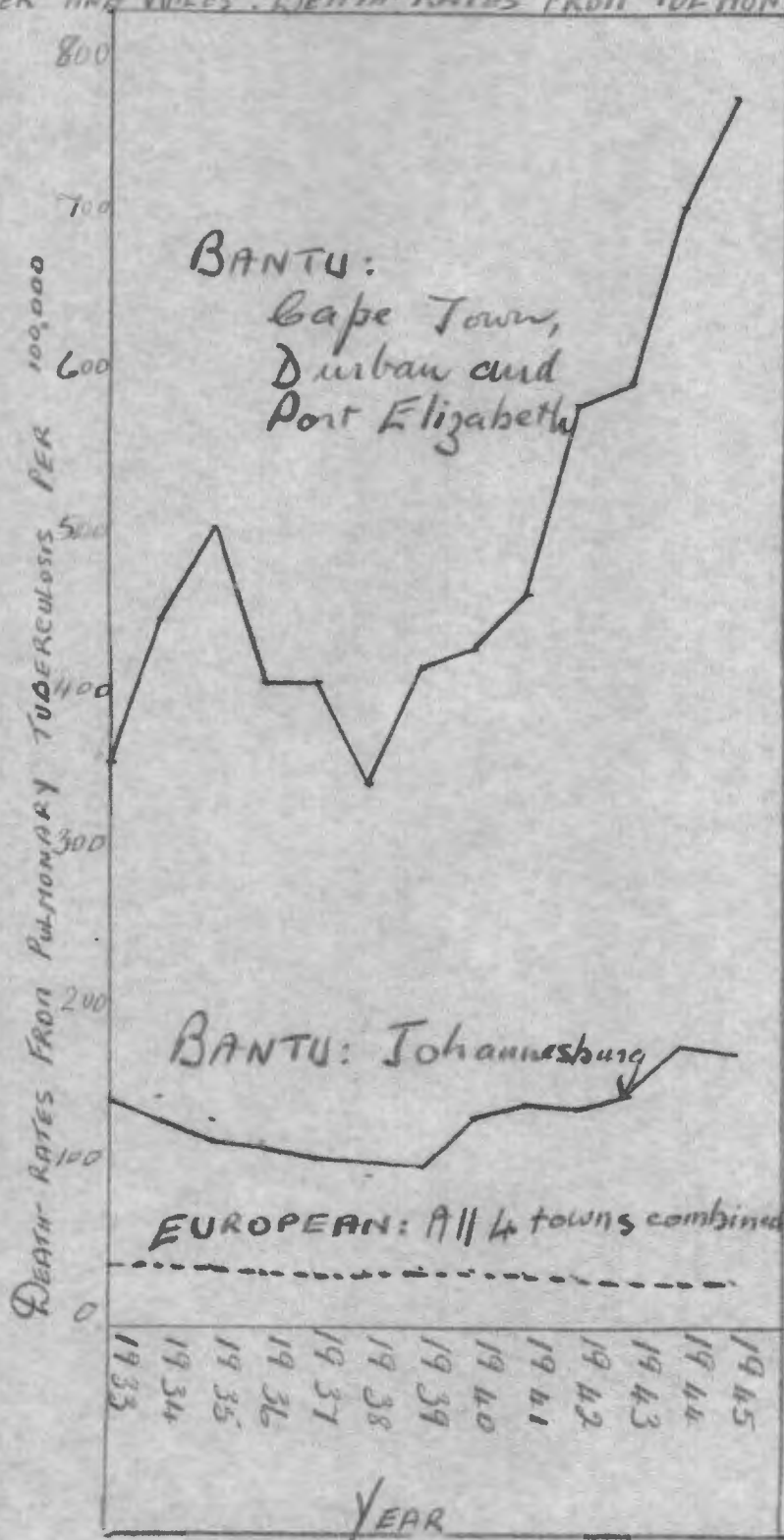
In Basutoland, with a similarly low rate, tuberculosis was found to be comparatively rare. The Principal Medical Officer of Basutoland is quoted as saying that it had only been introduced in the previous 40 years or so, by mine boys returning from work.

In the Ciskei Allan collected figures which showed that between the ages of 5 and 15 the incidence of tuberculosis is greater in females than in males.

Dormer and Wiles (1946) quote municipal health departments mortality figures for recent years as the most accurate available (Fig.1) It will be seen that urban mortality figures are very high. According to the authors, the

FIGURE 1.

DORMER and WILES: DEATH-RATES FROM PULMONARY TUBERCULOSIS



comparatively low Johannesburg rate is due partly to the fact that mine Natives are repatriated as soon as the diagnosis is made if they are fit to travel; but making a calculation to allow for this they still estimated that the death-rate for 1942 would be 252 per 100,000 or less than half that for the other municipalities.

Two reasons for this difference are offered. Firstly, "Natives recruited for the mines are medically examined before leaving home, and X-rayed or again examined on arrival at the Witwatersrand, thus eliminating those medically unfit. In 1942, for example, of 89,470 recruits from the Transkei and Ciskei, 75 were rejected because they had pulmonary tuberculosis. The second factor is that mining Natives have proper diet scales laid down and are much better fed than the average industrial labourer".

As stated above, among children tuberculin-tested by Griffiths and Kreher (1953), 26% of the Africans and 10% of the Europeans gave positive tuberculin reactions. Of the positive reactors, 129 Africans and 33 Europeans were X-rayed, giving the following results (glands or definite infiltration were regarded as "activity"):

	<u>Calcification.</u>	<u>Activity.</u>
African	13%	23%
European	24%	12%

(If this 129 and this 33 were random samples of all the positives, and if we regard "activity" as being disease, this gives the following prevalence rates per 1,000:

African	60	European	12)
---------	----	----------	-----

These figures lead the authors to conclude that the Bantu children appear to cope with primary tuberculosis infection adequately, but that the immunity produced by such a primary infection is of doubtful and inconstant value for the individual child. (It seems questionable whether the first conclusion is justified, since the Bantu children had a much higher rate of "activity" and lower rate of "calcification" than the Europeans).

2. Kenya. Haynes (1951) notes that the incidence of the disease has increased during the present century and is still on the increase. In the survey incidence rates were determined for three provinces:

- Nyanza Province: 9.65 cases per 1,000
- Coast Province: 10.9 do
- Central Province: 12.3 do

Clack (1951) in Kenya, estimates the incidence of known cases at 4.47 per 1,000 of the population per annum, but he thinks the true incidence must be considerably higher.

3. Tanganyika. Davies (1952) found that 4.5% of the population of the Mbulu district of Tanganyika were suffering from tuberculosis. (Overall tuberculin sensitivity rate in those tested was 47%). The 4.5% were made up as follows:

- "Early and symptomless disease": 2.2%
- "Disease with symptoms": 2.3%
- (including 0.5% "infectious disease").

Conclusions:
The morbidity and mortality rates observed in the various parts of Africa are very unreliable.

1. Morbidity.

The Bulawayo notifications are of no value, except to indicate a minimum rate of 123/100,000 which is almost certainly far below the truth. Graham's notification figure for his non-urban region, of 141/100,000 is however high enough to indicate that tuberculosis is well established in the rural areas of the Midlands, and therefore probably of the country as a whole.

For other countries, figures for annual incidence, per 1,000, vary from .47 (or more) in the Fort Hall Reserve, Kenya (Clark, 1951) to 45 in Mbulu, Tanganyika (Davies, 1952). Other rates estimated include about 10 for parts of Kenya (Haynes, 1951) and 20 for the Transkei (Allan, 1952).

All that can be concluded from these figures is that, while reliable figures are not available, Southern Rhodesia probably has as much tuberculous disease as the other African territories, and that, as in them, it has reached the dimensions of a grave problem.

2. Mortality.

Mortality statistics for Southern Rhodesia as a whole are entirely unreliable, besides the fact that they have been discontinued since 1929.

In Bulawayo, over the years 1946-49, the rate was .59/1,000, a figure which is probably not grossly inaccurate, though allowance must be made for some who will have gone home upon finding that they were ill. The rate compares favourably with those recorded by Dormer and Wiles (1946) for Cape Town and Port Elizabeth (5 - 6/1,000), and their somewhat lower

figures for Johannesburg (2.5/1,000) which are due to the careful selection of miners and better conditions on mines than in industry generally.

VI. THE EPIDEMIOLOGY OF TUBERCULOSIS.

A. Spread in isolated communities.

The Tuberculosis Research Committee, 1932, continuing their report on tuberculosis in isolated communities (page 14) gave their opinion that "the return of individual cases infected elsewhere to such communities does not invariably or even usually light up epidemic tuberculosis. Provided that the community still retains its isolated character and continues to follow its primitive mode of life, the spread of the disease when thus introduced may, it seems, be arrested or only proceed very slowly". This may be due to the tendency to spontaneous arrest of cases in the glandular stage, as noted by Borrel, when placed at rest and suitably dieted. If more cases are introduced from time to time, a type of benign endemic tuberculosis may be established in the community.

"A type of contact more frequently observed is that in which, under conditions of segregation, a previously isolated community is placed in unfamiliar surroundings and provided with "civilized" substitutes for its primitive foods, habitations, customs and manners. In these circumstances each member of the community, if infected with tuberculosis, tends to behave just as has been described above for the Senegalese troops of Borrel, and the result is an approximation to epidemic rather than to endemic tuberculosis". Such an

epidemic described by Ferguson as occurring in Canadian Indians is quoted.

When "civilization" penetrates gradually into the heart of a previously isolated community, the issue tends to be more obscure; but when the penetration leads to or is accompanied by a marked change in the diet and mode of life of the community, the results may be very similar to those described by Ferguson.

Thus members of isolated communities exhibit a marked susceptibility to tuberculosis when brought into contact with infection; and although the disease may be fairly well tolerated by the community under natural conditions, this susceptibility is very dangerous when there is at the same time a sudden change in occupation, food, housing, and mode of life.

B. The Epidemiology of tuberculosis in certain Native Areas.

The work of Allan and the Tuberculosis Research Committee (1932) strongly suggests that tuberculosis has become highly endemic in the coastal parts of the Native Areas of South Africa due to its introduction from the pool of infection on the Rand. However, in his field survey, Allan found that at the present time many of the cases had no connection with industrial employment. Though family infection was noticed in several cases, in the majority no infecting member could be traced. The cases were found scattered throughout the district, and were not confined particularly to either sophisticated or "raw" Natives. Moreover, though the widespread distribution of the disease made it hard to judge, it appeared to Allan

that there was little spread due to returned mine boys. In addition, MacVicar (1907) is quoted as having observed that "in spite of conditions which seem so favourable to its spread, the tubercular infection, when introduced into a family, does not as a rule spread rapidly from person to person. In some families, while some members suffer, others escape, regardless, it would seem, of age, and in those who are attacked, the period of incubation is long - two years and even longer".

Allan suggests the following reasons for the non-spread of tuberculosis by returned miners:

1. The more acute cases die very soon after their return home.
2. The Natives at home sit outside in the sunshine as much as possible when ill.
3. Certain hygienic habits among these Africans are mentioned which prevent the spread of infection.

For comparison with tuberculosis as seen among the Natives in their natural surroundings, an account is given of tuberculosis as occurring among Africans living in urban conditions. The state of affairs is not materially different in the two situations.

In Kenya, Clark (1951) notes that the constant migration to and from Nairobi of young workers appears to be important in the dissemination of tuberculosis in the reserves. In the same country, Haynes (1951) considers that the deficiency of first-class protein in the African diet may have an important bearing on the tuberculosis problem. However, he quotes the frequency and severity of tuberculosis found in Africans
outside Africa

as showing that the dietetic factor is less important than racial susceptibility. (No mention is made of physical stress and unfamiliar environment).

The view of Haynes (page 31), that the coastal parts are more infected because they are the routes of ingress of disease-bearing immigrants, may hold good for East Africa but is not tenable for South Africa since the Native Territories havenot been the route of ingress of immigrants.

Among industrial workers, the Tuberculosis Research Committee in South Africa laid importance on the sudden commencement of strenuous work, the unaccustomed food, the absence of assistance of their women-folk, and the psychological trauma of being away from home, as important factors in causing the breakdown of an existing focus or the development of the fresh disease.

A point made by Allan and the Tuberculosis Research Committee deserves special note. The incidence of tuberculosis among mine workers who had come from the areas of low endemicity was higher than among those from the areas of high endemicity (about 12/1,000 against about 8.8/1,000); on the other hand, in those individuals who were tuberculin-tested on commencing work on the mines, the incidence of tuberculosis occurring later was $2\frac{1}{2}$ times as great in those who were positive than in those who were negative. The Committee conclude that tuberculosis must often be due to the breakdown of a previous infection acquired at home, or at least to the allergic state caused by one. They do not, however, suggest a reason for the

relatively high incidence in those from the less tuberculized areas. We are not told whether these cases occurred in the negative or the positive reactors; but in either event it may be concluded that some form of immunity, apart from allergy and the persistence of latent infection, must be built up amongst the individuals of the highly tuberculized community.

C. General rules governing the spread of tuberculosis in African territories.

The distribution of infection and of disease is a large and rather complex subject. In general three kinds of area may be defined.

(1) Urban areas, particularly industrial centres, have a high endemicity coupled with a high disease rate, due to the presence of plentiful opportunity for infection combined with the low resistance associated with unsatisfactory and unaccustomed conditions of living. The high urban mortality figures given by Dormer and Wiles (1946) (page 42) show how serious the disease is in large towns. The findings of Dormer and Wiles also suggest that mining, per se, is not as important in the production of tuberculosis as has been thought; those in industries other than mining are hit much harder by the disease. Here we see the importance of adequate provision for feeding and housing the worker, and silica dust may be of relatively little importance.

The work of Gampel, 1952, (page 30) and that of Griffiths and Kreher, 1953, (page 30) show that in urban areas

tuberculization of children starts at an early age and reaches high levels early in life, as compared with European children in Johannesburg or children in England. This difference can reasonably be put down to the conditions in which the African children live, which predispose to the ready spread of infection and to the higher incidence of infectious cases among African adults.

(2) Rural areas which have had little contact with the disease have a low endemicity, and when the disease occurs its spread depends on the concentration of the population, and the number of manifest cases depends on their state of nutrition and living conditions. (This type of area is considered in conjunction with (3) below).

3) Rural areas with much migration to and from industrial centres have a high endemicity with a disease-rate again depending on the standard of living.

Thus, as seen from Peter Allan's work (page 27) where the kraal conditions are satisfactory, with adequate nutrition, and the country is not over-populated, tuberculosis is not a serious problem even when it has become relatively endemic. Such regions are Basutoland and the Matati ele area. In overcrowded areas where malnutrition is common, tuberculosis when introduced has a high incidence. For example, to take two areas of high prevalence, the Transkei and parts of Kenya both suffer from poor nutrition, though in the former the tuberculin-sensitivity rate is high, and in the latter, low.

We may roughly piece together the development of tuberculosis in a newly-infected African country as follows. At first the rural areas are fairly free of the disease. As labour is recruited from them, some of the workers fall prey to it, and owing to lack of immunity coupled with unfavourable conditions, develop a severe and fatal form. Before they die, however, many are repatriated, and their contacts develop it also. In some areas these secondary cases appear to suffer the same type of severe disease, as in Uganda; but in most, infection appears to pass almost imperceptibly from one to the other, the amount of active disease depending on the conditions of living. This process continues until the majority of the population have received an infection, and a large proportion of the recruits for industry are tuberculin-positive. These are then even more likely than their negative associates to develop the acute severe disease, ^{possibly} ~~probably~~ due to a resurgence of the original infection which had been lying latent.

Some stage in this process is the sort of picture which we may expect to find in Southern Rhodesia when our investigations are completed.

D. The epidemiological situation in Southern Rhodesia as far as we know it.

The fact that there have been increasing numbers of cases of tuberculosis occurring in the Reserves, and Graham's incidence figure of 141/100,000 for the Midland region, indicate that the disease has a fairly firm hold on rural

Southern Rhodesia. The surveys on school children show, furthermore, that a fair degree of endemicity has been reached.

In Southern Rhodesia and the adjacent regions, tuberculosis appears to have reached a stage where infection is acquired in the kraals, and breakdown occurs mainly in industrial employment. The reasons for this conclusion are as follows:

- (1) The tuberculin tests in the Western, Midlands and Northern Regions have shown clearly that in rural Southern Rhodesia conversion to tuberculin-positivity starts early in life, and by the age of about 17 reaches a fairly high level.
- (2) An investigation will be described (page 89) showing that sensitivity rates increase very little with duration of industrial employment, but do increase with increasing age of employees.

VII. NON-PULMONARY TUBERCULOSIS.

A. Incidence of non-pulmonary tuberculosis in Southern Rhodesia.

The different types of tuberculosis causing death are listed according to the international classification in the Public Health Reports from 1913 to 1929. The totals for these years are given in Table X. They will be some guide to the proportions of different types occurring. The possible sources of error are: that non-pulmonary types may be more common in children, and thus occur more in the reserves and

not be certified as tuberculosis; on the other hand, urban life, which yields a greater proportion of recorded deaths, may cause more acute and disseminated disease.

Table X.

Types of tuberculosis causing death in S.R., 1913 - 1929.

<u>Type of disease.</u>	<u>African.</u>	<u>European.</u>
Pulmonary tuberculosis	1039	248
Acute miliary tuberculosis	9	6
Tuberculous meningitis	13	4
Abdominal tuberculosis	30	5
Pott's Disease	18	1
Tuberculosis of other organs	17	3
Disseminated tuberculosis	22	8
Total	1148	275

The proportion of pulmonary to non-pulmonary cases was therefore, for Africans, 90%:10%, and for Europeans, 90%:10%. The Africans show a fairly high incidence of Pott's Disease.

In England and Wales the proportion of pulmonary to non-pulmonary cases, in 1931, was 82.8%:17.2%; and in 1951, it was 87.2%:12.8%

B. Non-pulmonary tuberculosis among the Bantu of other countries.

McVicar (1935) found that 20% of admissions to his hospital in the Ciskei (South Africa) over 8 years had been for bone and joint disease. With reasonable accuracy he was able to calculate for one region an incidence rate of .53

per 1,000 for bone and joint tuberculosis.

His experience, and that of Allan, and of Girdwood, all reported to the Tuberculosis Research Committee (1932), was that glandular tuberculosis, though it tends to suppurate, does not usually develop into pulmonary or generalized tuberculosis, but goes on to recovery with scarring. Such cases are accepted without harm for work on the Rand gold-fields. The reaction to local glandular tuberculosis appears to be the same as that of the European child.

Allan (1932) found that in the Transkei pulmonary tuberculosis is by far the commonest form; in one practice there were 298 cases of tuberculosis of which 272 were pulmonary, 14 glandular, and 12 spinal. Miliary tuberculosis does occur, and also bone tuberculosis (mainly spinal), and lupus. Pott's disease is fairly common among adults as well as children.

As regards the Ciskei, Dr. McVicar is quoted as having found that pulmonary tuberculosis was by far the most common form, and that next in frequency came infection of the cervical glands. Abdominal tuberculosis he found rarely.

Allan found that of 3,000 consecutive admissions to hospital in 1927, there were 74 cases of tuberculosis divided as follows:

Pulmonary	55	Tuberculous glands	5
Spinal	5	Abdominal	3
Other forms, not specified	5	Genito-urinary	1

Haynes (1951) found in Kenya 71.8% pulmonary and 21.2% non-pulmonary cases.

Kaplan (1952) found in South Africa that Pott's disease in Bantu children occurred more in the cervico-dorsal and lumbo-dorsal regions, and less in the dorsal and lumbar regions, than in a comparable series of children in Lancashire. The primary focus in the vertebral body was the same. The disease was generally more extensive. The immediate recovery rate compared favourably with the Lancashire series.

C. Infection by the bovine bacillus.

In Southern Rhodesia, typing of tubercle bacilli is not often done, and no bovine bacilli have been found. There has however, been a recent increase in the amount of non-pulmonary tuberculosis among Africans, and the Secretary for Health, in his 1951 report, stated that the subject of bovine disease deserved review.

Haynes (1951) reviews the literature on bovine bacillus infection in Africa. Most observers, he says, have found little evidence of it. 4 cases out of 297 tested in Uganda had the bovine bacillus. One case has been found in the Union of South Africa.

According to Davies (1952) no case of non-pulmonary tuberculosis, judged by numerous specimens both from biopsies and autopsies, has in Tanganyika been due to the bovine bacillus.

Summary:

In general non-pulmonary tuberculosis in Africa appears to provide about the same proportion of cases as in the United Kingdom. Pott's disease is relatively more common than other forms and has a different distribution in the spine.

Bovine bacillus infection is probably not important in Southern Rhodesia, but has been found from time to time in other parts of Africa and should not be forgotten.

VIII. TUBERCULOSIS ON MINES.A. In Southern Rhodesia.1. Morbidity and mortality statistics for phthisis on mines.

These are summarized in Table XI

Table XI.

Morbidity and mortality from tuberculosis among African mine workers in Southern Rhodesia.

<u>Years.</u>	<u>Av. total No. of Employees.</u>	<u>Av. No. of cases p.a.</u>	<u>Cases /1000 empl.</u>	<u>Average deaths p.a.</u>	<u>Deaths /1000 empl.</u>	<u>Case fatality per cent.</u>
1907-	31,978	185	5.78	78	2.44	42
1911-	36,608	135	3.69	60	1.64	44
1916-	36,022	173	4.80	49	1.36	28
1921-	38,455	88	2.28	31	.81	35
1926-	44,115	78	1.77	45	1.02	58
1931-	50,906	57	1.12	39	.77	68
1936-	86,375	54	.63	46	.53	85
1941-	78,238	Not given		59	.75	
1946-49	65,513	84	1.28	59	.90	70

The individual years in the last group had these rates:

Years.	Av. total no. of employees.	Av. No. of cases p.a.	Cases /1000 empl.	Average deaths p.a.	Deaths /1000 empl.	Case Fatality per cent.
1946	70,545	83	1.18	56	.79	68
1947	69,712	96	1.38	66	.95	69
1948	63,794	77	1.21	52	.82	68
1949	58,000	79	1.36	62	1.05	77

The mortality statistics from the mines, like those of the general population, have severe limitations. Africans found to have the disease on mines, if certified fit to travel by a medical officer, are sent home after an interval of some months required to complete the necessary formalities. Others, too ill to travel home, are treated in Government hospitals and their death is thus not reported by the mine. Mine morbidity figures, though the only ones which can lay any claim to reliability, have the drawback that often cases only report sick, or are discovered, in the terminal stages; and also with the rapid turnover of labour many may contract the disease and leave the mine before it becomes evident.

The table shows a general reduction in the morbidity-rate up to the 1936-40 period, which may be accepted as real, since the tendency has been towards improved medical services on mines, and therefore better diagnosis. After the war period, in which morbidity was not recorded, the cases were at a constantly higher level than before it.

The mortality rates show a similar but less marked decrease up to 1936-40, and since then have risen steadily. Some comparable mortality rates for England and Wales are

shown in Table XII. Though these seem to compare unfavourably, there must be many ~~repatriated~~ cases in Southern Rhodesia who die after returning home.

Table XII.

Annual deaths from respiratory tuberculosis per 1,000 miners aged 25 - 60 in England and Wales, 1930-32.

Coal miners:	.22
Metaliferous miners (below ground)	1.49
Stone miners and quarriers	1.11

A better comparison is with the morbidity and mortality rates of the Rand mines, some of which are given in Table XIII.

Table XIII.

Morbidity and mortality rates in the Central Mining member of the Rand Mines Group. Tuberculosis.

<u>Year.</u> (in reverse order)	<u>Morbidity.</u> per 1000	<u>Mortality</u> per 1000
1949	2.52	0.71
1948	2.53	0.62
1947	2.39	0.55
1946	2.26	0.60
1945	2.11	0.57

The mortality rates can scarcely be compared, as there is no measure of the proportion of ^{sufferers returned home} ~~repatriated~~ in either group. The morbidity rates are higher than those for Southern Rhodesia; it is not possible to say to what extent this is due to better case-finding methods on the Rand.

The case fatality rates of the S. Rhodesian miners have shown a marked increase since about 1920. This is the reverse of what one would expect with improved diagnostic methods. It has been the practice since the earliest days for cases diagnosed as tuberculosis and able to travel to be ^{returned home.} ~~repatriated at once.~~ It is possible that with improved mine hospitals more cases have been kept at these for treatment, and have thus died while still on the mine.

For the three years 1926 - 29, the deaths from various diseases among mine workers from different countries was recorded. Table XIV gives these data for tuberculosis. These results are consistent, and suggest strongly that those who leave their own country to work in Southern Rhodesia are much more liable to die from tuberculosis than are Southern Rhodesian Natives. The worst sufferers are those from Portuguese East Africa, then those from Northern Rhodesia and Nyasaland. There are many possible reasons for these differences; among them may be the degree of "tuberculization" of the community from which they come, the state of nutrition in that country, and the physical and psychological effect of being away from home.

TABLE XIV.

Deaths from tuberculosis among mine workers
by countries of origin.

Country	1927		1928		1928	
	No. empl.	Deaths DR/1000.	No. empl.	Deaths. DR/1000.	No. empl.	Deaths. DR/1000
S. Rhodesia	12,062	4 .33	12,669	5 .39	12,645	5 .40
Portuguese E.A.	3,704	6 1.62	3,741	7 1.97	4,055	8 1.97
N. Rhodesia	12,422	18 1.45	12,555	16 1.27	13,875	13 .94
Nyasaland	13,321	13 .98	14,015	17 1.21	15,156	17 1.12
Other	537	3 5.6	723	1 1.4	1,080	1 .9

However, industrial workers from Portuguese East Africa have been shown (page 90) to have a higher sensitivity rate than those from Southern Rhodesia. Those from Nyasaland may have suffered in nutrition and standard of living from the high concentration of the population there. None the less, the effect of being away from home still seems to be a very important factor.

A similar gradient is shown by the mortality from all diseases and accidents among mine workers from these different countries. The figures for accidents are in general proportionate to those for disease. Here Nyasaland miners were the worst sufferers, followed by Northern Rhodesia, then Portuguese East Africa.

2. Report of the Silicosis Commission, 1938.

In a preliminary enquiry, Dr. Martin, the Medical Director of Southern Rhodesia, had obtained from various reports and returns the following results:

(1) Five European and sixteen African miners who had worked solely on Southern Rhodesian mines were found radiologically to have silicosis, thus confirming the suspicion already held that cases of silicosis were being produced in these mines.

(2) The incidence of pulmonary tuberculosis, whether accompanied by silicosis or not, was very considerable

amongst Natives employed in mining and appeared to be in excess of that found in the non-mining population.

He called attention to the danger of the disease spreading to the kraals, both in Southern Rhodesia and neighbouring territories.

The causation of silicosis:

The Commission points out the principal phases of mining which produce a high concentration of silica dust in the air and hence lead to the greatest amount of silicosis. They are in order of importance.

(1) Blasting produces the heaviest concentrations, and the concentration is highest after blasting in development ends. After blasting, the air also contains dangerous quantities of carbon monoxide and of highly irritant nitrous fumes. Repeated inhalation of such nitrous fumes tends to accelerate the production of silicosis.

(2) Drilling, particularly machine-drilling.

(3) Handling and transport of broken rock, to a less extent.

H.S. Paterson, on the Witwatersrand, is quoted as putting the order of danger in underground proceedings as (a) Machine-drilling (b) Machine-stopping (c) tramming (d) lashing. In general machining is much more dangerous than hand operations.

With the gradual reduction in the concentration of dust in mine air, which has been effected in the course of time,

silicosis has assumed a progressively less serious, more slowly developing, and more chronic type, and its incidence rate has been very greatly reduced.

General characteristics of silicosis:

The cardinal symptom of silicosis is dyspnoea. In the marked cases there is chronic emphysema and dry bronchitis. The radiographic appearance is of a more or less symmetrically distributed nodular fibrosis in the lung substance and under the overlying pleura. There is always a similar fibrosis of the root glands, which are in fact commonly the first areas to show obvious fibrosis; and there is some accompanying bronchitis and in established cases varying degrees of emphysema.

The relation of silicosis to tuberculosis:

The majority of cases that die as a result of silicosis have tuberculosis, because the lung becomes predisposed to it. Three divisions of silicotic disease may be made:

- (1) simple silicosis, as above.
- (2) tuberculo-silicosis: with super-added tuberculosis the fibrous process continues, and the infection may become arrested for long periods. Here unmodified active tuberculosis is only seen as a terminal phenomenon.

(3) Tuberculosis with silicosis: on the other hand, active tuberculosis may supervene on silicosis, and this is common in Native mine labourers, but uncommon in Europeans.

It may result from:

- (a) breakdown of previously existing areas of tuberculo-silicosis.

(b) another latent focus of tuberculosis in the respiratory organs.

(c) external infection.

These variations depend on the relative prominence of:

(1) the dust factor, in respect of concentration, fineness and duration of exposure; and

(a) the infective factor, in respect of susceptibility and opportunity of exposure to tuberculous infection.

The prevalence of silicosis and tuberculosis on the gold mines of Southern Rhodesia:

At the time of the investigation it was very rare to find European cases of silicosis who also had tuberculosis at the first discovery; but in Africans it was found in 40% of silicotics (65% ten years earlier). A larger number still of Africans on mines had uncomplicated tuberculosis.

Prevalence of silicosis and tuberculosis on 13 selected gold mines:

A. Europeans:

In 167 miners who were a fair sample of men who had worked only in Rhodesian mines, and of whom radiographs were taken:

- (1) 77% showed no abnormality.
- (2) 14% showed "abnormal increase in striation".
- (3) 9% showed definite silicosis.
- (4) There was no case of silicosis complicated by tuberculosis.

In group (3) the duration of service was as follows:

Under 5 years: No cases.
 5 - 9 years: 1 case (2% of that service group)
 10 - 14 years: 5 cases (18% of that service group)
 Over 15 years: 9 cases (50% of that service group).

The Committee concluded that "(European) miners who have been certified to have contracted an uncomplicated silicosis elsewhere may be permitted to work in the mines of Southern Rhodesia, provided that they submit themselves to medical examination at prescribed intervals in order that the supervention of active tuberculosis, should that occur, may be detected and notified".

B. Africans.

The numbers of Africans examined were as follows:

<u>Length of Service.</u>	<u>Total Complement.</u>	<u>No. examined.</u>	<u>% examined.</u>
Under 5 years	5,718	221	4
5 - 9 years	1,196	503	42
10 - 14 years	485	260	54
15 - 19 years	244	159	65
20 years and over	104	88	85

The first group is made up mainly of casual workers, and consists predominantly of those in the first two years of service.

All these 1231 Africans were radiographed, and the results are shown in Table XV and Fig. 2.

1. Silicosis. The diagram shows a sharp decline in the number of cases showing "normal" radiographs from the

Table XV.

Thirteen selected gold mines: observed prevalence of silicosis and other specified conditions amongst Native mine labourers in the several periods of underground service stated.

Years under ground service.	1. Radiographs within normal limits.		2. Radiographs indicative of abnormal increase in striation.		3. Radiographs indicative of definite silicosis. Without active tuberculo- silicosis.		Total silicosis.		Radiographs indicative of simple tuberculosis	
	No.	% of group	No.	% of group.	No.	% of group	No.	% of group	No.	% of group.
Up to 5	214	(97)	4	(2)	-	-	-	-	3	-
5 -	449	(89)	47	(9)	5	(1)	-	5	2	(1)
10 -	195	(75)	38	(15)	21	(8)	4	25	2	(10)
15 -	88	(55)	49	(31)	19	(12)	2	21	1	(13)
Over 20	46	(54)	17	(20)	19	(22)	3	22	-	(26)
ALL periods 1, 128	992	-	155	-	64	-	9	73	8	-
ALL periods of 5 years and over 1, 007	778	(77)	151	(15)	64	(6)	9	73	5	(7)

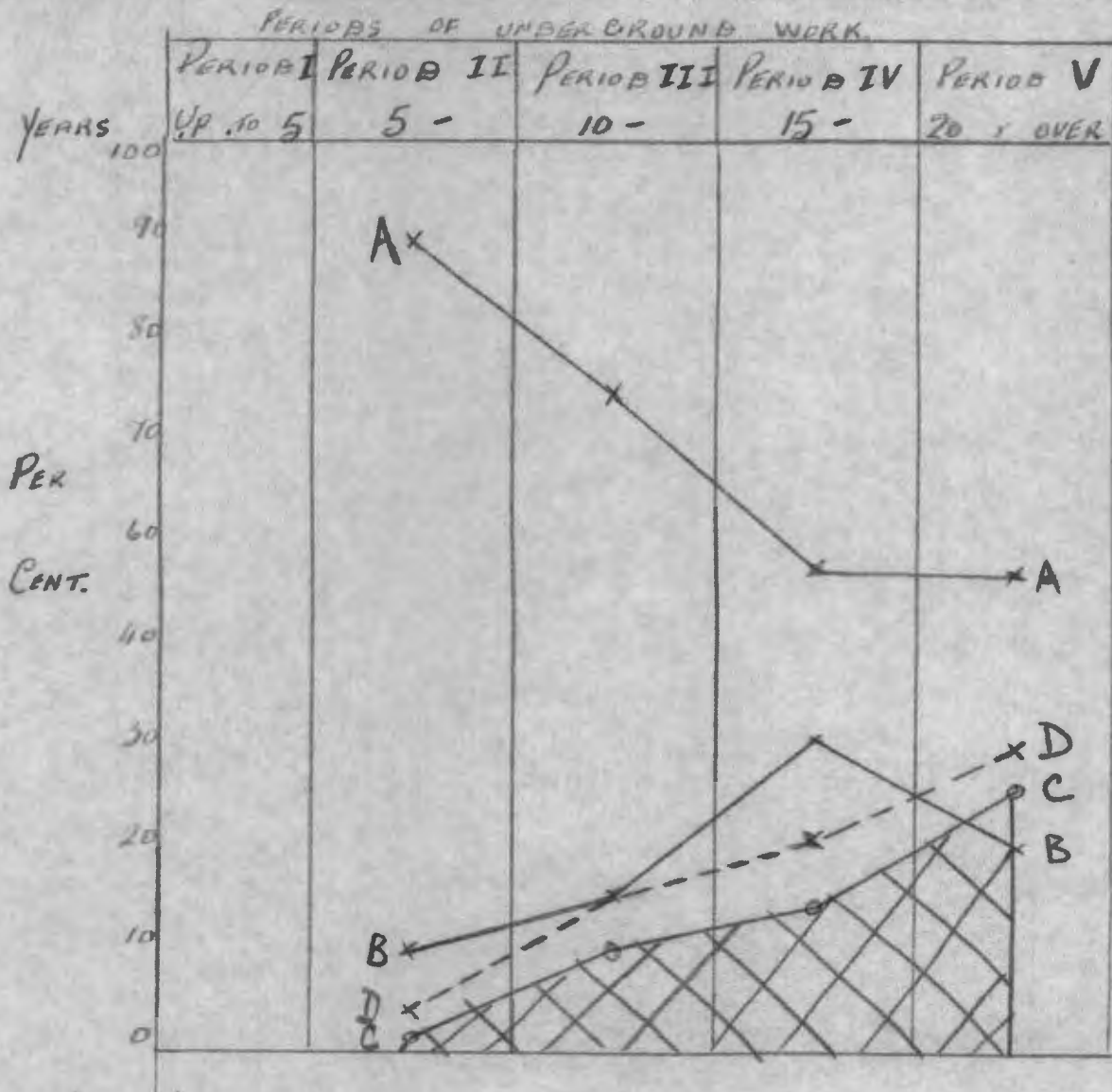
(Percentages have been put in brackets).

FIGURE 2

SILICOSIS COMMISSION: 13 SELECTED GOLD MINES.

NATIVE MINE LABOURERS.

PREVALENCE RATES PER CENT. OF SILICOSIS ETC. AT SUCCESSIVE



The graphs show the percentage prevalence at successive periods of underground service of:

- A. Cases whose radiographs were within normal limits.
- B. Cases whose radiographs showed "abnormal increase in linear striation".
- C. Cases whose radiographs showed appearance of definite silicosis.
- D. C plus B, multiplied by 0.3, to indicate the steady increase of the two taken together, suggesting that abnormal in striation later develops into true silicosis.

second to the fourth 5-yearly service periods. Thereafter there is no correspondingly significant decrease, suggesting that the remaining "normals" in Period 5 represent cases which are more or less immune to respiratory infection.

The number of cases of "abnormal increase in striation" falls from the 15 - 20 to the 20-plus service group, but the corresponding rise in cases of "definite silicosis" suggests that there was conversion from the former into the latter.

The authors conclude that the respective amounts of increase in the prevalence rate of "abnormal increase in striation" may be a measure of the respective risk of ultimate liability to the contraction of silicosis or tuberculosis, produced by the occupational conditions existing on different mines. The low proportion of appearances of this type met with in the non-silicious shallow Wankie coal mines, strongly supports this view.

The general conclusion reached is that among both Europeans and Natives, the risk of contracting silicosis appears to begin in the latter part of the second five-yearly period of service and to increase steadily thereafter.

These observations apply primarily to the large type of mine employing over three hundred Natives, of which the thirteen selected gold mines are representative. In the numerous small-sized properties the main risk is blasting

rather than drilling, but they too need consideration in formulating a system of preventive measures.

2. Tuberculosis. From the total number of observed active cases in this survey, i.e. 9 with silicosis and 5 apparently uncomplicated, the authors estimate that there is a prevalence rate of the disease of 7.5 per 1000, and assuming that the average duration of African cases does not exceed 18 months, this gives an annual production rate of not less than 5 - 6/1000.

Only 2 of these 14 cases had previously been diagnosed.

It is pointed out that the "Health and mortality returns" from the mines published annually in the Public Health Reports are probably based on cases in an advanced state, as illustrated by the high death-rate shown, and give a false idea of the morbidity-rate from tuberculosis on the mines. The need is stressed for securing that specific measures be prescribed by regulation for the detection and notification of cases of active tuberculosis amongst mine Natives.

The Committee considered that on the whole the working hours, food and housing on the mines were satisfactory.

Asbestos mines:

Asbestosis is more severe and more fatal than silicosis. As with silicosis, progress is slow; it usually takes more than 7 years mining to produce a serious degree.

The two asbestos mines studied showed a smaller number of dust particles underground and in the mills than the

gold mines. There were more in the crushing and grading mills, mainly small (and therefore dangerous).

In the asbestos mines, among 34 Europeans and 250 Africans distributed over all the service groups used above (mainly) 5 - 10 and 10 - 15 years' service) there was no case of developed asbestosis, but 12 (all African) men had "increased striation". 2 possible - not definite - African cases of tuberculosis were found.

Wankie Coal Mine:

The conclusion was reached that this was not productive of silicosis.

B. Tuberculosis on mines in the Union of South Africa.

The Tuberculosis Research Committee, 1932, gauged the incidence of tuberculosis on mines, and considered that it could be fairly accurately discovered from the records, as the mine-workers were under regular medical supervision. The rates were, however, stated to be under-estimates, as they took no account of cases which might be expected to occur later amongst Natives who had only developed the disease after return to their kraals. The mortality figures were much less reliable owing to repatriation of cases fit to travel. The incidence was as follows :

<u>Year</u>	<u>All types, per 1000.</u>	<u>Respiratory, per 1000.</u>
1926 - 7	6.81	5.82
1927 - 8	7.53	6.68
1928 - 9	6.62	4.93

The Committee concluded that although the incidence rate was not much higher than might have been expected, the case-fatality rate in Native mine workers is very high. (It should be noted that these figures are much higher than those given in an earlier section for 1945 - 9 (see page 59).

A comparison was then made between Native and European miners' morbidity- and mortality-rates, using Allan's follow-up of repatriated miners, described in the next paragraph. Native case incidence was about twice that of Europeans, and the case-fatality rate of Natives in the first year was estimated at 59%, or twice that of Europeans.

Dr. Allan followed up a series of repatriated mine workers in the Native Territories. Out of 694 boys repatriated from the mines with tuberculosis during the years 1926 - 9 the fate of 475 was ascertained. Two years after repatriation 60% had died, 10% were alive but unfit for any work, 26% were alive and fit for light work at their homes, and 4% were alive and fit to return to heavy work.

:As a corollary to this, the Tuberculosis Research Committee found from the records that in the three years 1926 - 7, 1927 - 8, and 1928 - 9, 722 cases of tuberculosis died in the mine hospitals, while 3389 cases were repatriated. This means that if 60% of these 3389 died within 2 years of repatriation, there were 2033 such deaths for 722 on the

mines, or 2.8 deaths for every one recorded on the mines.

Possible factors in the production of tuberculosis

on the mines: 1. Conditions in Native areas. The Committee noted the conditions of malnutrition prevalent in the Native Areas of the Cape Province, and contrasted these with the apparent plenty in Portuguese East Africa. They quoted Pirie and Navrogordato, who had found that out of 263 autopsies on Portuguese East African Natives, 56% were "acute lesions", and 44% "chronic tuberculosis associated with silicosis"; whereas of 274 autopsies on British South African Natives, 70% were "acute lesions" and only 30% "chronic tuberculosis associated with silicosis".

2. The Compounds. It was found that there was no relation between poor housing conditions on the mine compounds and tuberculosis, although some of the compounds were bad. None the less, an improvement of compound conditions was recommended, both because overcrowding is likely to be a cause of spread of the disease, and because it increases the likelihood of infections prevalent on the mines such as influenza and pneumonia, which are able to play a role in lighting up dormant tuberculous lesions.

3. Feeding: The diet, though adequate in quantity and well cooked, was deficient in Vitamins A and D, which are important in combating tuberculous infection. In addition, there were only two meals a day; a light one before starting work in the morning, and a large one in the evening. This was regarded as ^{an} unsatisfactory arrangement.

4. Acclimatization. It is emphasized that introduction of new recruits to deep-level mining under humid conditions, and to hard manual work, must be done gradually.

5. Exposure to silica-dust. Evidence is adduced to show that the silica-dust present in the mines may have a part in reactivating the healing tuberculous lesions of those Africans coming newly to the mines who have had a previous infection. According to this theory, therefore, silica dust plays two distinct roles in relation to tuberculosis in miners.

6. Factors leading to tuberculosis in "long-service" Natives.

The Committee gave reasons for believing that the chronic late form of tuberculosis is due to an exogenous re-infection, aided by the simultaneous inhalation of silica-dust and resultant silicotic fibrosis. In fact, they said, the silicotic fibrosis plays a large part in raising the fibrous tissue barriers that localize the disease. At autopsy it is found that the long-service Native who does not present a silicosis usually has generalized tuberculosis.

The pool of infection which exists in these long-service cases is recognized as a danger, and the monthly weighings, radiological examinations of mine workers, and constant attention to the elimination of clinically tuberculous persons, commended and encouraged.

Conclusions:

Morbidity figures are open to the criticism, given by the Silicosis Commission, 1938, that they probably

represent only advanced cases. In the years 1936 - 41 the average number reported was .63/1,000 whereas the Commission in 1938 concluded that the annual production rate was between 5 and 6/1,000.

As far as mortality is concerned, the policy of repatriation is the same as that in the Union; hence the ratio of those dying in mine hospitals to those dying after repatriation is probably about the same (1:2.8). If this is so the mortality figures published for mines are of little value.

Thus as far as can be judged from figures available, morbidity and mortality figures appear to be similar to those occurring on the Witwatersrand.

In considering the part played by silicosis in tuberculosis on the mines, the investigation of the Silicosis Commission was very thorough and speaks for itself. Silicosis is seen to occur in Africans after many years of mining, and to predispose to a very chronic form of pulmonary tuberculosis, just as in Europeans. A newer concept is the opinion of the Tuberculosis Research Committee in the Union, 1932, that silica dust predisposes to the development of tuberculosis in Africans in the early stages, quite independent of the development of silicotic fibrosis.

An interesting fact is that several times more Africans recruited from the neighbouring territories develop tuberculosis and die from it than those who are indigenous to Southern Rhodesia. This might be because their

communities have less experience of tuberculosis, though this does not appear so from the jute mill investigation (page 90). Possibly their nutrition at home is inferior, though the facts do not seem to support this either. We are led rather to lay still more stress on the unfamiliar environment, strange living conditions, the absence of home comforts and the severance of their connection with their families. These considerations are not perhaps as nebulous as they at first appear, and show how close is the relation between social and physical health.

PART II.
FIELD WORK.

The object of the field work was to carry out a fact-finding survey as a preliminary to a large-scale tuberculin-testing and B.C.G.-vaccination programme for the Eastern Region of Southern Rhodesia. The following subjects were studied:

1. The incidence of tuberculin-sensitivity in the region.
2. The best criterion of a positive reaction in the Bantu of this region, which may be applied provisionally to the Bantu in general.
3. The significance^{of}/positive reactions of different types.
4. The B.C.G. conversion rate among the Bantu. Complications of B.C.G.
5. The best technique for the carrying out of a tuberculin survey and B.C.G. campaign on the population of an area such as the Eastern Region.

Extracts from the literature dealing specifically with one of these subjects have been included under the respective headings, instead of in Part I, for greater clarity.

I. SURVEY OF TUBERCULIN-SENSITIVITY RATES IN THE EASTERN REGION, SOUTHERN RHODESIA.

This is reported under the following heads:

- A: Tests on school children and teachers.
B: Tests on industrial employees.

A. Tests on School Children and Teachers.

These were carried out with Koch's Old Tuberculin, 0.1 cc. Altogether 6891 tests were done. The first 1488 were done with 2 T.U. of tuberculin, and after the commencement of B.C.G. inoculations, on the recommendation of the W.H.O. monograph No.12, the remainder were given 5 T.U. The two groups are not therefore comparable and will have to be dealt with separately.

A few of those given 5 T.U. had superficial ulcers, and one a deeper ulcer with a purulent base.

The tests were done partly with a 1 cc syringe, and partly with a tuberculin syringe. The results were read after 72 hours. The age of each child was estimated.

The map, Appendix III, shows the places in which tests were done and the numbers done in each.

(I) Tests with 2 T.U.

These were done at 4 schools: St. David's Mission, Bonda, Old Untali Mission, Mutambara Mission and Nyenyadzi Mission School. The results were interpreted as follows:

Induration of 5 - 10 mm	:	+
Induration of 10 mm or more:		++
Induration with vesiculation	:	+++

RESULTS:1. Crude sensitivity rate.

of 1488 children and teachers tested, 508 or 34.1% showed positive reactions.

2. Sensitivity rates at ages. These are tabulated below.

<u>Age.</u>	<u>No. tested.</u>	<u>No. positive.</u>	<u>Percentage positive.</u>
6	16	1	6
7	81	8	10
8	73	11	15
9	64	11	17
10	90	17	19
11	106	41	39
12	168	62	37
13	150	47	31
14	167	52	31
15	187	72	38
16	165	82	50
17	82	28	34
18	45	21	47
19	19	16	55
20	22	11	50
Over 20	43	28	65
<hr/>			
All ages	1488	508	34

Although the numbers are small, a gradient is seen to be emerging from these figures so that in general the rates become higher with increasing age. This gradient is seen better with the more numerous 5 T.U. tests.

3. Degrees of positivity:

Of the 1488 subjects 186 (12.5%) had reactions which were++ or***, and of these 29 (1.9%) were +++. Thus the proportions of each grade found were approximately:

All positives : 1/3 of total
 ++ or more : 1/8 of total
 +++ or more : 1/50 of total.

The ++ and +++ reactions were scattered in a random fashion about the positive reactors of all age groups.

The chest and neck glands of 25 ++ and +++ reactors were negative to clinical examination.

4. Sex and tuberculin sensitivity.

All mixed classes were included; senior classes with only one sex were omitted.

All told, of 559 girls, 182 or 32.4% were positive; and of 663 boys, 209 or 31.5% were positive.

The crude rates for individual schools were as follows:

	Boys %	Girls %
Bonda	22.6	19.7
Old Umtali	29.8	21.6
Mutembara	45.1	49.5
Nyanyadzi	32.2	38.0

A comparison of the rates at ages for boys and for girls gives the following results (which are, however, vulnerable owing to the small numbers):

Age.	Boys tested.	Girls tested.	Boys % pos.	Girls % pos
6	11	6	(9)	(0)
7	39	41	10	10
8	44	30	11	20
9	21	40	24	15
10	34	51	15	22
11	45	57	38	40
12	77	79	31	43
13	74	64	30	36
14	78	67	33	27
15	76	72	37	42
16	80	37	56	57
17	34	13	44	(31)
18	18	2	(56)	(50)
19	2	-	(100)	-
ALL AGES	633	559		

There is little difference between the two sexes, other than a somewhat greater tendency on the part of the girls towards a sharp rise from the age of 11 onwards. (A greater difference between the sexes is seen with the 5 T.U. tests, where the boys are seen to rise sharply from 8 years and the girls 2 - 3 years later.)

Standardization was done by the indirect method, using the rates for all the boys as the standard. In addition, on the evidence suggested by the crude figures, a division was made into a "northern group" consisting of Bonda and Old Umtali, and a "southern group", Mutambara and Nyanyadzi. The following results were obtained:

	Crude Sensitivity rate %		Standardizing Factor.		Standardized Sensitivity rate %		Diff-er-ence	2 X Stan-dard Error	Result Thus
	Boys.	Girls	Boys.	Girls	Boys.	Girls			
All Schools	33.0	32.5	1.000	1.107	33.0	36.0	3.0	5.5	Not sig- nificant
North- ern Group	28.4	31.2	1.046	1.157	29.7	24.5	5.2	7.5	" "
South- ern Group	37.8	41.7	0.960	1.068	36.3	44.5	8.2	7.8	signifi- cant

In the northern group boys are more affected, and in the southern group girls. In the latter case the difference is statistically significant. This difference is, however, not borne out by the 5 T.U. tests, in which boys have uniformly higher rates.

5. Comparison of different schools:

The schools were representative of their different areas,

particularly those children who were in Std. 3 or below, all of whom were day scholars and none of whom were urban dwellers.

The crude sensitivity rates for the four schools were as follows:

School.	No. tested.	No. positive	% positive	No. ++ or +++	No. ++ or +++ of no. positive.	as %
Bonda	213	53	(24.4)	22	(40)	
Old Umtali	580	178	(30.7)	55	(31)	
Mutambara	295	135	(45.8)	50	(37)	
Nyanyadzi	400	143	(35%)	58	(20)	
TOTAL	1488	508	(34.1)	186	(37)	

Standardization was done by the indirect method owing to the small numbers in certain age-groups. The standard rates at ages used were those for the four schools together. The results of standardization are as follows :

School.	Crude Sensitivity rate.	Standardizing Factor	Standardized Sensitivity Rate.
Bonda	24.4	1.082	26.4
Old Umtali	30.7	0.961	29.5
Mutambara	45.8	0.874	40.0
Nyanyadzi	35.8	1.105	39.6

The groups are thus defined: the northern group consisting of Bonda and Old Umtali, which has a relatively low rate, and the southern group comprising Mutambara and Nyanyadzi, with a higher rate.

It will be seen that at Bonda, the school with the lowest rate, the proportion of positives who gave strong reactions (++ or +++) was high (40%) whereas at Nyanyadzi with a high sensitivity rate, the proportion of strong reactions was much lower (20%)

To determine more precisely the endemicity in the four areas, the above calculation was repeated using only children in Std. 3 and below, all of whom were day scholars. To avoid very small numbers, only children of 15 years and below were included. The results follow:

School.	No. Tested.	No. Pos.	Crude S.R.	Standardizing Factor	Standardized S.R.
Bonda	118	25	21.2	1.149	24.4
Old Umtali	215	33	15.4	1.175	18.0
Mutambara	110	41	37.2	0.818	30.4
Nyanyadzi	385	135	35.1	0.928	32.6
ALL SCHOOLS	836	234	28.0		

Here the two groups remain the same but the proportions within them are reversed. The inference is that at Old Umtali and Mutambara the seniors, who are largely boarders, make a particularly large contribution to the number of positives.

6. Family spread of tuberculin sensitivity.

Possession of the same name by children attending the same school was taken as a rough indication of children in the same family. Two schools - Nyanyadzi and Bonda - were

analysed in this respect. In the table of results which follows "+" indicates a positive case in the family, and "-" a negative case. The "distribution expected by chance" is based on the average sensitivity rate of the two schools (30%)

Families of	Number of Families	Distribution expected by chance	Distribution noted.
2	51	++	5
		+ -	21
		--	25
3	17	+++	0
		+ - -	3
		- - -	8
		- - -	6
4	10	++++	0
		+++ -	1
		++ - -	3
		+ - - -	4
		- - - -	2
5	7	+++++	0
		++++ -	0
		+++ - -	2
		++ - - -	2
		+ - - - -	2
		- - - - -	1

In the few larger families, the only ones worthy of note are one of six, with five positives and one negative, and one of seven, all of whom were negative.

It will be seen that the distribution of positives and negatives in "families" is very much the same as would be expected by chance. It must, however, be remembered that these "families" are merely children with the same family name, not necessarily living together. A more

intensive study is required if actual proved families are to be compared. Making allowance for this, it still seems that tuberculosis infection does not spread particularly in families. Distinction should be made from actual disease, which is well known to spread in families. A possible inference is that family susceptibility to disease is a result, not so much of transfer of infection, as to the transfer of a large dose of organisms coupled perhaps with a low resistance of environmental or genetic origin.

II) Tests with 5 T.U.

Of these there were 5403.

RESULTS:

1. Crude sensitivity rate:

Of 5403 children tested 2332 or 43.1% were positive.

2. Sensitivity rates by age and sex.

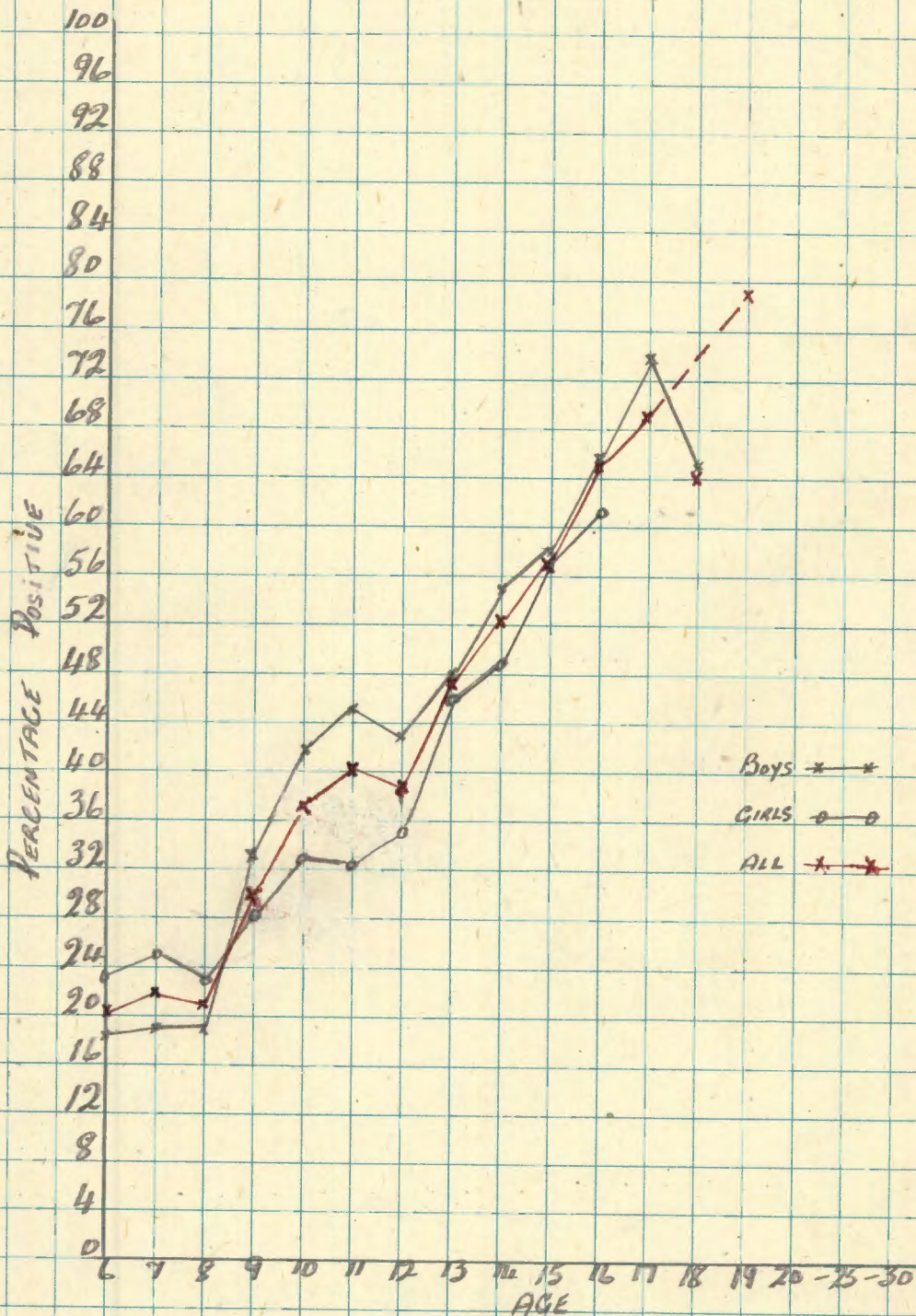
Details are given in Table A, Appendix IV.

The following are the rates from ages 6 - 30, any rates based on a total number of less than 70 being omitted:

	Age	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	25	30	All Ages
Males	18	19	19	33	42	45	43	48	55	58	66	74	85	85	--	--	--	--	47
Females	23	25	23	28	33	32	35	46	49	57	61	--	--	--	--	--	--	--	37
Both Sexes	20	22	21	30	37	40	39	47	52	57	65	69	64	--	--	72	--	--	43

Figure 3 shows these results graphically. It will be seen that the girls' rate is higher than that of the boys up to 8 years of age. The boys' rate rises rapidly from 8 years to 10 years of age, and then flattens out to 12 years;

FIGURE 3
REACTION RATES TO O.T. 1/2000, 0-1cc., of
5403 School children and teachers.
Feoch. reading based on not
less than 70 observations



at this stage the girls have a rapid rise and the two then continue upwards together. The drop at 18 - 19 is due to the fact that these consisted almost entirely of boys from Rusitu and Mount Selinda, where the rates were unusually low.

3. Comparison of the different schools involved.

For this study only children known to live locally were taken. This meant the exclusion of certain large rural senior schools. The other schools were grouped together and combined to give a single percentage, according to the area in which they lay.

The distribution of boys and girls was fairly even at all levels in all schools other than the Mount Melleray and Inyanga North groups. Standardization was done only for ages, not for sexes, so that the figures obtained for these two groups are rather high. As in the 1/5000 group, only children of 16 and below were included. Comparison was made by means of "comparative sensitivity indices"; i.e. the standard is 1.0 and each school has a figure by which its rate can be compared with each other school. In order to conform to the standardizing method used with the 2 T.U. tests, the standard used was the rates at ages for the average of ^{the boys in} those tests, and the nett rate of the standard, which was 28.6%, is regarded as a C.S.I. of 1.0. It is therefore clear that the schools tested with 5 T.U. will have C.S.I.'s, on the average, considerably higher than 1.0.

The tables and formula from which the C.S.I.'s were

derived are given in Table B.

The following list shows the C.S.I.'s for the 10 groups tested with 5 T.U. of O.T. (See map, Appendix III):

Umtali Schools	1.26
Rusape Schools	1.11
Hode	1.35
Mutema Group	1.65
Rowa Group	1.72
Chipfatsura Group	2.42
Dowa	1.52
Mount Selinda	1.18
Mount Melleray	1.18
Inyanga North	1.37

(The last two, as stated above, should be lower owing to their predominance of boys).

Of the towns, Umtali may be classed as definitely urban and Rusape as semi-urban. All the other schools are rural. The highest rates come from the schools which are in the flat, low-lying hot areas, particularly those near large river valleys; these results are in conformity with those for the 2 T.U. tests, where Nyanyedzi and Mutambara had the highest levels. The urban schools, unexpectedly, do not give unduly high rates.

To attempt to find a factor which will raise the rates of the schools tested with 2 T.U. so as to make them comparable with the 5 T.U. schools, is a tricky procedure; however, a tentative effort may be made, bearing in mind the limitations of the method.

The C.S.I's for the 2 T.U. schools were as follows:

Old Umtali	0.63
Bonda	0.85
Mutambara	1.06
Nyanyadzi	1.14

Let us take two groups of schools. Mount Melleray C.S.I 1.18 or a little less) and Bonda (C.S.I 0.85) are both in the hilly, cool Inyanga district. The ratio between their rates is 1.39. Nyanyadzi (C.S.I 1.14) and the Mutema group (C.S.I 1.65) are both in the hot Sabi Valley, very near to the river and not far from each other. Their ratio is 1.45. These ratios are reasonably close together. Approximately, then, the multiple we should apply to the 2 T.U. group to make them comparable with the 5 T.U. group may be taken as 1.42.

Applying this multiple the following C.S.I's are obtained:

Bonda	1.21
Old Umtali	0.89
Mutambara	1.50
Nyanyadzi	1.62

The tendency among the "southern" schools for girls to have a higher rate than boys, found with the 2 T.U. tests, is not confirmed by the more extensive 5 T.U. tests; there is a universal tendency for boys to have a higher rate than girls.

B. Tests on industrial employees.

1. Tuberculin sensitivity rates.

1739 male employees were tested with 5 T.U. of O.T. 1392 of these were in a jute mill, the remainder being from a large department store, a mineral water factory, a timber yard and four oil companies. The results were as follows:

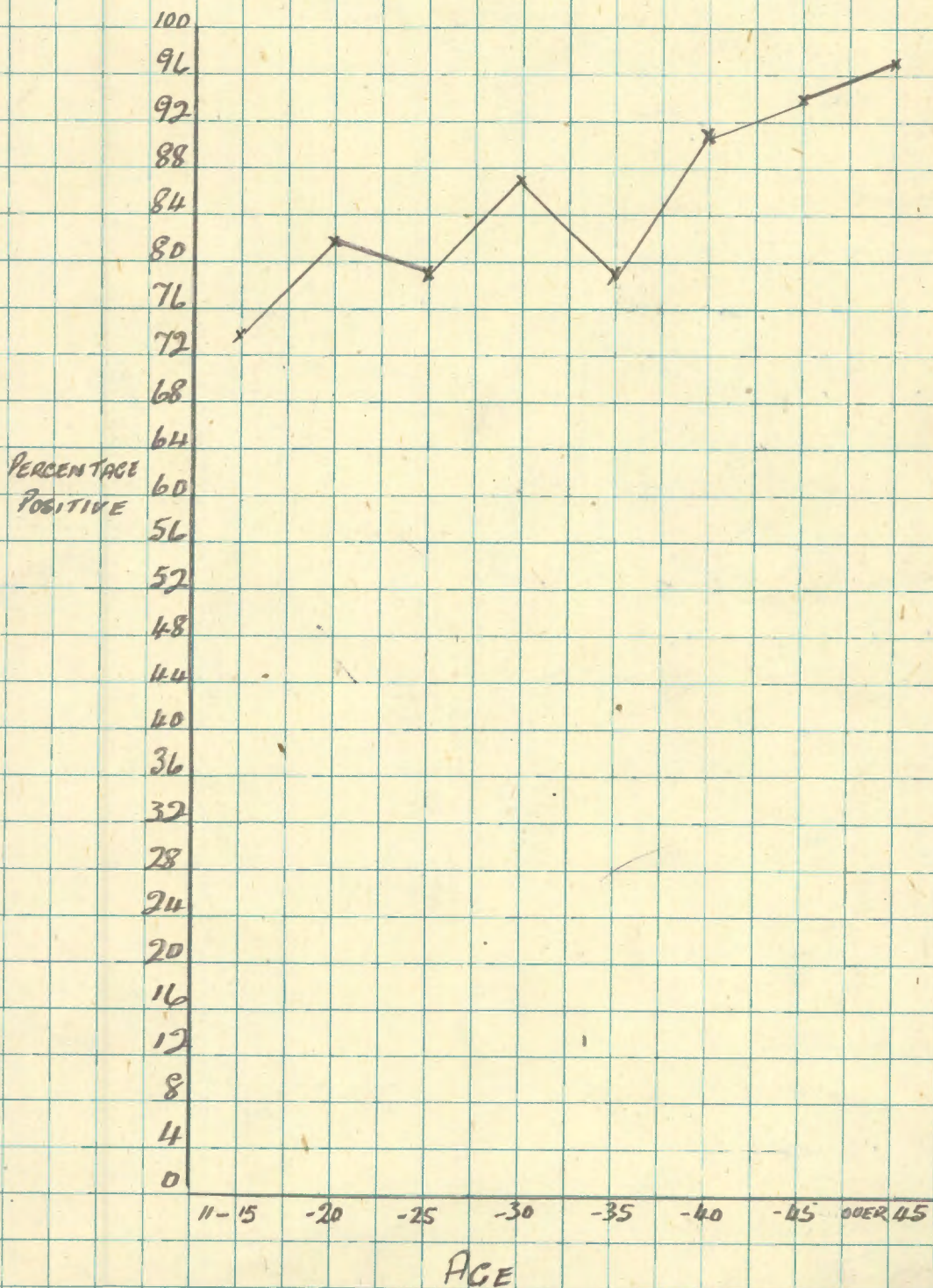
Age	Positive	Negative	Total	% Positive
11 - 15	86	31	117	74
- 20	473	107	580	82
- 25	259	69	328	79
- 30	232	35	267	87
- 35	146	38	184	79
- 40	113	11	124	91
- 45	65	4	69	94
- 50	40	1	41	-
- 55	20	1	21	-
- 60	7	-	7	-
- 65	1	-	1	-
All ages	1442	297	1739	83

The low figure for the 31 - 35 group was due to the fact that one firm with 200 employees had 21 out of 48 negative in this age-group. The reason for this was not explained.

These results are presented graphically in Figure 4.

The overall rate for the jute mill alone was also 83%, so that the other firms together had the same rate as the jute mill.

FIGURE 4
REACTION RATES TO 0.1CC OF K-O-T 1/2,000
OF INDUSTRIAL EMPLOYEES IN UTAH,
BASED ON NUMBERS NOT LESS
THAN 69



2. Tuberculin sensitivity and duration of employment.

The tests on the Jute Mill employees were done in April 1955. The mill employees were analysed for sensitivity rates according to the year they took up employment. The results are given in Table C. These results had to be standardized for ages, and the standardized sensitivity rates, based on the average rates for all (the indirect method being used) were as follows:

Employed in 1955	79%
" " 1954	85%
" " 1953	84%
" " 1952	86%
" " 1951	86%
" " 1950	84%
" " 1949	86%

All employed before 1955 have about the same rate, ranging round 85. This figure is significantly different from the 79% of 1955 ($2 \times S.E. = 4.84\%$; $O.D. = \pm 6\%$).

Thus, although the majority have already acquired an infection before entering employment, there is a definite increase in the number acquiring it in the first few months of employment but no further increase after that.

On account of this difference, a standardization was made of the ages according to whether employed in 1955 or in another year. The results which are shown in Table C are not materially different from those obtained without standardization (also shown in the table).

3. Tuberculin sensitivity and nature of employment.

The jute mill employees were divided into those working inside and those working outside the mill building. The age distribution in the two groups was approximately the same. The following results were obtained.

Working outside : 58 employees.
 positive : 47 "
 negative : 11 "
 % positive : 81%

As the rate for all was 83%, there is no appreciable difference between work outside or inside the mill as predisposing to tuberculous infection.

4. Tuberculin sensitivity and tribes and countries of origin.

The jute mill employees were divided according to tribes. The following overall rates were found for the tribes supplying most employees to the business:

P.E.A. Tribes.

<u>Tribe.</u>	<u>Positive</u>	<u>Negative</u>	<u>Total</u>	<u>% Positive.</u>
Ndau	64	11	75	85
Shangaan	41	10	51	80
Sena	378	40	418	90
Manyika	52	14	66	79
<u>Other P.E.A. Tribes</u>	<u>114</u>	<u>18</u>	<u>132</u>	<u>86</u>
All P.E.A. Tribes	649	93	742	87.4

S. Rhodesian tribes.

<u>Tribe.</u>	<u>Positive.</u>	<u>Negative.</u>	<u>Total.</u>	<u>% Positive.</u>
Muduma	47	11	58	81
Ndau	36	25	61	59
Karanga	45	6	51	88
Mboca	42	11	53	79
Mjindwi	52	19	71	73
Manyika	102	31	133	77
Other	142	30	172	83
All S.R. tribes	466	133	599	77.8

The difference between all Portuguese East Africa tribes and all S. Rhodesian tribes is significant (2 X standard error = 4.2; observed difference = 9.6)

Table D shows the tribes subdivided into years of employment: 1955, 1954 and before 1954. The purpose of this was to determine if employment in the mill has the effect of diminishing the differences in rates in different tribes. The rate for all employees engaged in each period was thus found, and the variations of the rates for each tribe in that period from this average calculated. The 12 variations thus obtained in each case were averaged to give the "mean variation" in the last column. The mean variations thus obtained were:

Before 1954	7.17%
1954	7.83%
1955	11.92%

To decide the significance of the difference between the

"before 1954" and the "1955" variations, the standard error of the difference between these was calculated as follows:

The standard error of each of the 12 variations making up the mean variation is the same as that of the proportion from which it is derived. These 12 S.E.'s were averaged. The number of readings on which the result is to be based is however 12 times larger than if we took the average S.E. of the 12 proportions; thus the S.E. diminishes by a factor of $\sqrt{12}$. In this way the S.E. of each mean variation was determined and from them the S.E. of the difference between them.

The S.E. of the difference thus calculated was 3.51%; 2 X S.E. was thus 7.02%. The observed difference is only $11.92 - 7.17 = 4.75$, so that the difference is not significant. It is however worthy of note that the higher variation is in the 1955 group, whereas the 1954 and before-1954 groups have levels almost the same. The results are in line with those for the standardized sensitivity rates in different years of employment; 1955 employees, $3\frac{1}{2}$ months or less in the firm have both lower rates and greater tribal variation; all other years are approximately the same in positive rate, and more or less "ironed out" in tribal incidence.

In support of the significance of the difference between 1955 and before-1954 levels is the comparison for individual tribes; all the 12 groups designated have lower variations from the mean in the before 1954-group, and 10 of the 12 have

the variation on the same side of the mean in each case, only 2 having changed to the opposite side.

Summary and conclusions from sensitivity-rate surveys.

There is every evidence that tuberculosis has become well disseminated throughout the Eastern Region. School children as young as 6 are 20% tuberculin-sensitive, and the rate increases progressively to about 80% at 20, thereafter increasing further to about 95% at 45. An urban upbringing does not seem to make the rate any higher in children, but those living in low-lying hot areas, such as river valleys, are more likely to be positive than those living in the cooler uplands or hilly areas. Girls have on the average rates 10% lower than boys, commencing a little higher but becoming lower at the age of about 8 - 9. The higher rates for boys than for girls are observed throughout all the individual schools making up the sample for the 5 T.U. tests. The rates for both sexes remain at the minimum for the first 3 years of school life (6 - 8) and this may therefore be the optimum time to give B.C.G. to school children. Those about to undertake employment are so high (at 16 - 17, two-thirds of children are positive to 5 T.W) that the administration of B.C.G. to industrial recruits or high-school leavers would involve much labour in tuberculin-testing with relatively little return in inoculations done. In a population as highly tuberculized as this one, age 6 - 8 is the one of choice for the inoculation of children,

since it is in the next 2 - 3 years that the highest rate of natural infection occurs. In practice this will mean concentrating on the "kraal schools" (kindergarten and early primary), which are attended by the great majority of African children. Heaf (1955) states that infants should be vaccinated if the reactor rate is more than 10% by the age of 5 years. In S. Rhodesia the nearest we can come to vaccinating infants is to do the school entrants.

No evidence could be elicited that transmission of tuberculous infection, as shown by tuberculin sensitivity, occurs more within families than between members of different families.

Roughly, the rates obtained with 5 T.U. of old tuberculin are 40% higher than those with 2 T.U. In any case there is a marked difference, indicating that probably many positive reactors are missed by the weaker dose (even with the use of the 5 mm criterion instead of 6 mm for a positive reactor). On the other hand, there were a few very strong reactors to 5 T.U. who developed large superficial ulcers for a short period, and one was known of who developed quite a deep ulcer with a purulent base. Probably the best procedure for routine pre-vaccination testing is the use of 5 T.U. with a wary eye, when reading tests, for large vesiculating reactions which may develop into ulcers. A suitable dressing such as lint and elastoplast should be carried when reading tests and should be supplied to such

reactors, the teachers also being instructed to watch for ulcers developing later.

Among industrial workers, the rate of tuberculin sensitivity does not level out after 20 years of age but continues to rise until at 45 it is 95%. Employment in industry, as represented by a jute mill, results in an increase of the sensitivity rate for the first few months only; after this it remains constant. Concurrent with this, there is probably a decrease in the variation in rates between different tribes. Portuguese East African tribes have a markedly higher sensitivity rate, on the average, than Southern Rhodesian tribes. Work in the dust of a jute mill causes no increase in the infection rate - though this does not, of course, mean that the disease rate may not be higher among those exposed to such dust.

II. THE CRITERION OF A POSITIVE REACTION IN THE BANTU.

Preliminary: study of reaction sizes by W.H.O.

W.H.O. Monograph No.12, on B.C.G. vaccination (1953) describes an introductory study on tuberculin reactions.

The diameter of the reactions was measured in school children from four different countries: Denmark, Egypt, Mexico and India. The results are expressed in histograms formed by grouping the reaction sizes in twos. In the case of Egypt, and less in India, two distinct waves were found, instead of the expected one, in the formation of the histogram. Denmark and Mexico did not show this feature. The

report states that this shows tuberculin sensitivity to be quantitative rather than clear-cut positive or negative. "But quantitative measurement may reveal qualitative differences". The authors conclude that there are at least two different kinds of accidentally acquired sensitivity to tuberculin. "one kind, found everywhere, is manifested by a strong reaction to a weak dose of tuberculin; this high-grade sensitivity is undoubtedly caused by infection with virulent tubercle bacilli. The other kind, not found everywhere, is reflected in smaller reactions to the same weak dose, and is generally designated as non-specific; it is apparently unrelated to tuberculous infection. This "non-specific" sensitivity, whose cause is still unknown, was disclosed primarily because tuberculin reactions were measured and because data were collected in a uniform way in different areas". The low-grade, non-specific type of reaction was therefore considered to be common in Egypt, but absent in Denmark and Mexico.

The question is asked whether the incidence of tuberculin sensitivity may be over-rated in some areas due to the presence of this non-specific sensitivity.

A. Measurement of reaction sizes in Eastern Region School Children.

Method.

2173 of the children had their reactions measured for this study. The injection in all these cases was given with

a tuberculin syringe, and any considered not to have had a sufficiently accurate amount of tuberculin were excluded from this reaction-size series. The children were from 7 schools in 4 well-scattered areas (see Table E and map, Appendix III). 4 of the schools contained younger children (mainly less than 12) and 3 older (mainly over 10). A few teachers were also included.

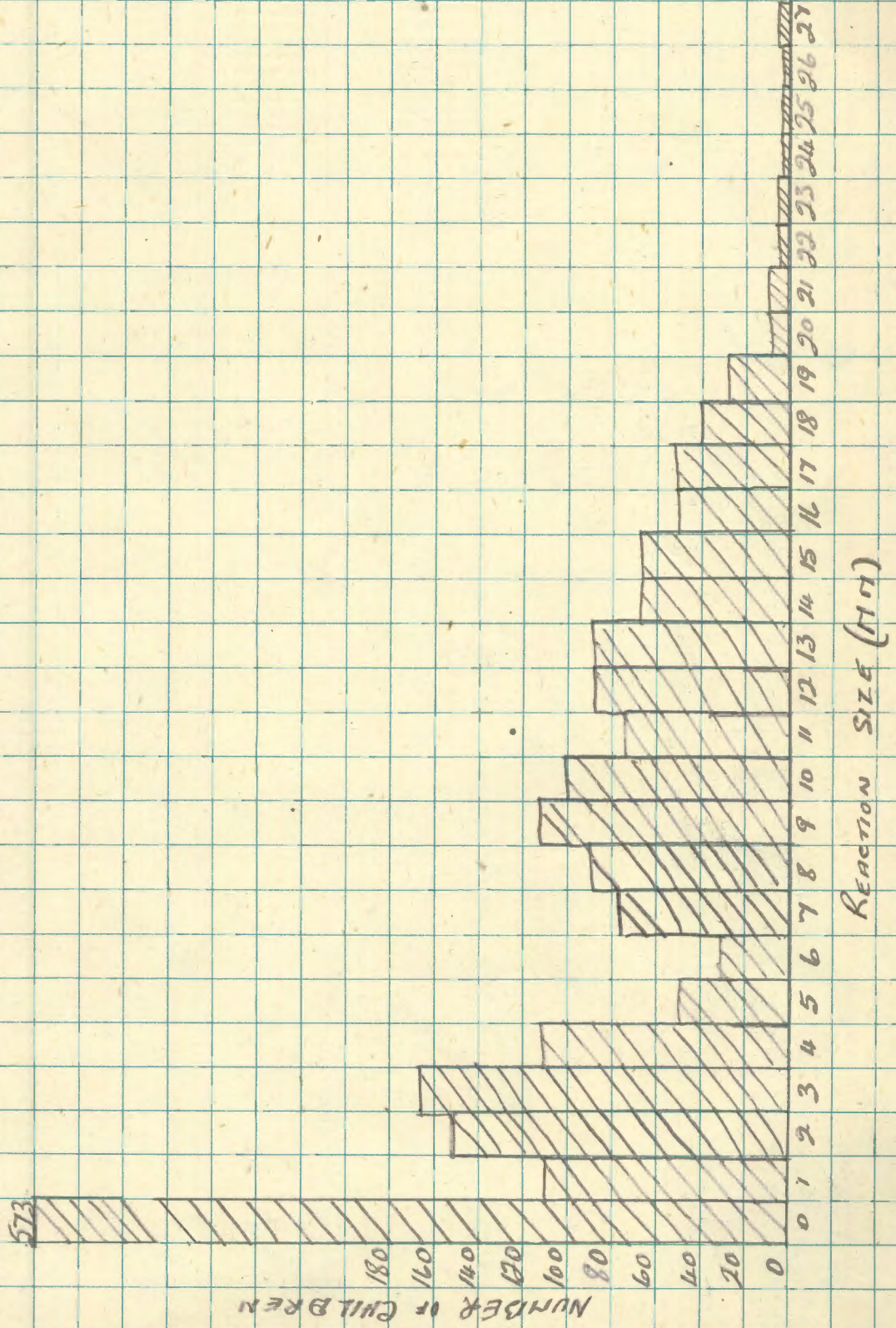
RESULTS.

These are expressed in full in Table E.

- (1) The curves given by boys and girls are approximately the same.
- (2) Study of the results from individual schools showed that they all conformed roughly to the general pattern.
- (3) The charts for individual schools also showed that there was no correlation, positive or negative, between age and reaction size. From the age-distribution of the group, however, it is clear that this result only applies up to the age of 17 or 18.
- (4) Figure 5 shows the distribution of reaction sizes through these 2173 children. If this result was expressed the same way as those of W.H.O., i.e. in groups of 2 millimetres, the effect would be very similar to those of Denmark and Mexico. However, in 1 mm groups, an initial wave reaching its peak at 3 mm and ending at 6 mm is defined. The second peak, that due to the true positive reactors, occurs at 9 mm. There is a small resurgence at 12 - 13 mm, and this too was seen in several of the component schools.

FIGURE 5

SIZE DISTRIBUTION OF 2173 TUBERCULIN REACTIONS



Its significance is doubtful; as it is small it should be ignored.

Conclusions:

- (1) The "non-specific" factor defined in the W.H.O. Monograph appears to be present in the population under study, since there is a clear convex curve in the range 1 - 6 mm. On the other hand, this factor does not cause any significant increase in the number of persons described as tuberculin-positive, since the meeting place of the first and second curves (at 6 mm) is almost at the base-line.
- (2) The generally accepted criterion of 6 mm. for a positive tuberculin reaction in the Bantu is here confirmed as the correct one, when Koch's O.T. 1/2,000 is being used.

B. Tuberculin tests with control injections using heated tuberculin.

Object.

It was intended to destroy the active principle in the tuberculin by heating it, and administer this as a control at the same time as the normal test was given. It was hoped by this means to "isolate" the non-specific reactions if they were due to something other than the factor causing positive reactions, and decide thus to what size non-specific reactions may reach.

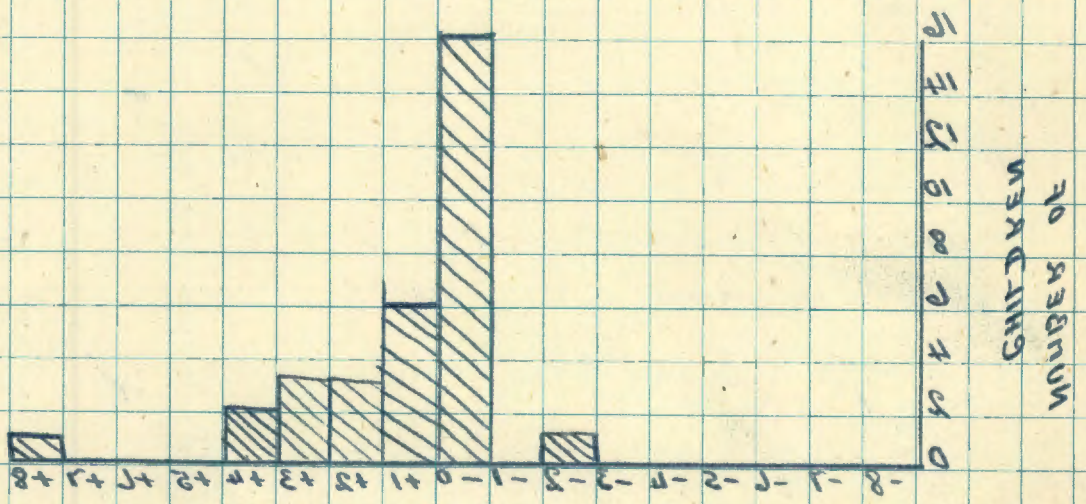
Method.

A 10-cc bottle of tuberculin was mostly immersed in boiling water for 5 minutes. This was then given in 0.1cc

INCREASE IN SIZE OF CONTROL OVER NORMAL TUBERCULIN REACTIONS

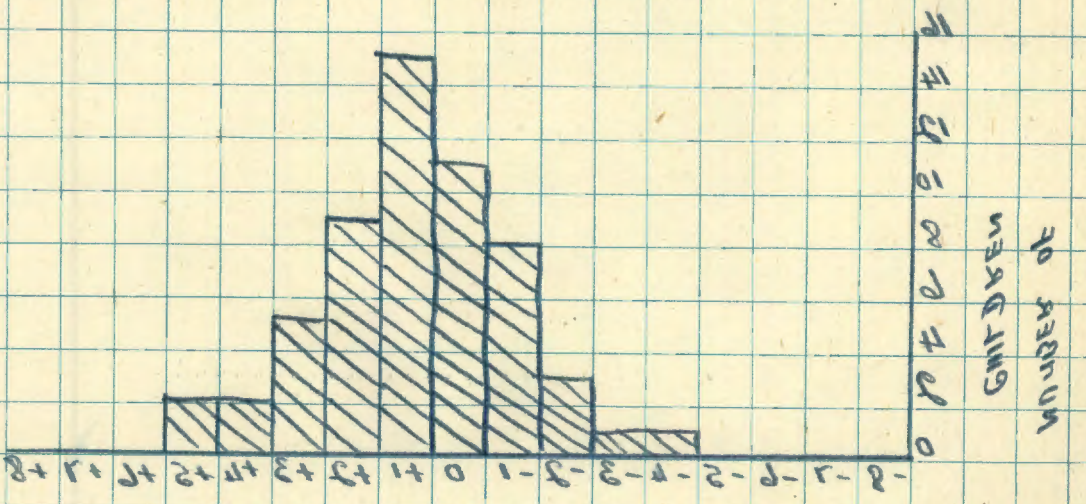
FIGURE 6

A. NEGATIVE REACTORS (2 MM OR LESS TO NORMAL TEST)



NORMAL REACTION SIZE COMPARED WITH CONTROL REACTION SIZE

B. POSITIVE REACTORS (2 MM OR MORE TO NORMAL TEST)



doses to 89 children as a control in the left forearm, while untreated tuberculin of the same batch was given in the right forearm.

Results.

The period of 5 minutes' immersion in boiling water may have been insufficient; in any event the results of the experiment were entirely unexpected. All positive and negative reactions were approximately the same size as with the normal tuberculin, but were on the average just under 1 mm larger. Table F shows the results, expressing them as (size of control reaction minus size of normal reaction) for each individual. In Figure 6 these results are expressed graphically.

Among negative reactors, only 1 control out of 32 was smaller than the normal. Positive reactors show a fairly symmetrical distribution around a point a little below +1 (+0.79). Scrutiny of the individual cards showed that in general, the larger the positive reaction, the larger is the control reaction in relation to the normal one.

Conclusions:

- (1) The results with the negative reactors suggest that the heating enhanced the effect of the non-specific factor.
- (2) The same effect is seen in the results with the positive reactors, in a general raising of the average to +0.79 over the normal readings. (The average increase among the negatives is similar, i.e. 35/32 or 1.09).
- (3) A second effect in the case of the positive reactions

is seen in their wide distribution round this mean of + 0.79. This means that heating the tuberculin leads to a decrease of the accuracy of it for reading the size of positive reactions.

(4) The result of the experiment supports the idea that there are both specific and non-specific factors involved in the tuberculin test.

III. THE SIGNIFICANCE OF DIFFERENCES IN THE NATURE OF POSITIVE REACTIONS.

The literature on this subject is reviewed on pp. 35-38. On the whole there is agreement by these authors that strongly positive reactions are correlated with the development of active tuberculosis. This conclusion is reached by means of a difference in proportions or a significant correlation coefficient; no attempt is made to put the fact to practical use and find cases by means of it.

The experiment described below was precipitated by certain occurrences. As a result of the 2 T.U. tests 12 children with vesiculating reactions were X-rayed. 4 of these showed calcification in the chest. Another child, aged 12, with a vesiculating reaction, due for an X-ray, fell ill and went home before this was done. The sister at the Mission hospital thought he was suffering from tuberculosis. Soon after arrival home he died. A woman tested in a village with 5 T.U., who gave a "brawny" reaction \approx 15 mm in diameter, was notified 5 months later

as having been admitted to hospital with tuberculosis.

The question now arose: Is there any criterion for the nature of a tuberculin reaction, by which one can say that it is worth while having the subject X-rayed? This question must, of course, be seen in its Rhodesian setting, in which X-rays are available only in small numbers, and in most areas hard to reach.

Method.

Mutanda School, Umtali, was chosen for the experiment. All the tests were done with tuberculin syringes and the diameter measured. At this school, where 593 children were tested and read, there were 301 positive reactors. Of these, 270 had erythrocyte sedimentation rates done, the exclusions being teachers, absentees and those who had difficult veins. Of the 56 with E.S.R.'s higher than 12 mm in 1 hour (Westergren) 46 were X-rayed, the exclusions being 2 absentees and 8 children in the 13 - 14 E.S.R. group, who had to be omitted because the availability of the X-ray machine was limited. X-rays were also done on the three vesiculating reactors who had low sedimentation rates. The radiologist reading the X-rays was not told of the reaction or E.S.R. until he had pronounced upon the film.

19 E.S.R.'s were taken from negative reactors as controls.

The E.S.R. estimations were completed within a month of the tests being carried out.

Urine tests were carried out on 37 of the children with high E.S.R.'s, and on a control group of 35 children whose

E.S.R.'s were normal, in an attempt to find another cause for some of the many high E.S.R.'s.

Results.

In Figure 7, reaction sizes are plotted against E.S.R.'s. Reactions felt to be "hot" are inserted in red, and vesiculating reactions in green.

The first matter to be considered is what constitutes an abnormally raised E.S.R. Whitby and Britton (1946) referring to the Westergren method, give the normal for females as 4 - 7 mm in 1 hour and for ~~females~~ males 3 - 5 mm in 1 hour. It is, however, generally recognized by clinicians in this country that higher levels are very common and often unassociated with any evident disease. Patients do frequently harbour parasitic diseases which are not searched for, e.g. ancylostomiasis, schistosomiasis, ascariasis, or malaria, and this may be a factor causing the high levels found. The Annual Report of the S.A.I.M.R., 1954, states that in mining recruits, "grossly abnormal E.S.R. values were found to be common, values which in white subjects would indicate grave or fatal pathology". The values tended to become normal after the subjects had been eating for months a good balanced mine diet. It is presumed therefore that the E.S.R. can be raised markedly merely by the consumption of an inadequate diet.

A glance at the last column in the figure shows that the steady descent of the numbers of E.S.R.'s from a level of

4 mm to higher readings, is broken by a dip at 10 mm. This might be suggestive that any level above 10 mm should be regarded as abnormal. However, if this were done it would mean that 91 of the 291 E.S.R's were abnormal. The level chosen was an arbitrary one - 12 mm in 1 hour. In this way 58 E.S.R's were put in the abnormal class.

E.S.R's in negative reactors: In the 19 tuberculin-negative children, 2 high sedimentation rates were found: one of 20 and one of 36. In the former there were no symptoms, and no clinical or urinary abnormality; the latter had had a 3-month's illness, away from school, ending 7 months before, and stated that he still suffered from headaches. The urine showed a trace of albumin. Clinically there was no abnormality.

E.S.R's and reaction sizes. Figure 7 shows that 8 out of 10 reactors (80%) over 20 mm had raised E.S.R's; whereas only 48 out of 262 with reactions under 20 mm (18%) had high readings. (The difference is well within the bounds of significance; twice the standard error is 26%; observed difference 62%). It will be seen that all the 8 who combined a high E.S.R. with a reaction size over 20 millimeters had reactions classed as "vesiculating" or "hot". There was no correlation between E.S.R. and reaction size if the latter was less than 20 mm.

All the 9 vesiculating reactors were either less than 20 mm with a low E.S.R.(5), or more than 20 mm with a high E.S.R. (4).

FIGURE 7

ESR'S AND REACTION SIZES TO 5 TU OF K-O-T

ESR #	REACTION SIZE (MM)													NEG REACTIONS	MODERATE REACTIONS	TOTAL HOT	TOTAL VESICULATING GRAN D	TOTAL
	1	2	3	4	5	6	7	8	9	10	11	12	13					
36														3	1	1	4	
34	1													1	1	2	2	
32														1	1	2	3	
30														1	1	1	1	
28														1	1	1	1	
26														1	1	1	1	
24														1	1	1	1	
22	1													2	1	3	3	
20		2												1	4	1	6	
18			1											7	1	1	9	
16				2										8	2	1	10	
14	1	1	1	1	1	1	1	1	1	1	1	1	1	13	2	2	17	
12	2	1	2	2	2	2	2	2	2	2	2	2	2	3	29	1	33	
10	1	2	2	1	1	1	1	1	1	1	1	1	1	19	1	2	20	
8	2	3	2	2	1	4	3-1	4	2-2	5	3-2	3-3	3-3	3	31	6	48	
6	3	4	4	4	4	3-1	2	4	5	1	4	2	2	6	43	4	54	
4	7	10	3	5	6	4-1	1	2	1	2	2	2	1	3	50	6	60	
2	1	1	2	1	1	2	3	10	1	1	1	1	1	2	13	2	18	
0	9	22	26	26	17	27	19	25	14	18	16	10	4	19	23	29	91	
TOTALS	9	22	26	26	17	27	19	25	14	18	16	10	4	19	23	29	91	
GRAND TOTALS	9	22	26	26	17	27	22	31	18	20	17	15	7	2	3	2	1	

MODERATE REACTIONS
 BLUE 1
 HOT REACTIONS
 RED 1
 VESICULATING REACTIONS - GREEN 1

X-rays done: Calcification was found in 6 chests: 4 of these were in the large-reaction, high-E.S.R. category, 1 having a vesiculating and 3 a "hot" reaction. Of these 4, one was regarded as probably active tuberculosis. The other 2 X-rays with calcification were two of the three smaller vesiculating reactions associated with low E.S.R's.

Examination of neck glands: All the children with high E.S.R's were examined for enlarged neck glands. A number had soft, moderately enlarged glands. One with large firm tonsillar glands, which were associated with pitted tonsils, fell into the large-reaction, high-E.S.R. class. The glands could fairly be diagnosed, on clinical grounds alone, as tuberculous.

In none of the other X-rays was tuberculosis diagnosed or suspected.

Thus of those with a reaction over 20 mm combined with a raised E.S.R., 5 out of 8 had definite evidence of tuberculosis, present or past.

The high proportion of raised E.S.R's in the over-20mm group of reactors is evidence that these are in fact active cases at the time of examination. This conclusion, however, cannot be drawn until after a study of the results of the urine examinations

Urine examinations. The only abnormality found was schistosomiasis. The whole school had been examined for this a year previously, and the positives then found treated.

On this occasion, of 35 children with normal E.S.R.'s, 2 (5.7%) had *S. haematobium* ova in the urine (E.S.R.'s 7 and 8). Of 37 children with raised E.S.R.'s, 9 (24.3%) had ova. (Significant difference: $2 \times S.E. = 16.2\%$; $O.D. = 18.6\%$). This means that bilharzia can be regarded as the cause of a raised E.S.R.

No less than 4 of the 8 children in the large-reaction, high-E.S.R. groups had *haematobium* ova in the urine. 3 of these had calcification in the chest X-ray - one being the child with evidence of active tuberculosis - and one was the child with tuberculous tonsillar glands. If we allow that these last two would have produced a high E.S.R. on account of active tuberculosis, we are compelled to put the other two into the "low-E.S.R." group in order to make a fair comparison allowing for the presence of bilharzia as a cause of a high E.S.R. The position may now be worded as follows: in the over-20 mm group there are 6 out of 10 children (60%) with a raised E.S.R. which is unexplained or associated with tuberculosis; and in the 20-mm-or-less group there are 43 out of 262 children (16.4%) in the same class. (This number may be high, since not all these children had their urine tested; but it will serve as a maximum for comparison. Any further bilharzia cases would increase still more the difference between the two groups). The difference is still highly significant ($2 \times S.E. = 31.3\%$; $O.D. = 43.6\%$)

Discussion and Conclusions:

1. A comparison of the sizes of the reactions with the radiological and clinical results shows that there is an indisputable relationship between the size of the reaction - whether "over 20 mm" or "20 mm or less" - and the existence, past or present, of a definite well-marked attack of tuberculosis. 5 out of 10 who had reactions of this type, showed evidence of such disease.
2. A study of the E.S.R. levels in the over-20 mm and in the 20 mm-and-under groups, with due allowance for the presence of urinary bilharzia in some of the children, shows a clear statistical difference. It is true that there must be other causes at work to raise some of the sedimentation rates, but these will be evenly distributed through both groups. Thus there is some factor raising the E.S.R.'s of those with large tuberculin reactions, and it is fair to assume that this factor is active tuberculosis of some degree. There are, moreover, in the group, two children shown by their clinical and radiological features to be almost certainly mild active tuberculosis cases.
3. Of five vesiculating reactors having a reaction diameter of 20 mm or less combined with a normal E.S.R., 3 were X-rayed and 2 of these showed calcification, indicating a well-marked infection in the past. This suggests strongly that a vesiculating reaction (i.e. a high degree of allergy) means that a heavy infection has been contracted at some time.
4. From the radiological findings, it appears that the

nature of the reaction - whether merely indurated, hot, or vesiculating - is an indication of the degree of allergy, which is related to the heaviness of the infection which was originally sustained. This is in accordance with the view of Haynes (page 36). The size of the reaction, (whether over 20 mm or not) is on the other hand probably a useful guide to the presence of activity in the lesion. All the vesiculating reactions associated with low E.S.R's were smaller than all those associated with high E.S.R's.

5. These results indicate that wherever X-ray facilities are accessible and transport is available, it is worth X-raying all children giving reactions over 20 mm in diameter, whether vesiculating or not. Though hospital accommodation is at such a premium, it is well worth while to know about early cases, so that they can have the opportunity to rest and be observed, and perhaps given extra supplies of milk or other protective foods, under the supervision of the Mission superintendent, or teacher.
6. The question arises whether, in view of the above findings, any light can be shed on the controversy as to whether the markedly allergic state found in Africans who tend later to develop tuberculosis, is due to the persistence of active infection in a larval form, or merely to a severe state of sensitivity, found after the infection has been overcome, and leading later to an unfavourable response to a re-infection with the disease. Five of the nine

children with vesiculating reactions had also normal E.S.R's: these are unlikely to have had any activity therefore. The possibility remains that the "larval form" of disease may be so mildly active that the E.S.R. is not at all raised. However, though it cannot be stated for certain, it appears that active infection dies down and disappears after the primary attack, while the allergy remains.

7. From the conclusions drawn in 3, it is further reasoned that these severe reactors are children who have had a clear radiologically recognizable attack of glandular tuberculosis, and are not among those who pick up a small dose and imperceptibly become tuberculin-positive. Thus even if the primary attack does disappear leaving only an allergic state, it takes a large primary attack to produce a highly allergic state, and thus the danger of a secondary attack. Whichever theory one supports, the effect is that it takes a large primary attack to produce a secondary attack. This is a strong argument against the contention of Heaf (1955) and others, that B.C.G. protects against a primary attack of tuberculosis but is of doubtful value against the secondary attack. Calwell, in his review of Heaf's paper in the Bulletin of Hygiene, also criticises this view as illogical. There is, however, the possibility, discussed below, that B.C.G., by laying the same allergic foundation as a primary attack, paves the way for secondary infection.

IV. B.C.G. CONVERSION RATES AND COMPLICATIONS TO B.C.G.

A. The use of tuberculin allergy as a guide to B.C.G.-induced immunity.

W.H.O. Monograph No.12 (1953) studies this subject. It discusses the difficulty of studying immunity induced by B.C.G., which can only be done by means of "long-term control studies, in which some are vaccinated and others are not. Allergy, on the other hand, is a readily available yardstick for measuring the immediate effect of vaccination in an individual person at any point in time. The allergic response is so apparently a protective phenomenon that, in the absence of a direct measure of immunity, tuberculin allergy warrants intensive study as a guide, however imperfect, to the effectiveness of B.C.G. vaccination".

Those who work with Africans must accept this decision by the W.H.O. Committee, as the most authoritative available, and there is every reason to suppose that allergy and immunity following B.C.G. inoculation go hand in hand. It might be well, however, to remember at the same time some of the features brought to light about tuberculin allergy in the Bantu. The S.A.I.M.R. Tuberculosis Research Committee found that a higher percentage of positively-reacting recruits to the Rand mines develop tuberculosis later than those who are negative reactors. Also, the greater the allergy, the more the liability to develop tuberculosis later. The question presents itself: does the allergy produced by

B.C.G. represent the same state of affairs as naturally-acquired allergy? If Haynes (see page 36) is right and secondary tuberculosis is due to re-infection of a highly allergic reactor, and not to the persistence of a "larval form" of tuberculosis in the glands, B.C.G. may well be dangerous.

The question (always assuming that the "allergic" and not the "larval" theory is the correct one), boils down to: to what size of infection is a dose of B.C.G. equivalent? Is it always like a very mild encounter with the tubercle bacillus, producing a gentle conversion to Mantoux-positivity; or can it be equivalent to a larger dose, with a high degree of resulting allergy and the danger of a tuberculous infection of the "secondary type"? The character and development of B.C.G.-induced positive reactions are discussed on page 113. These questions, however, must remain unanswered.

B. Nett conversion rate.

The pre-vaccination tests were done on the right forearm anteriorly; follow-up tests on the left.

Most of the children in the Umtali schools who had received B.C.G. were re-tested 6 - 10 weeks later. Exclusions were about 300 children from Dangare School (Sub A, Sub B and Standard I) and those who were absent on the days of re-testing. Also excluded from the following analysis are about 150 children of Mutanda School, who were subjected to an experiment described in the next section.

Of all re-tested, 16 out of 765, or 1.9%, failed to convert to positive.

C. Conversion rates by level of schools and by interval after giving of B.C.G.

The following table gives the details:

School.	Standards.	Period since injection.	No. re-tested.	No. negative.	% negative.
Dangere	Sub.A to 1	6 weeks	268	0	0
Mutanda	1 - 5	6 weeks	150	8	5.3
Sakubva	1 - 6	10 weeks	347	8	2.4
ALL SCHOOLS	Sub.A to 6	6 - 10 wks.	765	16	1.9

The numbers failing to convert did not concentrate on any particular age-group in Mutanda and Sakubva Schools, being fairly evenly distributed through the ages.

From the above figures it appears:

- (a) that conversion is more complete in younger children, i.e. those of about 6 - 9 years of age - there is a significant difference in the percentage of negatives from Dangere and Mutanda Schools (2 X S.E. = 3.3%; O.D. = 5.3%).
- (b) that conversion may be more complete after 10 than after 6 weeks: Mutanda and Sakubva schools have children of approximately the same age (Standards 1 - 5 and 1 - 6 respectively.) The children excluded in Mutanda school were a fair cross-section of the ages in the school and would not materially alter the age-distribution of those remaining. The result given is

not significant: $2 \times S.E. = 3.8$; $O.D. = 2.9$. As it transpired, the results of the experiment with old and with frozen B.C.G., done on the remaining Mutanda School children, (page 125) were entirely negative; hence it is reasonable to include the subjects of the experiment to give a complete picture of the school. If this is done, there are 16 out of 271 children who failed to convert after 6 weeks, or 5.9%. The standard error of the difference between Sakubva and Mutanda schools then becomes 3.25% and the observed difference 3.5%, giving a significant result.

D. Complications to B.C.G.

The following table summarizes these:

School.	Stds.	Period	no	Glands	ulcer	Scab	Total	% com-
		since	comp-	enlarged.	over	over		plica-
		inject-	lica-	sl. mdd.	10 mm.	12 mm.		ted.
		ion.	tions					
Dangere	SubA							
	-1	6 wks.	224	14	1	4	-	243 7.8
Mutanda	1 - 5	6 wks	265	20	-	2	-	287 7.7
Sakubva	1 - 6	10 wks	325	10	1	-	2	338 3.8
ALL	Sub A	6 - 10						
SCHOOLS	- 6	weeks	814	44	2	6	2	868 6.2

One of the Mutanda school children with "ulcer over 10mm" had developed a number of superficial ulcers in the neighbourhood of the original, which though wide was not deep. Frequent ~~homemade~~ dressings had been applied to the injection site.

None of the complications was serious. It will be seen that the rate at 10 weeks was considerably lower, indicating

that glandular swelling subsides to a great extent during these 4 weeks.

E. 6-month reversion rates.

These were done only on Dangare school, the one with young children which had a 100% conversion rate at 6 weeks. These tests were actually performed 5½ months after the B.C.G. inoculations.

Site of injection: The 6-week follow-up tests high on the volar aspect of the left forearm, and the 6-month tests on the left forearm anteriorly near the wrist. 321 children, including some who were not tested at 6 weeks, were tested. There were 23 negative reactors (7%).

It was noticed that most of those who reacted positive to the test had very clear-cut and firm reactions; they retained the large diameter noticed in the original follow-up tests, but had definitely more induration than in those tests. They were more like natural positive tuberculin reactions. This may mean that those who react in this way have consolidated their resistance to tuberculosis, some perhaps with the aid of a natural infection, though it would seem unreasonable to suppose that all who showed firm reactions (the majority) should have acquired a natural infection in six months. Whatever the explanation, the 1, 2, 3, 4 and 5-year follow-up tests on the children of the Umtali schools will be of great interest to see if a flattening-out of the reversion rates occurs, due to

"consolidation" of tuberculin-positivity because of natural infection or any other factor. It is obvious that the success of a B.C.G. campaign must hang very largely on whether the immunity induced lasts permanently, or at least a long time, in a good proportion of those vaccinated.

F. Size of B.C.G.-induced positive reactions.

1. Size distribution compared with that of natural positive reactions.

W.H.O. Monograph No.12 (1953) observes that naturally-induced positive reactions have a complicated size-distribution, with specific and non-specific components, varying in different countries; sizes also vary with the degree of allergy and activity. Those brought about by B.C.G.-inoculation, however, are arranged symmetrically about a mean. "A single, stable, and more informative figure.... This enables discrimination of differences in the level of allergy produced by vaccination in different groups of people".

2. Size distribution of B.C.G.-induced positive reactions.

The mean size of the 111 natural positive reactions obtained from the first group of Dangere School children was 13.44 mm. The mean size of the 268 B.C.G.-induced positive reactions in the corresponding group of negative children was 14.47 mm. Inspection makes it clear that the standard error of the one will easily embrace the other, hence the difference is not significant. The clinical

impression, that the positive reactions following B.C.G. inoculation are considerably larger than those occurring naturally, is therefore not supported. The distribution of the reactions in a symmetrical fashion round their mean, is however well shown (Figure 8). This histogram should be compared with that of Figure 5 (following page 97). The figures on which it is based are given in Table G.

3. Size distribution in relation to complications to B.C.G.

The relevant figures are given in Table G. There was no relationship between the size of the reaction and the development of complications to the inoculation.

4. Average size of reactions at 6 weeks in children who had reverted to negative after 6 months, compared with those who were still positive after 6 months.

The mean size of the 17 6-week reactions of those who had become negative by 6 months was 11.3 mm (Standard deviation 3.39 mm). The mean size of the 268 6-week reactions was 14.5 mm (S.D. 3.37 mm). Twice the standard error of the difference between those two means is 1.7 mm and the observed difference 3.2 mm, so that the difference is significant.

Thus those who are shortly going to lose their allergy tend to have reactions decreasing in size beforehand, and a group with large reactions 6 - 10 weeks after B.C.G. may be regarded as likely to retain a high proportion of conversions, and vice versa.

FIGURE 8
SIZE DISTRIBUTION OF 268 POST-VACCINATION REACTIONS
5 T.U. OF K.O.T.



Summary

The post-B.C.G. positive reaction in the Bantu may sometimes have dangers as well as advantages.

The conversion-rate in 765 school children was 1.9%, younger children being more completely converted, and probably also those who are tested after 10 instead of after 6 weeks.

Complications to B.C.G. were all of minor degree, and occurred in 7.7% of those tested at 6 weeks and 3.8% of those tested at 10 weeks.

Among 321 younger children (aged 6 - 10), all of whom had converted to positive originally, 7% were found to be negative again after 6 months. Those reverting had had, on the whole, smaller reactions than usual at the 6-week test. The majority of the others had firmer reactions than at 6 weeks. This "consolidation" may mean that the change to positive has become permanent, but this must be confirmed by follow-up tests in successive years.

The sizes of B.C.G.-induced positive reactions are symmetrically arranged about a mean. The size is not related to the occurrence or not of complications at the injection site.

V. FIELD METHODS TO BE ADOPTED.

A. Technique of giving injections.

In the campaign to vaccinate and keep vaccinated all school children in an area containing about 400,000 people, speed is of the essence. At the same time sufficient care must be taken in giving injections to avoid false positive

or negative tuberculin reactions, unnecessary complications to B.C.G., or a lowering of B.C.G. conversion rates. Certain points must be studied in determining the amount of care needed in giving injections.

(1) Tuberculin tests.

1. Volume injected. No actual experiment was done to determine the effect of variations in volume. Guld (1954) after varying the volume but not the total dose given, concludes "that efficient syringes which do not leak are necessary for accurate Mantoux testing". It is felt that to get the best combination of accuracy and speed, tuberculin syringes should be used and not ordinary 1 cc syringes. However, for simple determination of positive or negative reactions, without measurement of the diameter of the reaction, an error of up to .02 cc in an occasional injection is unlikely to be of serious importance.

2. Depth of injection. In this study the method used was to insert the needle nearly parallel to the skin, and then push the point upwards until on depressing it a clean-cut dimple appears on the surface. This guarantees a superficial injection, but it is more painful than simply pushing in the needle, and takes a little longer. It was therefore decided to determine, on a small scale, whether a less careful technique could be regarded as satisfactory.

Guld (1954) found that "reactions to deep tests were on the average of the same size as those to superficial tests, but the former were the more difficult to read". The

subjects were Danish school children.

In the course of the present survey, two small experiments were done.

A. About 12 children were given a superficial injection in the right forearm, and one which was intra-dermal but not sub-epidermal in the left forearm. 5 gave positive reactions to the former injection.

B. Another 12 received a superficial test in the right arm, and in the left arm one which was as superficial as possible without hooking the needle upwards to catch the epidermis. 6 gave positive reactions.

The following results were obtained:

A. Intra-dermal, not superficial.

	<u>Size.</u>				
Right arm	7	7	7	10	11
Left arm	3	-	6	-	11

B. Superficial, but no hooking.

	<u>Size.</u>					
Right arm	8	8	8	9	10	16
Left arm	8	-	6	7	10	17

Even these small numbers are sufficient to show that the less painful and more rapid techniques are unsatisfactory, and the epidermis should be "hooked down" in every case.

3. Repeated injections at the same site.

No trial was done of this; however the W.H.O. workers (Monograph No.12, 1953) reported that different results were obtained if an individual was re-tested in the same place. The method used has therefore been, and will be

for the future:

Original test, middle of right forearm (all anterior)

2-month follow-up, high on left forearm

6-month follow-up, left forearm near wrist

1-year follow-up, right forearm high up

2-year follow-up, right forearm near wrist.

For further tests new sites will be selected.

(II) B.C.G. inoculations.

1. Volume injected.

W.H.O. Monograph No. 12 (1953) goes into the technique of B.C.G. inoculations deeply. The authors report that, as regards the production of tuberculin allergy in vaccinated children, it is clear that small differences in the amount of B.C.G. vaccinated have little practical importance.

2. Depth of injection.

The same work reports that the depth of injection does not influence the degree of allergy induced, but has a marked effect on the size and severity of the local lesion.

B.C.G. can therefore be given with an ordinary 1-cc syringe, and this is more convenient than a tuberculin syringe when the point of the shoulder is being used. Care is, however, needed to ensure that the injections are given superficially, to avoid complications.

B. Organization within a school.

Kraal schools (up to Standard 3) which form the great majority, are only open in the mornings, so that all work

must be done at this time. The operator takes with him all equipment, including a primus burner, and one assistant, who need not be a trained nurse but must be intelligent and trained to sterilize needles and change them on syringes. Cards, which may be printed or prepared from a rubber stamp, are sent out beforehand and filled in by the class teachers with the name, age, sex, standard and school. The cards are issued just before the advent of the doctor.

On arrival a fairly large room with good window-space is needed; in this two tables are ^{placed} ~~needed~~, one for the cards and the bowl of swabs, and the other, which should be narrow, such as a standard African school desk, across which the doctor may give the injections. Between the two tables a school bench is placed lengthwise; children after being swabbed are sent down alternate sides of this with instructions to put their arm flat down on the table. In this way there is always another child waiting when ^{the doctor} ~~one~~ has finished one injection. Once this routine has been firmly established at the beginning of the session, the succeeding children will imitate exactly what their predecessors do. Also, if the first few children are told to hold out the arm sideways after being swabbed, so as not to touch anything, the others will do the same. The bench down the middle is a sine qua non, for to African children, though single-line queuing is a commonplace, the concept of a double queue is completely foreign; without the bench a single line will always be formed. Three assistants, either teachers or pupils, are asked for; one

collects and date-stamps the cards at the first table; one swabs the arms, having been carefully instructed in how to do this; and the third fetches new classes as the ones being done approach completion. During the half-hour that it takes to prepare for the injections, these three are put on to making swabs.

During the injections, one lot of 20 needles is kept boiling while the others are in use. The assistant changes the needle on one syringe while the second is in use, and they are then exchanged by hand. The assistant changes the bowls of needles with the sterilizer, and the doctor refills the syringes from the vial, when necessary. In this way 240 tuberculin tests can be done in one hour.

During the half-hour of preparation, the doctor arranges with the head teacher for the cards to be re-distributed to the classes after injection. Children arriving from outlying schools are injected after those from the central school. As the injections are being completed the schools are assembled, all together, in lines according to the ages written on the card. The doctor, together with the head teacher (who will have a much better idea of the real age of the children) spends a few minutes looking at the lines and changing any ages that are apparently wrong. This is desirable for the records, as children and their parents have very little idea of their age, and teachers often take their word for it. In the meantime the assistant is packing up the equipment.

The second visit is made three days later. Tests are read in the individual classes as "positive" or "negative", while the assistant "boils up". One of the teachers records the result on the card, which he has collected from the child. The cards are returned by another teacher to those who are negative reactors, and these are kept on the spot until they are given R.C.G.

For the inoculations a fourth helper is required, to hold the child while the injection is being done into the left shoulder; if this is not done each child moves further away and much time is wasted pulling them back. A single line is formed. The injections are best done standing, except in the smallest children. The speed is somewhat less than with tuberculin tests but about 180 per hour can be done. On this second visit the schools are visited individually.

Sometimes a second school or group may be visited on the first day. In most areas it should be possible to test 500 children in a morning.

The first visit must be made on a Monday or Tuesday, or exceptionally, on a Friday, so as to be able to revisit 3 days later.

C. Organization - general.

Many people are concerned - the Native Education authorities, Mission superintendents (who supervise nearly all the rural schools), head teachers of schools, and Native Health Demonstrators or whom there are one or two to a group of

Reserves or Purchase Areas. Organization should be considered separately for each area to be covered, according to the people who are in the best position to help.

A further complicating factor is the limitation of life of tuberculin and B.C.G. This will be considered first since the arrangements depend upon it. It takes about two weeks for tuberculin to arrive from the S.A.I.M.R. after ordering, and B.C.G. arrives from Copenhagen a week after despatch, 500 doses regularly every 2 weeks.

1. Life of tuberculin.

A small experiment was done on 5 positively reacting adult volunteers. All the tuberculin used had been kept refrigerated since arrival. Tuberculin of 6 different ages was injected in different places down the left arm, which had not previously been used for injections except in one case. The resulting reactions were measured and examined after 3 days.

Results: Size of reactions in millimetres.

	Age of tuberculin -3 months.	5½ weeks.	4½ weeks.	3 weeks.	2 weeks.	1 week
Subject 1	9	12	14	14	16	16
Subject 2	15	16	13	14	15	17
Subject 3	13	17	16	14	13	13
Subject 4	14	13	14	12	11	12
Subject 5	8	13	13	11	12	11

The induration was in general well maintained through the three most recent injections; the others were progressively less firm. Subject 5 showed vesiculation in the test with

3-weeks-old tuberculin. This was about half-way down the forearm and he had previously been injected in this arm; it was therefore presumed that this severe reaction had no significance other than a local skin allergy due to previous injection in the same spot.

It will be seen that the size of the reaction was well maintained with tuberculin as much as 5 weeks old. The 3-months' old tuberculin gave some readings much smaller than the newer material.

Conclusions: For pre-vaccination testing, tuberculin up to 5 weeks old may be used, provided it has been kept refrigerated. For size measurement, it may also be used up to 5 weeks; but if consistency is also under study, 3 weeks is the age limit.

2. Life of B.C.G.

W.H.O. Monograph No. 12 (1953) states: "So long as B.C.G. vaccine is protected from light and kept at near-freezing temperature, potency is lost very slowly - the loss is measurable only over a period of months. As the storage temperature is raised, the rate of loss increases. At 20° C, loss of potency is detected within a few weeks. Light has a similar injurious effect, but it acts much more quickly; indirect daylight produces measurable effects in a matter of hours, and direct sunlight in minutes or even seconds".

Heaf (1955) states "Fresh vaccine..... should be used certainly not later than 14 days after preparation".

In the particular case of vaccine to be used in the

Eastern Region, the ice in the box has invariably melted by the time it arrives in Umtali, and the temperature of the water has risen considerably. To determine the life of this B.C.G. from the day of preparation, therefore, the following experiment was carried out.

At one school, groups of children were given B.C.G. of different ages. Another group were given fresh B.C.G. which had, however, been frozen for a night; this was to determine whether accidental freezing of B.C.G. impairs its effect at all.

Results:

Age of B.C.G.	Tuberculin tests after 6 wks.				Complications
	Positive.	Negative.	Total.	%negative.	
5½ weeks	43	3	46	6.5	2
3½ weeks	39	3	42	7.1	3
1½ wks (frozen)	47	2	49	4.1	3
1½ wks (normal)	142	8	150	5.3	14

The results are entirely negative, showing that provided it is refrigerated after it arrives, and even if accidentally frozen for a short time, B.C.G. is no less effective for at least five weeks.

The latitude thus granted in the time for which both/ tuberculin and B.C.G. may be kept before use is of great help. 500 doses of B.C.G. have to be used up every fortnight, and it is often difficult to anticipate what proportion of children in an area will be negative reactors.

3. Arrangements for the different areas.

The cooperation of the Native Education Department must be sought, and also the consent of the Mission Superintendents. It is intended to cover one complete area at a time, now that the survey stage is over. The first school in an area may be informed and sent the cards through the Native Education Department and Mission Superintendents or through the Native Health Demonstrator, depending which can more easily reach the school. Thereafter cards and instructions can be delivered on the same visit to the area as one for doing tests or B.C.G.'s in another school.

If the overall positive reactor-rate for rural schools is 40%, on the average 830 tests will be done every fortnight, in two lots of two visits as far as possible.

As the setting-up prior to injections takes up much time if the process of injecting is to be efficient, it is important to have as many schools at one place as possible. A central school is selected and the cooperation of the Native Education Department sought to allow children to walk to that school from those in the neighbourhood. Small children cannot however be expected to walk more than about 3 miles, and in such a case it is Mohammed that must move. Where it is necessary to do a second school in a morning, a large number of needles may be boiled up at the first school and taken to the second to save the time involved in boiling; this applies also to the visits to individual schools for B.C.G. inoculations. Alternatively, the

the "age-parade" may be held first, while the boiling and other preparations are in progress.

4. Consent.

Consent has not been sought from parents for this procedure, although no child refusing injection is forced to have it. The obtaining of consent would so enlarge the task as to be quite crippling, and if ethical conduct was adhered to there is little doubt that B.C.G. vaccination, for the present at least, would be doomed.

At one school in an area where a certain creed which eschews all medicine is very widespread, it was understood from the head teacher that 90% of the children were of this religion. Not one protesting voice, however, was raised. The teacher explained that the children go to church, confess that they were injected because it was forced upon them, are forgiven and everything is in order. There is little doubt that these unprotesting children have faith in the medicines which they are receiving, and that to obtain parental consent in such an area would be itself unethical. With the element of force gone the injections can no longer be accepted without breaking the tenets of the religion, and parents would be compelled either to refuse the injections, perhaps against their will, or to go against their religion with considerable upset to their peace of mind. As long as there is no evidence of unrest or discontent as a result of the injections, it is felt that to obtain consent is not only unnecessary but in some areas harmful.

PART III.GENERAL CONCLUSIONS.

We are now presented with the picture of a newly-introduced disease, to which the Native people have only just begun to become acclimatized. Because of its newness, their reaction to it is severe and the fatality-rate is high. It has become widespread in the Eastern districts, as in the other parts of Rhodesia and most of Africa. In rural areas there is a better balance between the people and the disease; in industrial conditions, away from home, it is more likely to get the better of them. Miners suffer from it, but probably no more severely than other industrial workers, because many of them on the larger mines have good food and accommodation and well-regulated working hours. The miners and other industrial workers still carry the disease home with them to the kraals, but at the present time it certainly finds its own way from one person to another in rural areas, independent of returning sufferers.

We know too little about the people. Until we have made a study of them, their religious beliefs and their attitude towards disease and tuberculosis in particular, a satisfactory follow-up and contact-tracing service cannot be achieved. Though it is true that little of such work can be done with the present staff available, the need for such knowledge will increase as tuberculosis control progresses to personal-health-service level.

As regards their attitude towards tuberculosis, it is a newly-introduced disease and not a long-standing one to which much tradition and religious significance have become attached. Kraal dwellers have undoubtedly formed their own theories about the disease, and we may expect to find, on enquiry, that the Nganga has his own special way of curing it; but there will be no deep-founded tribal dogma such as makes it so difficult to deal with many diseases among primitive communities.

An infectious disease introduced among a people who did not have it before presents a special type of problem. As westernization has come to Rhodesia to stay, so has tuberculosis. It must be absorbed into the community and they must be adapted to live with it. Many good arguments have been put forward for a conservative attitude towards diseases such as malaria and schistosomiasis, which have been with the people for several centuries and with which they have established a relationship of comparative mutual tolerance. Tuberculosis, however, has been a sudden intruder, upsetting the balance of nature, and radical measures are justified to restore the status quo.

Prevention - general principles.

In considering the prevention of tuberculosis, "tuberculization" of the community should be regarded as an established fact. We cannot now eradicate completely the disease; our aim must be, as in Britain, to regulate its

transmission so that it passes mildly from person to person without producing clinical disease. The two great factors to be considered in this are: (1) the size of the dose and (2) the resistance of the recipient.

1. The size of the dose and frequency with which it is encountered. A small infection must be the one to render the subject tuberculin-positive, and he must not be repeatedly exposed to infection. The present study on the significance of the nature of the reaction (page 100) indicates that only, or mainly, those who receive a large initial infection will later break down with "secondary" disease. If we can ensure that the initial infection is mild, there is a good chance that a permanent immunity will be established. Two main objects must be pursued to this end:

- (a) the isolation of infectious cases.

Infectious persons are a key point in the epidemiology of tuberculosis; if they can but be pin-pointed and removed from circulation a great decrease in disease will occur. They and they alone are capable of giving the necessary large dose of organisms to other people. Their isolation is, however, impracticable for many reasons. The humanitarian standpoint, backed by public opinion, forbids the imprisonment for life of any individual. On the other hand, the laborious administrative machinery involved in returning sufferers to their own country means a delay of some months during which they are usually kept in hospital, and some even die there. Again, hospital accommodation being limited, it is only those with a fair chance of being cured that can be accepted for treatment. The facilities are now being expanded, and the treatment of all with the remotest chance of cure will mean that more infectious cases are

As things are, however, the fact is that many infectious cases are ultimately returned to their homes, and then we must depend on follow-up in the home by health workers. At present, little can be done by supervision of infectious cases in their homes. The Health Inspector can only visit them once or twice; the Native Health Demonstrator occasionally. Some try to carry out the instructions given for a while; many do not. Most have no conception of the nature of infection and the directions, meaningless to them, are soon forgotten. The subject deserves study from the point of view of the patient and his attitude towards the disease.

(b) The use of B.C.G. It is then upon this that we depend almost entirely at the present stage to prevent transmission of infection. It is discussed below.

The frequency with which the dose of infection is encountered is related to the degree of overcrowding, and can be classed with (2) below for practical purposes.

2. The resistance of the recipient. This is related to adequate nutrition, satisfactory urban housing conditions and amenities, etc., and is a social problem with implications extending well beyond the field of tuberculosis. The subject has been discussed earlier (page 12).

There are two sides to the public health aspect of tuberculosis in practice. The first is the purely preventive - aiming directly to diminish the transmission of the disease.

Measures under this head are the isolation or education of infectious cases and the use of B.C.G. vaccination. The second is the more humanitarian side, whose prime aim is the permanent cure and rehabilitation of the patient. This personal approach, largely carried out by voluntary bodies, has a long-term preventive advantage

in that such sufferers do not become infectious again. It is an expensive and far-sighted procedure, characteristic mainly of advanced countries. It is on the direct preventive, more economical, and sometimes rather inhuman approach upon which we, working among an under-developed community, should concentrate.

Prevention of tuberculosis may be considered either as a nation-wide concern, or from the point of view of the individual health worker in the field. It is proposed to take these two facets in turn.

General consideration of the prevention of tuberculosis.

A. The mines.

1. Recommendations previously made in Southern Rhodesia.

The National Health Service Inquiry Commission, 1945, recommended the medical examination of all mine Africans for phthisis before discharge, by a competent body, and also periodic mass radiography of mine employees. Africans in all forms of employment should be periodically examined, and their registration certificates endorsed accordingly.

The Silicosis Commission, 1938, made recommendations as to methods of keeping dust down in mines. They also suggested the setting up of a Miners' Phthisis Medical Bureau, as in the Union, to aid the early detection of silicosis and tuberculosis. Miners should be weighed every three months, and examined if they lost more than five pounds in that time. All who had worked more than five years in mines should have a clinical examination every six months.

No miner was to be discharged unless certified free of tuberculosis by his medical attendant.

2. Recommendations made in other countries.

A period of breaking-in is required for all new miners to enable them to become accustomed to the new conditions. This fact is emphasized by the Tuberculosis Research Committee (1932), and by Dormer (1946). In the Katanga Mining Union in the Belgian Congo, recruits are kept six weeks doing nothing, and then spend six months in labour camps, before going underground. (De Eriey, 1945)

The use of mass miniature radiography on Native mine labourers in the Union of South Africa is discussed by Retief (1943).

The Tuberculosis Research Committee in the Union, 1932, found the following features needing improvement: travelling conditions, sleeping quarters, amount of rest and times of feeding underground, protection of miners from chilling, and amount of Vitamin A in the diet.

Every effort must be made to keep a high standard of housing for the Africans, especially on the small mines, which employ the greater number. Dormer (1948) stresses this, and also the need to avoid overcrowding. The diet, he goes on to say, must be suitable by custom as well as nutrition. The housing arrangements should contain provision for family life.

Of very great importance is the establishment of a permanent society by providing accommodation for wives

and families on the mines. The social problem presented by the aggregations of single young men found in industry is great. Yet to convince any who are, understandably, tempted to think that a social problem is too vague a thing to tackle, it should be pointed out that this social problem can be measured in terms of life and death. There is a suggestion of this in the high tuberculosis mortality figures (as well as total mortality figures) among the labourers from other countries as compared with Southern Rhodesian workers. A more striking example is described by De Briey (1945) in an article on the policy of social stabilization by the Katanga Mining Union. The position was that a shortage of labour was developing, as there were not enough workers to work the mines as well as keep up the agriculture of the country; and secondly, miners went home often after only a few months of employment, and it was uneconomical to be constantly transporting them to the mines. As a result, wives and families were encouraged to settle at the mines, and facilities were provided for these. The author states that this has been the only effort in Africa to make a permanent society in labour. It has borne fruit, as shown by a great decrease in the death rate (51/1,000 in 1928, down to 4/1,000 in 1939), and an increase in the birth-rate.

B. Urban and rural.

The Public Health Report, 1952, states that the immediate need is for beds, rather than case-finding methods, and

goes on to say: "It has therefore been suggested that a tuberculosis scheme should be instituted and be based on outpatient clinics as an integral part of the Regional Preventive Service, but backed by an immediate increase in beds for tuberculous cases either in special institutions or in wings of existing hospitals. These clinics could do yeoman work in tuberculin testing of the young, in giving B.C.G. to the negative reactors, and in investigating those positive to a standard Mantoux test". The need for better housing and better nutrition is stressed.

The 1943-4 Public Health Report for Bulawayo states the need for special hospitals for the isolation of infectious cases so as to avoid sending them back to the Reserves, and for legal measures for the enforced removal of such cases. Subsequent reports recommend a system of provision for the family when the breadwinner is laid low so that the patient will come for treatment, and improvement of the nutrition and living accommodation of the Africans.

Comment: With reference to these suggestions, one may enlarge upon the need for beds mentioned. Without adequate hospital beds, no contact-tracing can be carried out, a severe handicap to the control of tuberculosis. Mass miniature radiography of particular groups is also severely limited in its usefulness. At the present time it is necessary either to refuse treatment to a great number of

sufferers, or to treat a larger proportion for only about three months.

As regards the tuberculin testing and B.C.G. vaccination of the young in clinics, experience from the present survey suggests that it is impractical to do school children in clinics, as only a very small proportion are in the large towns. As for the voluntary inoculation of pre-school children at clinics, it is thought that would only be successful after a very intense propaganda campaign; the response to the testing and vaccination of industrial recruits has been very poor.

The Public Health Report also speaks of "investigating those positive to a standard Mantoux test". It is suggested on the basis of the results from the E.S.R. and X-ray investigation (page 100) that in the primitive state of the health service as it is today such investigation should be confined to those giving a Mantoux reaction more than 20 mm in diameter. All such reactors should be X-rayed, and arrangements will have to be made for them to be brought in to a town with the necessary facilities. From the 35 negative clinical examinations made on strongly positive reactors to 2 units of K.O.T. (page 79) it is felt that clinical examination of these over-20 mm reactors will not repay the time spent on it. In almost all cases found in this way to have radiological evidence of activity, the disease will be early, and a quiet regime at the school with provision of extra protective foods, will be a fair guarantee

to the child of an uneventful recovery.

To turn to recommendations from other countries, various authors have suggested ways of organizing a tuberculosis service. Dormer (1948) says that case-finding should be done by combining tuberculin-testing with mass radiography of the positive reactors. Sputum testing of all cases found to be diseased will indicate which require isolation. He recommended the division of the country (the Union) into regions, each with a fully-equipped regional hospital providing all treatment, and facilities for rehabilitation. Attached to each hospital would be one or more circulating mass X-ray teams fully equipped for tuberculin testing, mass X-ray and sputum testing as well as clinical examination of suspect cases. The team would investigate those groups of the population most at risk. Each region could have simple isolation facilities in each sub-regional area, for the reception of infectious cases prior to their admission to a regional hospital for active treatment, for the permanent reception of untreatable cases, and for the housing of convalescent cases. Dormer emphasizes the importance of avoiding prolonged mental strain and worry, and lays down that the family of the breadwinner who is in hospital with tuberculosis should be protected against want while the patient is undergoing treatment and rehabilitation. A national, and not a local authority, approach to the tuberculosis programme is, Dormer says, essential.

Haynes (1951) suggested in Kenya the establishment of tuberculosis clinics in large towns, with health workers to find cases. He also said that tuberculin-testing should be done routinely in schools. There would also be African Health Workers to visit cases, trace contacts, and disseminate anti-tuberculosis propoganda.

Davies (1946) describes a hospital-sanatorium which he runs in connection with a chain of tuberculosis dispensaries, independently of the general hospitals. He stresses the need for mixing all cases together irrespective of the prognosis, since this is the only way in which the people will be persuaded to come to the hospital, as they see a fair proportion of the patients recover. The establishment of separate accommodation for the incurable, he says, would acquire a bad reputation for the service. (This is in contrast to the ideas of Dornier and Wiles (1945) who recommended the provision of cheap pavilion wards for the treatable cases, and isolation colonies in the Reserves for the untreatable).

It is not easy to suggest what sort of tuberculosis scheme would be most suitable for Southern Rhodesia. It will probably be best organized on a national basis, and the five regions at present used for public health purposes would be used for the execution of a tuberculosis service. Regional hospitals, with the use of wards in the existing Government hospitals, might be established. These regional hospitals would be fully equipped to give all surgical

and medical treatment for tuberculosis, and would take in cases from the area hospitals and clinics which required such special treatment. Working in conjunction with the regional hospitals would be health visitors (African) whose task would be the tracing of contacts and the following up of cases which have returned home. In the meantime Native Health Demonstrators can be trained to do some of this work.

One of the greatest tasks will be to achieve the cooperation of the Africans in the all-important work of tracing contacts and persuading those who fall ill with tuberculosis to come to hospital. Another will be the dissemination of anti-tuberculosis propoganda. It is for these two ends in particular that we need to know something of the African's inner life: his beliefs, habits of thought, the and concepts of disease, particularly of tuberculosis. We should know also the natural lines of authority, and through which channels we should expect propoganda and influence to percolate most readily. In approaching the concrete problem of tuberculosis, we should not forget the spiritual realms through which we have to pass.

Role of the African in a tuberculosis service.

As much responsibility as possible should be given to the Africans; it is highly desirable that as far as they can they should manage their own tuberculosis service. Such a process would be fraught in the early stages with inefficiencies and mistakes, but in the long run it must

pay to have the African responsible to his own people, and answerable himself for what he does; instead of being responsible to a senior European who has to take the blame, and whose only outlet is to relieve himself in no stringent fashion of his opinion of his junior. As things are, (in all walks of life), there is little stimulus to the African to work hard and set in a responsible fashion.

Vital statistics.

Every effort should be made to improve the vital statistics on Africans. Few of the figures obtained from the public health reports for this study have been of any value. The Tuberculosis Research Committee, 1932, noted the same difficulty in the Union of South Africa and observed that better vital statistics are essential in order to achieve improvement in any branch of public health.

The corner-stone - individual resistance.

Sound physical health is the only sure foundation on which to base a campaign against tuberculosis. This means raising the standard of living of the people, and though it is not directly in the hands of the medical man the need for it must be recognized. Raising the standard of living means, in the towns and on mines, proper feeding, adequate housing, recreation and well regulated hours of work. Until a satisfactory level is reached, we may remove the scum of tuberculosis from the surface of the African pool, but it will keep rising from the bottom. In the Reserves the standard can be, and is being, raised by help

and education in agriculture. Whatever the means, the end is very necessary; and not the least of its functions will be to act as the corner-stone upon which we may build an effective campaign against tuberculosis.

Programme of immediate preventive measures to be carried out by an individual regional health department.

Bearing in mind the object of breaking the cycle of transmission of large doses of infection from one individual to another, this may be stated as follows:

1. Careful follow-up of all infectious cases discharged from hospitals. For this, efficient liaison is required with all the hospitals in the region. The initial enquiry should be done by the health inspector, and subsequent visits paid by the Native Health Demonstrator, who will report to the Health Inspector.

2. Investigation of the contacts of all cases reported. As time goes on and more Health Inspectors and Health Demonstrators become available, this work can be covered more fully. Diseased contacts who are infectious are the ones who interest us at this stage; little can be done for any found to be sufferers from early or non-infectious disease other than to advise them as regards diet and rest. Fortunately perhaps, the Health Inspector's visit will not reveal early cases to him, so that no ethical problem will be created. Those with advanced, infectious disease will be evidently ill, and can be sent for an X-ray. The

hospitals will usually accept those who are curable; the incurable ones will be added to the Health Inspector's follow-up list.

The use of B.C.G. vaccination.

Tuberculin testing and B.C.G. vaccination represent the best method of direct preventive attack on the disease. Underdeveloped countries such as Southern Rhodesia which have been heavily invaded by tuberculosis are considered by some to be the ideal places in which to use B.C.G.

Age of giving B.C.G. It is necessary to decide what policy is to be adopted in the use of B.C.G. Heaf (1955) considers that the best age, in a country where the sensitivity rate is more than 10% by the age of 5, is infancy. ~~upon entering school.~~ The graph on sensitivity rates in school children in Southern Rhodesia (after page 34) shows that the most rapid rise in rates occurs, in boys, from 8 years of age and in girls two or three years later. The more tuberculosis there is in a community, the earlier and slighter is the amount of contact required to produce this rapid rise; for instance in S. Rhodesia it occurs soon after going to school, whereas in Uganda, with less universal sensitivity, it is at the age of about 20 (see page 32) i.e. soon after taking up work. Just before this period, wherever it occurs, is likely to be the best age for giving B.C.G. Another point to consider is the period of greatest danger for contracting tuberculosis. Though school children certainly develop the disease, the maximum

danger appears to be just after taking up industrial employment, i.e. from the age of about 17. This is undoubtedly related to the change of environment, which does not occur when the child goes to the kraal school. However, though a few of such children are to be found in secondary schools and a very few in kraal schools, the great majority go home after leaving the kraal school and are inaccessible to the needle. Another, minor point to consider is the age of maximum conversion by B.C.G., which appears to be in the younger children.

Thus in general it is the younger children whom we wish to protect.

At the same time, consideration must be given to the duration of the protection given by B.C.G. 93% of the children inoculated at Dangare School, and tested at 8 months, were still positive then. W.H.O. Monograph No. 12 (1953) states: "Up to two years after vaccination, there is no evidence in any of our studies of a loss of the allergy produced by standard-strength vaccine". The later follow-up tests of the Umtali Schools will be of great interest.

It has been claimed that B.C.G. only remains effective for 2 - 3 years; for this reason the use of the Vole bacillus, whose effect is longer, should be considered. It has been tried in Northern Rhodesia but found to have a rather high complication-rate. Wells himself, at a recent conference, gave his opinion that the Vole bacillus

needs further trials and should not yet be put to general use.

If the effect of B.C.G. only lasts for two or three years (though the W.H.O. Monograph just quoted suggests that the period is longer) this means that, theoretically, a certain proportion of these inoculated children will acquire a natural infection during these 3 years, but the rest will revert to negative, and we are left with a population of school children aged 10 of whom (as shown in Figure 3) 63% are negative reactors. In other words, we have protected those children for three years from tuberculosis, but beyond that nothing has been achieved. What we may hope for, however, is that the period of protection is really much longer.

Practical considerations. Having decided that the 6 - 8 group of school children is the most important for vaccinating, the question arises how this is to be carried out. In practice, whenever a school is visited (most being kraal schools, up to Std.III) all the children in that school are tested and the negatives vaccinated. The District Inspector of African Schools states that there are about 50,000 children in schools in the region. If 830 children are done every fortnight, (page 125A), and there are 36 school weeks in a year, it will take $3\frac{1}{2}$ years to cover the region once. A child spends, at the most, 5 years in the kraal school, so that by now it will be time to start again.

Another period at which it would be very useful to give B.C.G. is when the adolescent first comes to work in the town. As things are, however, it is impossible to collect such recruits systematically prior to employment. An attempt has been made to inoculate them at a certain place and time by asking firms to send all new employees in the previous month to be tested. Not only is the response to this very poor, but at least 80% are tuberculin positive when tested and very few inoculations are done. (The crude rate for all industrial workers tested was 83% - page 88)

Intended method of determining the effectiveness of B.C.G.

As coverage of the region by the inoculations becomes more complete, a record will be kept of all tuberculosis cases notified who have been tested in this way, and a comparison of natural positive reactors with those given B.C.G., and with any not tested at all, will lead to an assessment of the value of B.C.G. based truly on its protective qualities and not merely on its production of allergy to tuberculin.

Remarks on the system of using B.C.G. in schools.

The method used in this region is that of administering B.C.G. to all school children in the first instance, and then to school entrants. The question which will now be discussed is: is this an effective method of dispensing B.C.G., and if not, can any better be devised?

Certain rough figures may be used for the purpose of argument.

The African population of the Eastern Region is about 400,000. About 50,000 children are in schools. Suppose that a certain fixed proportion of the population are infectious tuberculosis cases. Let us say that the great majority of Mantoux-positive reactors have an immunity to tuberculosis, and that only the highly allergic reactors who had a large initial infection are liable to re-infection. In this way it is the Mantoux-negative reactors who constitute the susceptible population, say 50% of the whole population or 200,000. Let us say that as tuberculosis is still apparently on the increase, each infectious case renders, on the average, 1.1 other persons infectious.

Of the 50,000 school children, let us say that 56% are Mantoux negative (the figure discovered in this survey), i.e. 28,000. Of these perhaps 90% or 25,000 are injected and converted to positive by B.C.G.

Let us say that the effect of B.C.G. lasts 4 years, and that it will take 3 years to complete the inoculation of the whole school population.

Finally, let us say that the average period of survival of an infectious case is one year; though nowadays with the increasing chronicity of Bantu tuberculosis this may be an under-estimate.

In this way 25,000 out of the susceptible population of 200,000, or 12.5%, are protected by the end of 3 years, and this protection continues over another year, i.e. the period required to exhaust the average of the infectious cases in

existence. Since the number of susceptible subjects is in this way diminished by 12.5%, the number of effective contacts will diminish by the same amount; in other words, by the end of the 4th. year, the number of infectious cases is diminished by 12.5%.

As the years go on, supposing that the inoculation of children in the first 3 school years is kept up and repeated every 3 years, there will always be this proportion of children kept protected, or rather, since the effect of B.C.G. is being said to last 4 years, $12.5 \times 4/3 = 16.7\%$.

In addition, about 20% of the protected children will develop natural infections while protected by B.C.G. (see Figure 3), and these may be considered to have a permanent immunity; so that each 3 years another $20\% \times 12.5\% = 2.5\%$ will be subtracted from the total susceptible population, until after a period of decades a maximum of 20% permanently protected in this way is reached.

With this policy continued over twenty-five or thirty years, therefore, we may expect the proportion of susceptibles protected to increase, from 17% after 3 years, to 37% after 30 years, and the number of infectious cases will diminish likewise.

In spite, therefore, of adopting assumptions generous to B.C.G., we find that it can produce no greater diminution of infectious cases than 37%, over an indefinite period, if the present method is used. Tuberculosis will be reduced, but it will undoubtedly continue to flourish. It is

questioned whether this method of using B.C.G. is really worth while.

The alternative is one which requires a great deal of organization and inter-departmental cooperation, and a considerable increase, at least temporarily, of health staff. It consists of testing and injecting the entire population within the space of two years. If 85% of the (say) 200,000 negative reactors are thus protected successfully, and B.C.G.'s effect lasts for three or four years, the number of infectious cases at the end of that time will be diminished to 15%.

Though many of the figures used in the above discussion are very fanciful, they do serve to make an adequate comparison between the two systems of vaccination.

The second method is too heroic for use at the present time, but some such programme should be considered, as a far more effective alternative to the vaccination only of school children.

Use of strongly positive reactions for case-finding.

This subject is discussed above (page 107). There is no hospital accommodation available in the Eastern region to cope with sufferers discovered by this method; yet children attending a school, constantly under the eye of a teacher, have a much better chance of resting and adopting other suitable therapeutic measures than the ordinary run of Africans.

Other Aspects.

Other aspects of tuberculosis control - after-care and occupational therapy are important from the preventive standpoint in that by ensuring the cure of cases they diminish the incidence of subsequent breakdown into an active and infectious case. They are all facets which in Southern Rhodesia are in their infancy; but they are growing, and will come in time to play their part in the effective prevention of the disease.

APPENDIX I.HISTORY OF THE AFRICAN PEOPLE BEFORE 1890.

There is evidence that a race existed in South Africa (i.e. Africa south of the Zambesi and Kunene rivers) before the Bushmen, but it is clear that the latter had been in possession for some thousands of years before the advent of the Hottentot and Bantu races approximately one to two centuries before the discovery of the Cape of Good Hope by Europeans in 1492 (Theall, 1919). Over this period the Bushmen had occupied the whole of the African continent as well as large tracts of Europe and Asia, but by the year 1500 they had been entirely destroyed in all parts north of the Zambesi River, except for a few small isolated groups. By the time the Europeans began to colonize South Africa, the Bushmen had been driven by their stronger and more intelligent opponents into the more arid and mountainous central parts of the country, from which, however, they were still waging a form of guerilla warfare on the intruders. The Hottentots and Bantu, following the precedent of other conquering races further north, often saved the young females from the general destruction of the Bushmen, and among their descendants today there is doubtless a small admixture of Bushman blood. At the present time there are very few Bushmen remaining in South Africa,

and these are mainly in the region of the Kalahari Desert towards the west coast.

The Hottentots appear to be the product of a union between a band of ancient Egyptians, perhaps soldiers, who migrated to the region now known as Somaliland, and the Bushman women whom they captured there. It is thought that they migrated from here to the region of the Great Lakes, and after an unknown period of time were forced to move south-west from here by the wave of Bantu people who began to move south from Asia minor, occupying the Lake Region and advancing down the East coast. Before reaching the Zambesi valley, the Hottentots were forced to move towards the west coast because their cattle were killed off by the tsetse-fly in that region, and their subsequent course was down the west coast to the region south of the Orange river. For this reason they were never in Southern Rhodesia. Their arrival at the southern shore of Africa must have preceded that of the first Portuguese explorers by less than two or three centuries.

The Bantu are the race who today occupy almost the whole of the southern part of the African continent, and of whom all the tribes living in Southern Rhodesia are subdivisions. Their forbears entered North-Eastern Africa at some time after the Hottentots, and became intermingled with the races who were already living in the continent; the hamitic family in the north, negroes along the south border of the Sahara, various lighter coloured communities of Asiatic

blood south of the Red Sea, and Bushmen occupying the rest of the continent. The Hottentots would then be somewhere between the Gulf of Aden and Lake Tanganyika.

Theall even suggests that at this early stage Arabs and Indians may have been mining gold in Rhodesia, and speculates at length on the mysterious visitors who in ancient times must have visited Southern Africa and constructed Great Zimbabwe and the other mines and fortifications found between the Zambesi and Sabi rivers. In any case he considers that the wealth of Solomon the king of Israel, brought in Phoenician ships by collaboration with Hiram the king of Tyre (and recorded in I Kings, Chapters 9 and 10), was obtained on the East Coast of Africa. The Phoenicians, who could have given valuable information about the people of that part, appear to have been very security-conscious and left no record of their journeys. In the first century a.d., the Greeks found Bantu as far south as 7°S, and there were also Arabs and their Indian servants on the coast, trading and intermarrying with the Bantu. In the second century the Bantu are recorded at 10°S, and by the 10th. century an Arab writer, who describes them accurately, announces their presence south of the Zambesi, in the northern part of what is now Mashonaland and the territory between it and the coast.

This people had migrated from the country adjoining the trading station of Rhapta, somewhat south of Zanzibar, and had the blood of Arabs and Persians, Indians and even

Greeks mixed with that of Bantu in their veins. They were thus of superior intelligence, as illustrated by the feat of crossing the Zambesi. When found by the Portuguese in 1508, they were mining and smelting gold for trading with the Mohammedan community at Sofala on the coast, whose blood was a mixture of Arab and Bantu. They called themselves Ma Karanga, and were probably the ancestors of the tribe that bears that name today. They did not go farther south than the Sabi river, and remained near the coast.

It was some centuries before more groups followed them across the Zambesi. The Bantu who passed through or settled in Southern Rhodesia consisted of two groups: There had been much overcrowding and warfare in the region of the Great Lakes, causing some of the Bantu to move right over towards the west coast. As the Western parts became more occupied, groups began to drift southward from the Great Lakes and down the east coast; by degrees they crossed the Zambesi and found their way farther south to what are now the Union of South Africa and Bechuanaland. They included the Basuto and Bavenda tribes. Before the beginning of the eighteenth century these were followed over the river by a people who were the ancestors of most of the tribes now known collectively as the MaShona. This tribal group had come from somewhere west of Lake Tanganyika, and they remained until driven to the north-east by raiding-parties sent in their direction by Mzilikaze about 1834.

Among these tribes were the Va Rozwi, who exercised to a small degree the authority of a ruling clan until the invasion of the AmaSwazi and AmaNdebele (Matebele). They crowned their kings as rulers of the Mashona, and claimed direct contact with the Mwari, or God of the Cosmos. They evidently had a higher culture than the rest, and appear to have established almost a State Government, remaining on friendly terms with their subject tribes. These tribes were disunited, fighting amongst themselves, and they also almost completely exterminated the Bushmen who had lived in the country. Their clans continued to migrate into Southern Rhodesia until the end of the eighteenth century. They differ from the MaKaranga in personal appearance, having coarser features and being blacker in colour and somewhat stouter in build.

The second group came from the region of the Guinea coast in the north-west, crossing the Zambesi during the fifteenth and sixteenth centuries, and passed through Southern Rhodesia to the south-east coast of South Africa, so that their route of migration crossed that of the first group. On reaching the coast, some of the tribes moved southwards; others northwards, including the Tongas, Shangaens, and Swazis. The Shangaens (AmaShangana) settled in the south-east corner of Southern Rhodesia in 1866, but were robbed of their fighting power by the Portuguese. The Ama Swazi also came into the country, conquering the VaRozwi, but after only a few years they

passed on to form the core of the ANgoni of Nyasaland and Northern Rhodesia. A group who did not move north or south but remained in the region of Natal were the Zulus. In the destructive wars of Tchaka early in the nineteenth century, the AmaNdebele, a member of this group closely related to the Zulus, were driven under their chief Mzilikaze westward. Encountering Boer emigrants moving northwards from the Cape Colony, they turned to the north and occupied the south-western part of Southern Rhodesia. They were a warlike and highly disciplined nation, and the Mashona tribes were quickly subjugated or expelled, some of the women however being kept, so that now there is an admixture of Mashona blood in the Matebele nation.

In the meantime the MaKaranga had suffered a succession of disasters. For several centuries they had continued their profitable trade in gold and ivory with the Mohammedan community at Sofala, and when the Portuguese formed their first settlement on the south-eastern coast, the MaKaSanga occupied the territory now called Mashonaland and the seaboard between the Zambesi and Sabi rivers. Before the commencement of the eighteenth century the tribe was broken up by war, and after this they suffered severely at the hands of the ANgoni from Nyasaland in the north, and also the Shangaans moving north from Natal. With the flight of the MaShona eastward from the Matebele, the deflated MaKaranga were

further molested by these, and finally they and the MaShona alike were brought under subjection by the Matebele. They are now included in the MaShona group.

In 1890 when Rhodes' column entered the country and set up Fort Salisbury, the Matebele were occupying mainly the south-western part around Bulawayo, where their chief, Lobengula (son of Mzilikaze) had his capital. They were a strong, warlike tribe, holding sway over the neighbouring MaShona and MaKaranga.

These two groups are now together called the MaShona; but it is clear from the above that a differentiation should be made between the MaKaranga on the one hand, and the many little groups that make up the remainder on the other.

Of these latter, the three main subdivisions are the VaSezuru (the V in these names is implosive, and is correctly written U), the VaManyika, and the MaKorekore. Thus the Mashonaland of today may be divided into four parts according to the tribe principally occupying it: the Fort Victoria region peopled by MaKaranga (or VaKaranga as they call themselves), the Salisbury region occupied by the VaSezuru, the Umtali region containing the VaManyika, and in a large area south of the Zambesi, the MaKorekore. There are also many smaller tribes related to the last three groups, including the VaRozwi in the Sezuru area.

(For distribution of tribes, see map, Appendix II.)

The history of the MaKaranga is of particular interest.

Today they are a downtrodden, pitiful nation, looked upon by black and white alike as the most stupid and indompetent of the tribes in Southern Rhodesia. Yet they are people whose Empire the Portuguese explorers described in glowing terms, with its King, the Monomotapa, in his great palace. It was they that had the initiative to cross the Zambesi centuries before their peers, and to dig and trade gold. They have even been credited by one writer (Bullock, 1950) with the construction of Great Zimbabwe. In spite of their present state, in the future there is perhaps more to be hoped for from them than from any other tribe in the colony. Theall (1919) writes: "There is no other tribe in South Africa which has so many individuals bearing traces of Arab, Persian and Indian blood as the MaKaranga, which is due to the long continuance of Asiatic intercourse with them in past times. All who have dealings with them state that, though now spiritless and degraded through constant strife and oppression during more than two centuries, they possess greater latent power of advancement, especially in mechanical arts, than any other Bantu in the country (South Africa)".

Other tribes on the borders are:

North and north-east: VaTawara, VaChikunda, VaKude. These peoples, descended from the first group of immigrants, live in the remote Zambesi areas, and differ somewhat from the average MaShona.

South-east: BaHlangwe and VaNdawu, both members of the Mashona group who have mixed with the invading Shangaans. South: the BaVenda and WaRemba, in the Limpopo valley. These people are advanced. They practice circumcision and show suggestions of Semitic, probably Arabian, culture on Bantu customs. Their language is different from that of the other tribes. They may have been the "Moors" found by the Portuguese at Sofala.

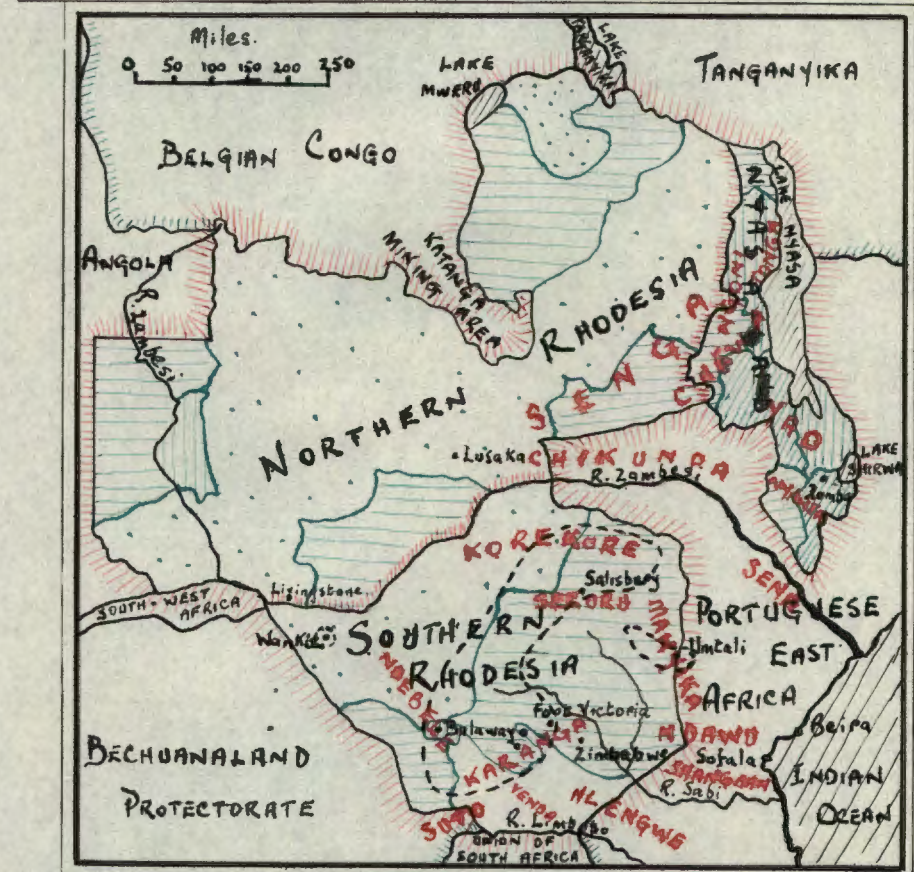
Other tribes living near Southern Rhodesia, from whom members are commonly recruited for the mines, include the ANgoni and Yao from Nyasaland, who were formed in part at least from a nucleus of AmaSwazi from the south. Tribes of Group I yielding many mine recruits are: the Chewa, Nyanja and Tonga from Nyasaland; the Senga from Northern Rhodesia; and from Portuguese East Africa, to the north the Sena and farther south the VaNdawu. From Portuguese East Africa also come many AmaShangana (of Group II).

According to the Rhodesia-Nyasaland Royal Commission (1939) under Lord Bledisloe (see map Appendix II), the population of Nyasaland is very much denser than that of Southern and Northern Rhodesia. In Nyasaland almost the whole country then had more than 25 persons per square mile, and a large part to the south had more than 50; whereas in Southern Rhodesia 25 was only exceeded in a very small area around Bulawayo. Present-day estimates give much higher levels for Southern Rhodesia and probably also for Nyasaland.

The Commission published with their report maps showing the tribal distribution and distribution of population in the Rhodesias and Nyasaland. The main features of these are shown in the map, Appendix II.

APPENDIX II.

MAP OF THE FEDERATION OF CENTRAL AFRICA
SHOWING TRIBAL DISTRIBUTION, POPULATION DENSITIES,
AND BOUNDARIES OF SOUTHERN RHODESIA MINING AREAS.
 (AS IN 1939)



KEY.

- Federal Boundary
- Intra-Federal Territorial boundary
- Extra-Federal Territorial boundary
- Boundaries of S.R. Mining Areas
- Tribes

-
-
-
-
- CHENWA

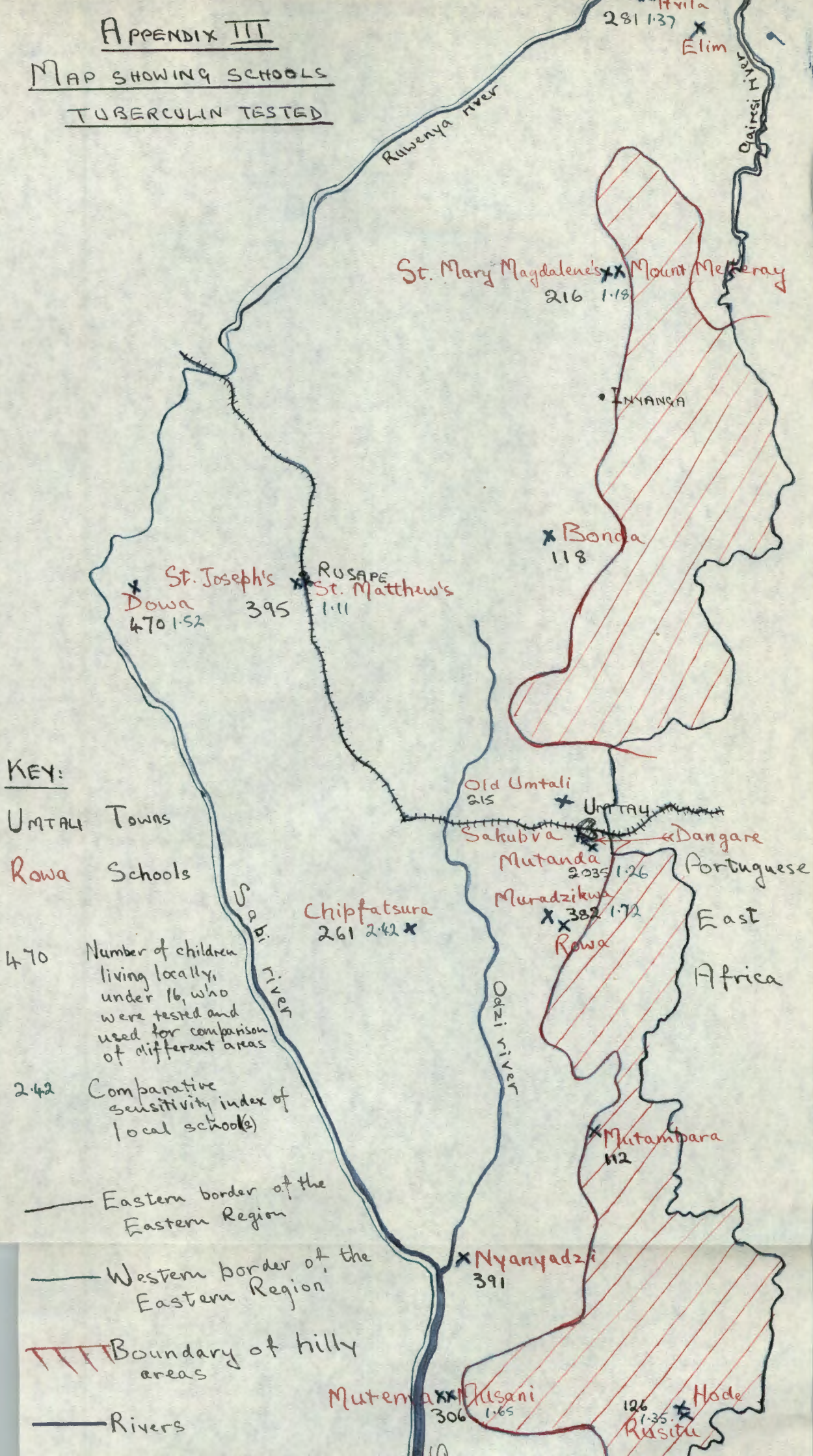
- Population densities:-
- Up to 5 per sq. mile
 - 6-25
 - 26-50
 - Over 50



APPENDIX III

MAP SHOWING SCHOOLS

TUBERCULIN TESTED



Appendix IV: Tables.

TABLE A.

Totals by ages and sexes. School children and teachers - 5 T.U.

Age.	Positives.			Negatives.			Total.			% positive.		
	B.	G.	All	B.	G.	All	B.	G.	All	B.	G.	All.
5	2	-	2	1	-	1	3	-	3	-	-	-
6	13	16	29	60	54	114	73	70	143	18	23	20
7	28	47	75	118	143	261	146	190	336	19	25	22
8	43	53	96	180	173	353	223	226	449	19	23	21
9	66	57	123	136	147	283	202	204	406	33	28	30
10	108	80	188	152	165	317	260	245	505	42	33	37
11	122	66	188	146	141	287	268	207	475	45	32	40
12	157	117	274	208	213	421	365	330	695	43	35	39
13	170	134	304	181	160	341	351	294	645	48	46	47
14	188	124	312	156	128	284	344	252	596	55	49	52
15	160	92	252	118	70	188	278	162	440	58	57	57
16	119	51	170	59	32	91	178	83	261	66	61	65
17	76	11	87	27	12	39	103	23	126	74	-	69
18	61	5	56	27	5	32	78	10	88	65	-	64
19	34	1	35	22	1	23	56	2	58	61	-	60
20	29	2	31	6	2	8	35	4	39	83	-	79
25	56	5	61	12	4	16	68	9	77	82	-	79
30	24	4	28	4	5	9	28	9	37	-	-	76
35	7	5	12	1	1	2	8	6	14	-	-	-
40	5	3	8	5	1	1	5	4	9	-	-	-
45	-	-	-	-	-	-	-	-	-	-	-	-
50	1	1	1	1	-	1	1	-	1	-	-	-
All	1459	873	2332	1614	1457	3071	3073	2330	5403	47	37	43

TABLE B.

Standardization for ages of schools tested with 5 T.U. of O.T. ages 16 and below.

Age.	Stand-ard rates	Umtali		Rusape		Hofe		Mitema		Rowe		Chipfat-sura		Dowa		Mount Malleray		Inyanga North		
		Schools.	Pop.	Exp. Pos.	Schools	Pop.	Exp. Pos.	Schools	Pop.	Exp. Pos.	Group	Pop.	Exp. Pos.	Group	Pop.	Exp. Pos.	Group	Pop.	Exp. Pos.	Group.
6	6.25	39	2.4	5	0.3	13	0.8	16	1.0	9	0.6	16	1.0	9	0.6	1	0.1	8	0.5	
7	10.0	159	15.9	10	1.0	24	3.4	33	3.3	20	2.0	26	2.6	7	0.7	2	0.2	19	1.9	
8	15.1	229	34.6	13	2.0	25	3.8	34	5.1	31	4.7	38	5.7	17	2.6	1	0.2	23	3.5	
9	17.2	206	35.4	9	1.5	33	5.9	24	4.1	33	5.7	33	5.7	18	2.1	2	0.3	23	4.0	
10	19.1	232	44.3	14	2.4	35	6.7	48	9.2	33	6.4	50	9.6	28	5.4	2	0.4	27	5.2	
11	38.2	203	77.6	12	4.6	32	12.2	37	4.1	20	7.6	58	22.1	15	5.7	5	1.9	39	4.9	
12	37.1	332	123.2	16	5.9	53	19.7	48	7.8	33	12.3	54	20.1	28	10.4	10	3.7	34	12.6	
13	34.7	249	86.5	9	3.1	41	14.2	46	16.0	25	8.7	72	25.0	11	3.8	26	9.0	39	13.5	
14	29.5	209	61.7	20	5.9	31	9.2	39	11.5	18	5.3	65	22.2	4	1.2	45	13.3	29	8.6	
15	34.6	129	44.6	13	4.5	18	6.2	32	11.1	30	10.4	40	13.8	1	0.3	58	20.0	21	7.3	
16	54.0	48	25.2	5	2.7	1	0.5	25	13.5	9	4.9	18	27	-	-	64	24.5	19	10.3	
Total Exp. Pos (= Index no. of positives)		552.1		34.2		82.6		106.7		68.7		134.5		33.8		83.6		82.3		
Observed pos.		696.0		46.0		136.0		163.0		166.0		204.0		40.0		99.0		113.0		
O.S.I		1.26		1.11		1.65		1.72		2.42		1.52		1.18		1.18		1.37		

ADDENDUM TO TABLE B.

Derivation of formula for Comparative
Sensitivity Index (C.S.I)

In the indirect method of standardizing:

$$\text{Standardizing factor (S.F)} = \frac{\text{Rate \% of standard}}{\text{Index rate (I.S.R)}}$$

$$\text{Standardized sensitivity rate (S.S.R)} = \text{S.F.} \times \text{crude S.R.}$$

$$= \frac{\text{crude S.R.} \times \text{rate \% of standard}}{\text{Index S.R.}}$$

C.S.I.

$$= \frac{\text{S.S.R.}}{\text{Rate \% of standard.}}$$

$$= \frac{\text{Crude S.R.} \times \text{rate \% of standard}}{\text{Index S.R.} \times \text{rate \% of standard}}$$

$$= \frac{\text{Crude S.R.}}{\text{Index S.R.}}$$

$$= \left\{ \begin{array}{l} \frac{\text{Crude positives}}{\text{Total positives}} \times 100 \\ \frac{\text{Index positives}}{\text{Total positives}} \times 100 \end{array} \right.$$

$$= \frac{\text{Crude positives} \times \text{total positives} \times 100}{\text{Index positives} \times \text{total positives} \times 100}$$

$$= \frac{\text{Crude positives.}}{\text{Index positives.}}$$

TABLE C.

Age and year of employment and tuberculin sensitivity.

Standardised for (a) 1955
or (b) other year of employment %

No year All
known. years.

1948 1949 1950 1951 1952 1953 1954 1955

73.3 73.67

85 31 116 469 104 573 230 55 285 173 22 195 73 15 88 57 7 64 28 3 31 40 40 3 3 1158 297 1395 83%

Age.	1955	1954	1953	1952	1951	1950	1949	1948	No year All known. years.	Standardised for (a) 1955 or (b) other year of employment %
15-15	23	45	14	2			1		85	73.3
	13	17	1						31	
Total	36	62	15	2			1		116	
- 20	116	198	114	32	6	2	1		469	62.10
	33	36	25	8	1	1	1		104	
Total	149	234	139	40	7	3	1		573	
- 25	52	98	38	20	9	7	3	1	230	80.66
	16	22	13	2	1	1	1		55	
Total	68	120	51	22	10	8	3	2	285	
- 30	43	57	35	15	4	14	5	2	173	88.77
	7	8	3	1	1	1	1		22	
Total	50	65	38	16	5	15	6	2	195	
- 35	18	24	15	4	2	4	1	2	73	83.0
	5	4	3	1	1	1	1		15	
Total	23	28	18	5	3	5	2	3	88	
- 40	5	24	10	6	3	4	4	1	57	87.87
		3	1	2	1	1	1		7	
Total	5	27	11	8	4	5	5	1	64	
- 45	4	7	5	3	4	2	2	1	28	89.65
	1	1		3	1	1	1		3	
Total	5	8	5	6	5	3	3	1	31	
Over 45	3	23	6	2	4	2	2	1	40	100
Total	3	23	6	2	2	1	2	1	40	
No Age estimated	1	1		1					3	
All	265	477	237	85	30	34	19	4	1158	
Ages	75	91	46	14	4	5	2	7	297	
Total	340	568	283	99	34	39	21	4	1395	
Standardized Sensitivity rates %	78	84	84	86	83	87	90	84	83%	
	79	85	84	86	86	84	86	86		

TABIZ D.
Tribe, country of origin, and tuberculin sensitivity.

P.E.A. Tribes.

S.R. Tribes.

Year of Employment.	P.E.A. Tribes.										S.R. Tribes.					Mean Variation.
	Ndau	Shangaan	Sena	Malyika	Other	ALL P.E.A.	Midema	Ndau	Karanga	Moosa	Mjindvi	Malyika	Other	ALL S.R.	S.R. + P.E.A.	
Before 1954	26	13	155	17	43	254	8	13	7	14	22	33	43	140	394	
1954	3	5	16	3	5	32	1	5	1	5	7	12	5	36	68	
Variation	29	18	171	20	48	286	6	18	8	19	29	45	48	176	464	
% Pos	90	72	91	85	90	88.9	89	72	88	74	76	73	90	79.6	85	
1954	5	13	6	0	5	253	4	13	3	11	9	12	5	211	464	
Variation	17	22	145	21	48	31	17	19	24	20	20	53	58	55	86	
% Pos	5	1	12	4	9	284	5	13	3	1	10	10	13	266	550	
1955	22	23	157	25	57	89.0	22	32	27	21	30	63	71	79.3	84	
Variation	77	96	92	84	84	77	77	59	89	95	67	84	82	79.3	84	
% Pos	7	12	8	0	0	142	7	25	5	11	17	0	2	115	257	
1955	21	6	78	14	23	30	22	4	14	10	10	16	4	42	72	
Variation	3	4	12	7	4	172	5	7	2	5	2	9	12	157	329	
% Pos	24	10	90	21	27	82.6	27	11	16	12	12	25	53	73.2	79	
1955	87	60	87	67	85	2	81	36	87	83	83	64	77	73.2	79	
Variation	8	19	8	12	6	689	43	8	16	15	15	2	142	466	11.92	
ALL YEARS	64	41	378	52	114	93	47	36	45	52	102	102	142	466	466	
% pos	11	10	40	14	18	742	11	25	9	19	31	31	30	133	133	
1955	75	51	418	66	132	87.4	58	19	51	71	133	133	172	599	599	
Variation	85	80	90	79	86	87.4	81	59	88	73	77	77	83	77.8	77.8	
% pos	85	80	90	79	86	87.4	81	59	88	73	77	77	83	77.8	77.8	

TABLE E.

Reaction Sizes. (5 T.U. of O.T.)

School.	Total Nega- tive.	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	Total Positive.
<u>L. Mlog</u>																														
Dova	122	49	6	19	25	12	11	6	16	16	24	19	6	6	6	13	6	3	3	3	-	1	1	1	1	1	1	1	130	
Mitanda	140	74	15	9	18	19	5	6	17	17	13	13	16	17	14	13	10	8	12	4	1	1	1	5	-	1	1	1	169	
Dangare II	120	73	15	13	13	1	5	1	3	5	3	6	5	4	2	4	5	5	3	3	-	-	-	-	-	1	1	1	50	
Rusitu	50	19	2	6	6	14	3	3	7	6	13	14	8	6	9	6	5	4	4	4	2	2	-	-	-	-	-	-	93	
Hode	51	39	1	3	9	7	2	3	3	4	1	3	2	1	-	2	2	1	1	1	1	2	-	-	1	-	-	1	28	
Mt. Selinda (Senior)	60	32	6	3	8	9	2	2	4	14	17	21	12	25	16	9	12	11	12	10	4	2	1	1	1	1	1	1	174	
Mt. Selinda (kraal)	45	23	7	7	4	3	1	1	1	-	2	2	2	-	2	2	2	-	1	1	3	-	-	-	-	-	1	1	21	
<u>ALL MALES</u>	588	299	52	60	83	65	29	22	51	62	73	78	51	59	49	42	32	36	26	12	6	7	3	2	2	1	1	1	665	
<u>2 Females.</u>																														
Dova	147	45	14	28	29	25	6	2	4	9	14	5	6	11	7	1	2	4	5	2	4	1	1	1	2	2	1	1	77	
Mitanda	156	73	18	30	21	6	8	3	5	10	13	8	12	8	19	8	12	9	3	3	1	2	-	-	2	1	1	1	120	
Dangare II	135	81	11	18	12	9	4	-	4	4	7	2	2	6	6	2	4	3	3	3	2	3	2	1	3	2	1	1	43	
Rusitu	21	10	2	5	4	3	2	-	7	1	2	3	2	-	3	2	1	-	1	1	3	2	-	1	-	-	-	-	29	
Hode	28	10	4	3	6	3	2	1	1	1	1	2	1	2	2	2	4	3	2	1	1	1	1	1	1	1	1	1	24	
Mt. Selinda (Senior)	41	24	4	1	5	7		1	1	3	2	1	3	1	1	2	1	6	1	1	1	2	1	1	2	1	1	1	26	
Mt. Selinda (kraal)	52	28	6	7	6	3	2	-	4	1	3	1	1	3	1	1	1	2	3	3	2	2	2	3	3	1	1	1	21	
<u>ALL FEMALES</u>	580	271	59	22	83	53	22	6	26	26	43	22	23	28	38	17	23	22	20	14	15	5	4	3	3	1	1	1	340	
<u>BOTH SEXES</u>	1168	573	111	152	166	118	51	28	77	88	116	100	74	87	87	66	65	54	56	40	27	11	11	6	5	3	1	1	2	1005

underlining indicates a peak point.

TABLE F.

Size of reactions from heated tuberculin compared with those from normal tuberculin.

<u>Reaction size</u>	<u>Negative reactors</u>	<u>Positive reactors</u>	<u>TOTAL.</u>
+ 8	1		1
+ 7			
+ 6			
+ 5		2	2
+ 4	2	2	4
+ 3	3	5	8
+ 2	3	9	12
+ 1	6	15	21
0	16	11	27
- 1		8	8
- 2	1	3	4
- 3		1	1
- 4		1	1
- 5			
- 6			
- 7			
- 8			
<hr/>			
TOTAL	32	57	89

References.

- Allan, P.: Tuberculosis survey of the Native Territories of the Union of South Africa. Included in the report of the Tuberculosis Research Committee of the South African Institute for Medical Research (q. v.)
- Allen, F.J.: Journal of the Medical Association of South Africa, 1927, vol.1, 554-9
- Bullock, C.: The Mashona and the Matebele. 1950. Cape Town: Juta.
- Central Mining - Rand Mines Group: Health Department Report for 1949.
- Clark, M.: East African Medical Journal, 1951, vol.28, No.9, 355-79.
- Cummins, S.L: Bulletin of Hygiene, 1944, vol.19, No.11, 837. Reviewing the paper by Dormer, Friedlander and Wiles (q. v.)(Ref.from p.18)
- Cummins, S.L: Comments made in the Report of the Tuberculosis Research Committee in the Union of South Africa, 1932. (q.v.)(Ref.p.15)
- Davies, H.N: East African Medical Journal, 1946, vol.23, 194-209.
- Davies, H.N: Tubercle, 1952, vol.33, No.4, 98 - 106.
- De Briey, P: International Labour Review, 1945, vol.52, 335.
- Dormer, B.A: Proceedings of the Transvaal Mine Medical Officers' Association, 1948, vol.27, 63.
(Ref.from p.132)
- Dormer, B.A: South African Medical Journal, 1948, vol.28, 82. (Ref.from p.136).
- Dormer, B.A., Friedlander, J., and Wiles, F.J.: Proceedings of the Transvaal Mine Medical Officers' Association, 1943, vol.23, No.257, 71-114.
- Dormer, B.A., and Wiles, F.J.: South African Medical Journal, 1946, vol.20, No.10, 262-5.

References - continued.

- Gampel, B.: South African Medical Journal, 1952, vol. 26, No.9, 170-4.
- Gilmour, W.S.: East African Medical Journal, 1952, vol.29 381-402. Quoted in Tuberculosis Index, 1953, vol.8, no.3, 249.
- Griffiths, J.M.: and Kreher, O.I.B: South African Journal of Clinical Science, 1952, vol.3, 130-42.
- Guld, J.: Acta Tuberculosea Scandinavica, 1954, vol.30, Nos.1/2, 16-36; quoted in Bulletin of Hygiene, 1955, vol.30, No.4, 293.
- Haynes, W.S.: Tuberculosis in Kenya. 1951. Nairobi: Government Printer.
- Heaf, F.R.G.: Lancet, 1955, Feb.12, 315-20; quoted in Bulletin of Hygiene, 1955, vol.30, No.5, p.408.
- Kaplan, C.J.: British Journal of Tuberculosis, 1952, vol.46, No.4, 209-13.
- Kayne, G.G.: The control of tuberculosis in England. 1937. London: Oxford University Press.
- MacVicar, N.: South African Medical Journal, 1935, vol.9, 543-5.
- National Health Service Enquiry Commission, Southern Rhodesia. 1945. Salisbury: Government Stationery Office.
- Public Health Reports, Bulawayo. 1935-6 to 1951-2.
- Public Health Report, Midland Region, Southern Rhodesia. 1953.
- Public Health Reports, Southern Rhodesia, 1906 to 1952.
- Read, Margaret: International Labour Review, 1942, vol.45, 605.
- Registrar-General's Decennial Supplement for England and Wales (Occupational Mortality), 1930-1932.
- Registrar-General's Statistical Reviews of England and Wales, 1931 and 1951.

- Retief, F.: Proceedings of the Transvaal Mine Medical Officers' Association, 1943, vol.22, No. 250, 181-6.
- Rhodesia-Nyasaland Royal Commission Report. 1939. London: His Majesty's Stationery Office.
- Silicosis Commission, Southern Rhodesia. 1938. Salisbury: Government Stationery Office.
- South African Institute for Medical Research, Annual Report, 1954, p.26.
- Theall, G.McC.: A history of South Africa. Vol.1: Ethnography and conditions of South Africa before 1505. 1919. Second edition. London: George Allen and Unwin, Ltd.
- Thompson, B.C.: Tubercle, 1955, vol.36, No.1, 18-20; quoted in Bulletin of Hygiene, 1955, vol.30, No.4, 294.
- Tuberculosis Research Committee: Tuberculosis in South African Natives. 1932. Johannesburg: South African Institute for Medical Research.
- Whitby, Sir.L.E.H. and Britton, C.J.C: Disorders of the Blood. 1947. Fifth Edition. London: Churchill.
- Wilcocks, C.: Journal of the Royal Sanitary Institute, 1953, vol.73, no.5, 480-2.
- Wood, S.R.: British Journal of Tuberculosis, 1948, vol. 42, 38-45.
- World Health Organization Monograph No.12, 1953. Geneva: World Health Organization.

Tuberculosis in the Bantu of Southern Rhodesia

by

D. H. Shennan

S U M M A R Y

S U M M A R Y.

PART I. Review of the relevant literature.

1q The home life and social background of the Bantu.

The MaShona have a patriarchal tribal system based on their belief in the inheritance by the son of the father's spirit. Government is democratic and through the family heads. Religion plays an important part, and has both socializing and debasing elements. Disease is regarded as having a supernatural origin, and the attitude towards it is fatalistic.

A severe social upheaval with resulting maladjustments both in the towns and in rural areas has resulted from the colonization of the country by the British. A reorganization of urban locations is required, and Africans both urban and rural should be encouraged to govern themselves.

II. The special nature of tuberculosis in Africans.

Under natural conditions the usual acute form of tuberculosis found in the Bantu under industrial conditions does not occur. Even under industrial conditions, tuberculosis has recently tended to become more chronic in the African. Whether active disease occurring during employment is due to the original or a secondary infection is a matter of dispute. Racial differences in reaction to tuberculous infection are probably less important than

environmental changes affecting industrial workers.

III. The history of tuberculosis in Southern Rhodesia.

It is unlikely that there was tuberculosis before 1890. The subsequent course of the disease is traced.

IV. Tuberculin sensitivity.

Most of Southern Rhodesia has been shown to be well "tuberculized", both in town and country, as have most other parts of Africa. On going into industry, those who are tuberculin-positive are more likely to develop the disease than are the tuberculin-negative. Strong positive reactors have greater tendency to develop subsequent disease.

V. Morbidity and mortality rates.

Although urban mortality rates seem to be somewhat lower than in the Union, tuberculosis is a serious problem in the towns of Southern Rhodesia, and it has also gained a firm hold on the country districts.

VI. The epidemiology of tuberculosis.

In scattered isolated peoples, tuberculosis when introduced tends to die out. In urban areas it has a high endemicity and a high disease rate. When it is introduced to rural areas, overcrowding produces a high endemicity, and poor nutrition and living conditions a high active disease rate. On going into industry, those from areas of high endemicity have more resistance to the development of

tuberculosis than those from areas of low endemicity. Towns and mines are the foci of infection, and from them it spreads to the Native Reserves.

Tuberculosis has reached a fairly high degree of endemicity in Southern Rhodesia. Most of those who develop it in industrial employment were probably infected at home before coming to work.

VII. Non-pulmonary tuberculosis.

The proportion is about the same as in Europe. Bovine infection is probably uncommon. Pott's disease appears to be particularly common in the Bantu, both adults and children.

VIII. Tuberculosis on mines.

The majority of fatal cases occurring on mines die after repatriation. Many cases occurring on mines are not diagnosed. The morbidity and mortality from tuberculosis on mines is approximately the same as that in the Union. Silicosis only occurs after many years of mining, and predisposes to a very chronic form of tuberculosis. Recruits from adjacent territories are several times more likely to die from tuberculosis, or any other cause whether disease or accident, than Southern Rhodesian workers.

PART II. FIELD WORK.

1. Survey of tuberculin-sensitivity rates in the Eastern Region, Southern Rhodesia.

5403 school children in general are 44% positive to 5 T.U. of Koch's Old Tuberculin, and 1488 children were 34%

positive to 2 T.U. There is a marked age gradient. Low-lying areas, not towns, have higher rates. Boys have rates 10% higher than girls. The most rapid rise is from 8 to 11 for both sexes. Family spread was studied. 5 T.U. is preferred as the pre-vaccination test.

Industrial workers have an overall rate of 83%, increasing with age, but only for a few months with duration of employment. Employees from Portuguese East Africa have a higher rate than those from Southern Rhodesia.

II. The criterion of a positive reaction in the Bantu.

The "non-specific" factor described by W.H.O. is present in the Bantu, but there is little overlapping, the minimum being reached at 6 mm diameter, which is therefore regarded as the criterion for a positive reaction. An experiment using boiled tuberculin is described.

III. The significance of differences in the nature of positive reactions.

270 erythrocyte sedimentation rates were done on positive reacting children, and 46 of those with raised E.S.R.'s were X-rayed.

There is a correlation between reactions of over 20mm and with high sedimentation rates, and also between such reactions and evidence of chest disease. Children giving such reactions should be X-rayed.

IV. B.C.G. conversion rates and complications to B.C.G.

The overall rate in 765 children was 98.1%.
Complications were all of a minor degree. The reversion rate after 6 months, among the younger children, was 7%.
The size distribution of B.C.G.-induced reactions is studied.

V. Field methods to be adopted.

The technique of giving injections and field organization are considered with a view to achieving the maximum speed and efficiency. Experiments relating to these subjects are described.

PART III. GENERAL CONCLUSIONS.

The general picture presented is briefly reviewed. There is need for more knowledge about the MaShona and their attitude to disease.

Radical measures are justified to deal with a newly-introduced disease.

Prevention.

Active disease, and not infection, must be eradicated. The principles underlying perpetuation of active disease are stated.

Prevention is then discussed under two headings - General considerations, and aspects particularly applicable to a regional health office. The method of applying B.C.G. vaccination in the field is discussed.

Appendices I to IV.

These comprise a brief account of the history of the Bantu, two maps and tables.