NON-EUROPEAN TUBERCULOSIS IN SOUTH AFRICA.

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PREAMBLE.

The seriousness of tuberculosis among Africans has been known for a long while, but only within comparatively recent years has the fact been turned to the benefit of phthisiology by using it as an approach to the understanding of some of the pertinent problems of the science. The result has been that no disease of Africans is now being more diligently studied. A vast literature on tuberculosis in the native has now accumulated. The more practical fact that the European population can never be safe from the disease unless the hazard of infection from the Bantu race is removed, is a strong motive for the study of those characteristics of the disease in Africans that indicate methods of control.

A number of excellent discussions on tuberculosis in Natives has been published, such as the exhaustive report of the Tuberculosis Research Committee, that by Lyle Cummins on Primitive Tuberculosis, the papers of Dr. Neil Macvicar, the publications of Dr. Dormer, Dr. Peter Allan, of Willcocks, Burrows and Matthews, and Lewis's publication, the Biology of the Negro. To these publications credit must be given as the source of most of the data herein cited.
HISTORICAL SURVEY OF TUBERCULOSIS IN SOUTH AFRICA.

It is agreed that tuberculosis was introduced into the African Continent by invaders from other countries. The first of the invasions was in Northern Africa, along the Mediterranean, and is of great antiquity. The length of time tuberculosis has existed there can be measured to some extent by the undoubted tuberculous lesions found among long-buried excavated human skeletons. In more recent times contacts were made with the disease through invasions that spread down the East and then the West coast as far as the extreme end of the Continent. Then finally the interior was invaded. With the advent of missionaries, slave traders, explorers, merchants, colonists and soldiers, there is scarcely a part of Africa that has not been touched by the white man, "whose footprints are left in the form of tuberculosis among the natives". (Lewis 1942). There is no question but that even within recent times tuberculosis was completely absent in the more inaccessible parts of the Continent. In some cases observers were able to note a lack of it and then to find, after an interval of a few years, unmistakable signs of its appearance. (Lyle Cummins).

In the Sudan among the Eastern Dinkas who were, to quote Cummins (1935) "until 1901 one of the most isolated and illusive of African tribes," and therefore such people as he had described as being free from tuberculosis 30 years before, Burrows (1935) found that tuberculosis had a rather wide distribution and that 32.7 per cent of the general population reacted positively to intra-dermal tuberculosis with a reaction rate of 56 per cent amongst adult males.

There is evidence that tuberculosis is spreading rapidly in French West Africa. Calmette found positive reactors in under 15 per cent of the general population in 1912. This had risen to 44 per cent in 1930. Soldiers arriving in Marseilles from the Ivory coast in 1932 had 46 per cent positive reactors, as compared with a general average of 8.4 per cent in the Ivory Coast in 1912. (Cochrane 1937).

At the present time tuberculosis exists in Africa in varying forms and in varying amounts, depending on such factors as
proximity of foreigners, and the amount of aggregation as in villages, and the length of contact with the disease. It is most common where civilisation has been longest established and it has apparently followed the main lines of traffic, just as in Tropical Africa it has followed the trade routes from the coast.

It is of interest to note that there are no records of virulent epidemic tuberculosis in Africans living in their own country and in their traditional surroundings, such as the epidemics recorded by Robert Louis Stevenson and others amongst the Marquesas in the South Seas, where whole valleys are said to have been depopulated in a matter of months from the time of the first introduction of the disease. (Bushnell 1920).

In South Africa the disease attracted little attention until 1895 when a system of registration of births and deaths came into operation in the Cape Province, due mainly to the efforts of Dr. Gregory, Medical Officer of Health for the colony, who drew attention to the ravages of tuberculosis amongst the non-Europeans. He believed the disease had been introduced by tuberculosics coming to South Africa from Britain and cited the high tuberculosis death rate in the towns which were the favourite health resorts; at Cradoc it was 6.4 and at Beaufort West 7.86 per 1,000 natives.

The earliest history of Tuberculosis in South Africa is very limited, and what information there is, has been gleaned from the writings of travellers and missionaries. (Tub. Res. Comm. 1914)

The first reference to "Consumption" was made by Cornelius de Jongh, the commander of a Dutch man-of-war in a book published on his travels to the Cape of Good Hope in 1761 - 1797. Percival and Barrow at about the same time also comment of the presence of "consumption". It can be inferred from their comments that tuberculosis was not of any serious moment. It was observed that the kaffirs and Xosas showed very little disease at all and they ascribe this to their simple diet, temperate life, limited exercise and few cares. It appears to have helped in the elimination of the Hottentots amongst whom it was reported by J.W.D. Moodie in 1820, to be very prevalent; the same observer noted that it was uncommon among the Dutch and English.
Dr. Hammerschmidt who practised in the Western Province of the Cape from 1858 to 1860 found only three cases of tuberculosis in over 1000 cases seen. Hirsch writing in 1881, said that Phthisis occurred mostly amongst the Hottentots inhabiting the coastal belt.

The Tuberculosis Research Committee (1932) thinks it would be rash to assume that even in remote times the coastal tribes of Bantu were free from tuberculosis and it may be taken as certain that the disease was frequently introduced among them from the sixteenth century onwards, and probably long before. There is little or no evidence that tuberculosis was prevalent amongst the Bantu when they were first encountered by the white race. Lichenstein encountered none in 1803 - 1806 and reports the absence of "sough, chronic disease and syphilis" amongst the kaffirs; Livingstone in 1857 stated that "tuberculosis did not exist" amongst the tribes of the interior with whom he was working. Theal speaks as follows: - "Consumption was almost unknown until recent years". The picture was not one of static contact with coastal Arabs or Portuguese but of a long series of tribal movements accompanied by all the stress and hardships of war and travel and stern selection by survival of the fittest. "Woe to the old, the infirm and the infected under the conditions of such a journey". The merciful custom of the tribes was to desert the old and the fatally ill and leave them in the bush to die; and as Theal says "all the weaklings were destroyed in infancy". In these circumstances a debilitating disease like tuberculosis was unlikely to flourish or spread. The state of constant warfare in which these people lived preserved them from the fate that had already overtaken their neighbours. Their mode of living militated against its spread even if it is accepted that tuberculosis did occur amongst them.

Macvicar in 1908 made extensive enquiries from long-established practitioners all over the sub-continent with a view to eliciting its origin and its prevalence. In his own experience in the Shire Highlands in Central Africa he saw "not a single case of any form of tuberculosis among the natives" at Blantyre Mission during the two years 1896 - 1900. He ascertained from the Reverend John Moffat that consumption in Southern Rhodesia "was of the rarest
natives who had contracted the disease whilst working in the colonies.

Both Livingstone and Moffat who spent a considerable amount of time in BechuanaLand in the middle of the nineteenth century, were of the opinion that there was no tuberculosis among the Bechuana people. The consensus of opinion was that in the Transvaal and Free State tuberculosis was unknown until the advent of the white man.

Ramsbottom (1905) states that up to 1885 with the exception of a few imported cases the Orange River Colony was free from tuberculosis. During the previous ten years he had seen cases occurring amongst the whites, and during the past three years the number had steadily increased. Amongst the blacks the disease was not uncommon and was spreading more rapidly than amongst the whites.

In Natal along the coastal region tuberculosis was found to be common but inland it was rare or absent. In the Eastern Cape Province the evidence pointed towards wide distribution of the disease among the Bantu, it was common in some districts and rare in others. Maevicar believes that this varied incidence is strong evidence that tuberculosis did not exist among those natives originally, because in countries where the disease has long been established, its distribution is fairly uniform.

D. Melville (1913) practising in the Transkei, noted that the first 3,000 cases he had treated in 1897, there were only some 1 per cent of these tubercular, whilst the first 3,000 cases treated in 1912 showed the enormous number of 40 per cent tubercular with lung cases predominating (diagnosed clinically).

In trying to establish which racial group was responsible for the introduction of tuberculosis into South Africa, Maevicar came to the conclusion that European immigrants and soldiers were largely to blame. He quotes figures from Parkes Practical Hygiene showing the average mortality for tuberculosis of the British Army from 1830 to 1846 to have been 8.86 per 1000. Large numbers of British troops were employed in the kaffir wars. In Portuguese East Africa there is a tradition according to Maevicar that consumption came from the Indians. It was certainly the case in Natal in 1908
that the Indians were infected and were probably infecting the natives. Evidence points to the Zanzibar Arabs as the chief introducers of tuberculosis in East Central Africa. (Maevicar 1908).

From 1880 onwards a stream of tuberculosis from Britain travelled to the Cape which had become widely recognised as a health resort. In glancing over the files of old medical journals of the Cape Colony one is struck by the fact that almost the only references to tuberculosis found are contained in papers written in praise of this or that health resort for consumptives. In 1895 when registration for births and deaths became compulsory it was discovered that tuberculosis had become widely disseminated about these health resorts. Beaufort West which had been one of the most popular, was in 1908 one of the most greatly affected towns in the whole sub-continent. The death rate for Europeans was 6.37 and for non-Europeans was 14.32 per 1000. Since that time the tendency of medical opinion has been to check the indiscriminate importation of consumptives. It is certain too that tuberculosis was disseminated in the Native Reserves by the native miners suffering from tuberculosis repatriated from the gold, coal and diamond mines in South Africa. In 1908 Maevicar placed it as the most important single factor in spreading the infection.

In May 1904 tuberculosis - all forms - was made a notifiable disease in the Cape Colony; and in the same year pulmonary tuberculosis was made notifiable in Natal except in Pietermaritzburg where it became notifiable in 1907. In 1905 the South African Native Affairs Commission pointed out the increase in tuberculosis amongst natives. In 1907 the Free State introduced notification on a voluntary basis at the will of the local authorities, the majority of which did in fact adopt it. The Transvaal lagged behind, the Municipal Association being against notification, but Johannesburg introduced voluntary notification in 1907. In 1906 a conference of Medical Officers of Health of the British South African Colonies and Territories passed a strong resolution on the great increase in tuberculosis among the natives and the risk of its further spread. In the same year the Federal Council of the Municipal Association of South Africa at a meeting in Johannesburg, passed a resolution urging their respective governments to appoint a commission on the subject.
In 1907 a deputation of Members of Parliament, Mayors and others waited on the Cape Government and urged the necessity for taking steps to prevent the spread of the disease. In the same year the Cape Town Municipality provided temporary accommodation for 20 tuberculosis patients in connection with the City Infectious Disease Hospital.

The first tuberculosis clinic in South Africa was started by the Medical Officer of Health in Cape Town. In 1908 the Cape Town Free Dispensary was opened and it devoted a special section to the out-patient treatment of tuberculosis. At about the same time a Society for the Prevention of Consumption was organised in Cape Town. The society has since done very useful educative and propaganda work. In 1911 the municipality of Durban appointed a Tuberculosis Officer and established a Tuberculosis Dispensary. In 1901 to 1911 at the various Medical Congresses, resolutions were passed urging the respective Governments to enquire into the whole matter of tuberculosis.

In 1912 following on pressing appeals by the Medical and Municipal congresses and other bodies, the Government of the Union appointed a commission to investigate and report on the whole matter. (Report submitted in 1914). Clauses were included in the Immigrants Regulation Act (1913) prohibiting the entrance of persons suffering from tuberculosis in a clinically recognisable form, except by permit, and subject to certain conditions. Owing to the Great War little further was done until 1919 when a Public Health Bill including provisions based largely on the 1912 commission, was introduced and passed into law. Measures under Public Health Act 1919 against tuberculosis may be summarised as follows:

(1) Improvement of housing and prevention of overcrowding;
(2) Improvement of general sanitation;
(3) Educative work: the teaching, dissemination and inoculation of the elementary principles of hygiene and healthy living;
(4) Enforcement and precautions against spread (through promiscuous spitting and so forth) by persons suffering from the disease;
(5) Disinfection of infected dwellings, clothing, bedding and other articles;
(6) Prevention of the use as food of tuberculous meat, milk or dairy produce;
(7) Notification of cases;
(8) Provision of facilities for early diagnosis and treatment, including facilities for laboratory examinations;
(9) The establishment of tuberculosis hospitals, sanatoria and farm colonies or settlements.

While this bill was being drafted an invaluable impetus to the whole movement was given by Mr. John Garlick who came forward
with a donation of £25,000. towards the cost of a sanitorium in the Cape. In 1920, the sanitorium was established at Nelspoort and pending its completion the then Superintendent Dr. Peter Allan carried out a tuberculosis survey of the Union. He reported that it was a very serious menace especially to the coloured and native races in the Union.

In 1925 a Tuberculosis Research Committee was established by the Government and the Chamber of Mines, which reported in 1932, after a comprehensive study. In 1934 accommodation was increased at Nelspoort and beds were made available at Springkell Sanatorium. Non-European cases were provided for at Rietfontein Hospital. The King George V Hospital was opened in 1939 in Durban. In 1938 the municipality of Cape Town appointed its first Tuberculosis Officer. In 1945 the appointment of a Tuberculosis Officer for the Union was announced by the Secretary of Public Health.
THE INCIDENCE OF TUBERCULOUS DISEASE AMONG THE BANTU.

The exact incidence of tuberculosis in the Bantu is impossible to determine. There is no accurate census of the non-European population, births are not registered and deaths are haphazardly notified; many natives who die are never seen by a medical practitioner and the cause of death is thus never ascertained.

In 1908 Macvicar had cause to write that the provisions of the Act (enforcing notifications of tuberculosis) had been carried out very mildly, and that only a fraction of the deaths were registered; in East Griqualand and Tembuland about one half, in the Transkei about two thirds and in Pondoland not more than one fifth.

Mitchell (1920) remarks that even in the Cape many Europeans and coloured cases are never notified. In other provinces where notification was a recent innovation, the percentage of cases was still higher. The number of notifications was no criterion of the numbers affected.

The position is only slightly improved today and the overall incidence can only be determined by wide generalisations. Small sections of the Bantu population have however been carefully examined and an accurate assessment of infection and disease has been made in these groups.

NOTIFICATION OF TUBERCULOSIS IN THE UNION.

<table>
<thead>
<tr>
<th>YEAR ENDING 30TH JUNE.</th>
<th>NOTIFICATIONS</th>
<th>POPULATION</th>
<th>INCIDENCE PER 1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>1925</td>
<td>624</td>
<td>1738937</td>
<td>0.358</td>
</tr>
<tr>
<td>1931</td>
<td>617</td>
<td>1828175</td>
<td>0.337</td>
</tr>
<tr>
<td>1936</td>
<td>792</td>
<td>2003875</td>
<td>0.395</td>
</tr>
<tr>
<td>1941</td>
<td>1216</td>
<td>2186200</td>
<td>0.555</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>NON EUROPEANS</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1925</td>
<td>5251</td>
<td>6038646</td>
<td>0.869</td>
</tr>
<tr>
<td>1931</td>
<td>5531</td>
<td>6317300</td>
<td>0.877</td>
</tr>
<tr>
<td>1936</td>
<td>7363</td>
<td>7585041</td>
<td>1.049</td>
</tr>
<tr>
<td>1941</td>
<td>14209</td>
<td>8333500</td>
<td>1.705</td>
</tr>
<tr>
<td>1944</td>
<td>18039</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

There has been a gradual increase in both European and non-European notifications. The 1941 figures show an increase of 55 per cent over the 1925 figures in the case of Europeans and 96 per cent in the case of Non-Europeans.
According to the Framingham standards, it is recognised that for every annual death from tuberculosis there are eight living cases of the disease. This ratio has been challenged, and proportions of six and seven active cases have been suggested. There is an agreement, however, that for negroes the ratio of annual deaths to living cases is much smaller than it is for whites, being estimated as from two to four cases per annual death, the reason for this being that the duration of the disease before termination in death is much less. Roth (1938) obtained from army statistics a white ratio of one annual death to 3.75 living cases, a close approximation to the Framingham standard. Under the same conditions he obtained a ratio for American negroes of 2.63 cases per annual death. (Lewis 1942).

Mitchell in 1920 estimated that there were 4000 Europeans and 20,000 coloured and native cases suffering from active tuberculosis. Gruver (1945) placed the estimate at over 4000 Europeans and 60,000 non-European sufferers from the disease in an active form. Dormer (1943) quotes an R.A.F. survey by means of mass miniature radiography showing approximately two cases with positive sputum per 1000 men. He thinks this figure could be applied to the white population and doubled in the case of the non-European. This gives the approximate number of cases which are infectious as 4000 Europeans, 3,140 coloured, 24,000 Bantu and 860 Asiatics.

Masvicar (1908) noted that the death rate over three years (1903-05) was 7.20 per 1000 for non-Europeans, giving a figure four times as great as that for England and Wales. The towns of the Cape Province in which the Bantu predominated, had as high a mortality as those in which the coloured race predominated. In the Eastern Cape Colony, whilst tuberculosis was "very prevalent", it was "not yet so excessively common as in the towns and villages of the central and western parts of the Colony". In the Bantu who lived east of the Kei River and who had come under European influence only half a century previously, tuberculosis was widely disseminated. In the districts along the coast it was very common although in some of the inland areas it had "not yet obtained any great hold on the people". The raw native was less affected than those in contact with the
European. In Natal although no figures could be obtained tuberculosis was found to be prevalent in the coastal areas and less common inland. The Indian death rate for tuberculosis was higher than that of Europeans in the Colony. In the Free State Macvicar stated that infection was spreading amongst the non-European elements. G. Turner, Medical Officer of Health for the Transvaal (quoted by Macvicar in 1908) found that disease was fairly common in and near towns, whilst in the native kraals away from white influence it was rare. In Swaziland and Basutoland disease was not uncommon, being noted especially in natives who had worked on the gold mines.

A commission was appointed in 1912 to enquire into the extent and cause of the prevalence and spread of tuberculosis among all sections of the community in the Union; and to enquire into the extent and causes of mortality of the natives on the Witwatersrand mines and to make recommendations thereon. The following is a "summary of the facts concerning the prevalence of tuberculosis as regards natives".

(1) Tuberculosis is of comparatively recent introduction among the Bantu tribes.

(2) We are satisfied that it has now become a most serious menace to the future of the native races throughout the Union; that it is increasing, and that unless effective measures are taken it is likely to materially increase.

(3) The disease however prevails to a variable extent among the different native tribes and communities, from a comparatively small degree in the native who remains in his kraal and has come into little contact with civilization, up to a very large amount occurring among those brought under the influence of European industrialism and living under conditions, to the native, of exceptional stress. There are many gradations between these extremes. No single term therefore will define the extent of its prevalence among the natives.

(4) The prevalence is in proportion to the degree and duration in which the following factors have operated.
(a) The adoption of civilized habits and modes of life as practised by the natives, including clothing, housing and diet.

(b) The change from the freedom and openness of kraal life to town locations, compounds, barracks, kaffir lodging houses and other close aggregations.

(c) The change from a leisurely life to one of continual labour under more or less arduous conditions.

(d) The coming into contact with massive infection by association with the European and coloured races.

(e) The indulgence in deleterious kinds of alcoholic liquor.

(7) Pulmonary and acute general tuberculosis predominate more especially among adult males; among females and children there is a large proportion of glandular cases, mostly cervical, especially among the less civilised and raw natives.

(8) Speaking generally, it is found to be least prevalent in Zululand and Northern Transvaal, more so in Basutoland, still more in the Cape Native Territories and most wide-spread among the natives in the settled districts of the Cape Province.

In the official history of the Great War (quoted by S. Lyle Cummins 1939) the liability of native troops to tuberculosis is clearly shown in the following figures:

<table>
<thead>
<tr>
<th>SOURCE OF TROOPS</th>
<th>CASES PER 10,000</th>
<th>DEATHS PER 10,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>British and Dominion</td>
<td>6.056</td>
<td>0.398</td>
</tr>
<tr>
<td>Portuguese Troops</td>
<td>33.836</td>
<td>9.242</td>
</tr>
<tr>
<td>Chinese Native</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Labour Corps</td>
<td>36.355</td>
<td>13.433</td>
</tr>
<tr>
<td>Indian Troops</td>
<td>93.464</td>
<td>17.249</td>
</tr>
<tr>
<td>Indian Native</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Labour Corps</td>
<td>142.040</td>
<td>53.384</td>
</tr>
<tr>
<td>South African</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Labour Corps</td>
<td>290.665</td>
<td>221.923</td>
</tr>
<tr>
<td>Cape Colony</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Labour Corps</td>
<td>444.115</td>
<td>103.627</td>
</tr>
</tbody>
</table>

The Cape Coloured Corps showing a higher incidence had a lower mortality.

Cummins makes the comment that European descent when inherited along with a native strain has a considerable degree of reactive immunity to tuberculosis; although it must be borne in mind that a number of the natives were probably experiencing their
primary infection under unfavourable conditions of climate and hard work.

Mitchell (1920) stated that tuberculosis was seriously prevalent amongst the native and colonial populations in the Cape Peninsula and South Western districts, in most of the larger centres of the Cape Province and in those districts of the Transkei and Eastern Province from which native labourers go freely to the mines. During the six years 1914–1919 the notifications in the Cape Province totalled 13,048 coloured and native cases an average of 2174 cases yearly.

Almost a third of South Africa's population lives in the Native Territories.

Tonkin (1944) believes that the greatest problem of medicine and public health is pulmonary tuberculosis. Maovior on the other hand places malnutrition ahead of tuberculosis with syphilis third.

Ryno Smit (1945) remarks that it is commonly accepted that tuberculosis is "rife" in the Transkeian territories although no evidence has been produced to prove it, in the absence of vital statistics. In the light of simple economic factors, however, he asserts that it can reasonably be said that there should be a great deal of tuberculosis.

Peter Allan (Rept. of Tub. Res. Comm. 1932) learned from various practitioners in the Transkei that in Butterworth between 8.8 and 10 per cent of natives consulting them were suffering from tuberculosis. In the Umtata district the figure was 18 per cent. In Ciskei of 300 consecutive admissions to Lovedale Hospital, 71\textsuperscript{b} were suffering from tuberculosis. Allan (1945) concludes that tuberculosis is widespread and endemic in the Transkeian and Ciskeian Territories, and while a certain number of sufferers can lead a sheltered life for many years, it is a different story when the unfortunate natives have to go out and face modern industrial conditions in urban areas. He observes that the same marked increase in the incidence of tuberculosis occurred in England one hundred years ago when England developed into an industrial country.

Maovior (1932) states that between 1913 and 1931 pulmonary tuberculosis maintained a steady level of frequency in
the Ciskei except in 1919-1920 and 1929-30 when, following severe
droughts, admissions rose above the general level. Of a total 418
admissions to Lovedale Hospital in 1913, 71 or 16.98 per cent
were for pulmonary tuberculosis. In 1936 of 1029 admissions 95 or
9.23 per cent were cases of pulmonary tuberculosis. Over the 24
years previous to 1936 the percentage of pulmonary cases averaged 12.10 of
all admissions of 10,017 cases seen from 1932-1936. Macvicar found
that 571 or 5.16 per cent were suffering from tuberculosis (all forms).
Of these 29.7 per cent were under 17 years of age, 38 per cent were
between 18 and 40 and 30 per cent were over 40 years.

Of 2,936 natives of all ages examined by Burton in his
private practice 161 (5.5 per cent) were suffering from tuberculosis
(all forms); of these 140 were suffering from pulmonary tuberculosis
(4.7 per cent), 13 or 0.40 per cent from glandular tuberculosis, 7
were suffering from tuberculosis of the spine (0.3 per cent) and 1
was suffering from peritoneal tuberculosis (0.03 per cent). In a
former series of 1,252 natives he had noted 115 cases of tuberculosis,
(9.18 per cent). During 14 years (1920-1933 inclusive) in the Transkei:
he examined 33,308 male natives between the ages of 16 and 45 years,
for native labour recruiting organisations. Of 5,592 rejected, 254
or .70 per cent had pulmonary tuberculosis (clinically).

In the way of warning to a too easy generalisation about
tuberculosis the Annual Report of the Union Health Department for
year ending 30th June, 1941, states that much confusion of ideas as
regards the prevalence of tuberculosis in the native territories
exists in the public mind. "There are not sufficient data available
"to make sweeping statements, but one fact has been ascertained, and
"that is that well nigh every spare native with a dry skin is labelled
"tuberculous, not only by the lay public but in many instances by
"medical men. "A brief survey was made of notified tuberculosis in
"five locations in Umtata district in 1939 and it was discovered that
"only 2 per cent of cases suffered with 'open' tuberculosis". "Many
"children are notified as suffering from glandular tuberculosis whose
"heads are infected with lice". "In the absence of registration of
"births and deaths and poor co-operation from colleagues in the
"matter of notifying all cases, no statements can be made regarding
"the prevalence of tuberculosis".

Ryno Smit (1945) states that attempts have been made by the Department of Public Health to encourage notification of tuberculosis but they proved valueless on account of the fact that the majority of District Surgeons and medical officers maintain that no good purpose is served by notifying cases, as nothing is being done for them. This is borne out by Tobias (1943) practising about 18 miles from Umtata who claims that tuberculosis is the greatest individual problem that medical officers have to deal with in the territories. During the three previous months he had notified 143 new cases of pulmonary tuberculosis and of these not more than 5 cases went to hospital.

Dormer and his colleagues working in Natal reported in 1943, after a comprehensive survey of 10,000 natives taking 6 years to complete, that in the native reserves there is the problem of the high rate of infection combined with the low rate of disease. In the Natal tribal reserves he found the amount of active disease was remarkably low, 0.25 per cent was the highest figure ever reached. Three types of disease were encountered in the reserves.

(a) The delayed disease characteristic of the European.
(b) The infants had the typical disseminated disease of all infants - European or native.
(c) The progressive primary disease or severe delayed disease in the native who had returned diseased from industry in towns.

In the mission type of reserves the rate rose to 0.75 per cent with the same types of disease as in the tribal reserves. In the peri-urban areas the rate rose to 1 to 1.5 per cent. Here the type of disease was mostly progressive primary disease or severe delayed disease (roughly 70 per cent of the former 30 per cent of the latter). In the urban areas the rate rose to 1.5 to 2 per cent. Here again the proportion of progressive primary disease and severe delayed disease occurred in the proportion of 70 to 30.

The only group of urban dwellers in whom they found a low incidence rate was in domestic servants in good homes and in the native staff of King George V Hospital, where there had been no case of tuberculosis in 5 years.
During 1941, 431 cases of tuberculosis were admitted to the Non-European Hospital, Johannesburg (Gillman and Gordon 1942). This represented 2.5 per cent of total admissions during 1940 or 6.8 per cent of all medical admissions.

**AGE AND SEX INCIDENCE AND MORTALITY.**

<table>
<thead>
<tr>
<th>AGE GROUP</th>
<th>TOTAL CASES - BOTH SEXES</th>
<th>TOTAL DEATHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 9</td>
<td>56</td>
<td>16</td>
</tr>
<tr>
<td>10 - 19</td>
<td>47</td>
<td>14</td>
</tr>
<tr>
<td>20 - 29</td>
<td>116</td>
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<td>30 - 39</td>
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<td>60 - 70</td>
<td>24</td>
<td>14</td>
</tr>
<tr>
<td>TOTAL</td>
<td>431</td>
<td>185</td>
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Williams L.S. 1945 (quoting Dr. Retief) reports that 26 per cent of all new East Coast natives had evidence of pulmonary tuberculosis as shown by mass miniature radiography. The cases were not all active.

Out of a total of 412 medical cases seen in the wards of King Edward VIII Hospital in Durban by Macfayden and Horne (1937) in the previous 12 months, they found 50 cases of pulmonary tuberculosis and 8 cases of tuberculous peritonitis. During a corresponding period at Addington Hospital there had been 26 pulmonary cases and no cases of tuberculous peritonitis.

Studying the death rate for tuberculosis in natives at New Brighton Location, Port Elizabeth, where the deaths are all notified and as correct as any in the Union, Ferguson (1932) found 9.45 deaths per 1,000 population for 1932, for Europeans in Port Elizabeth the rate was .78 per thousand and for Cape Coloureds 5.40 per thousand.

In the four largest towns where mortality figures are reliable, the death rate for coloured people during 1941 was ten times that for Europeans, for Natives seven times and Indians 5 times. (Gale 1945). From the available information in the Union, the death rate from tuberculosis is at least six times as great among the natives as among the white South Africans. (Peter Allan, 1945).
Peter Allan (Rept. Tuberculosis Res. Comm 1932) observes that the curve of non-Europeans tuberculosis incidence is following the European curve in its downward trend. He believes that each race living under constant conditions as regards habits, work and opportunities for infection, has its own index of mortality.

It is of interest to compare the findings of Kahn (1936) among the Bush Negroes of Dutch Guiana, since they show that although the opportunity for infection is present, this is very infrequent as revealed by the extremely low rate of positive tuberculin tests (2.34 per cent) and the great rarity of clinically recognisable disease. The isolation of the group which has lived under the natural conditions to which they were accustomed in Africa before transportation as slaves to America may account for these unusual findings, but Kahn suggests that the favourable circumstances of their life may be of considerable importance in bringing about this unusual situation.
Tuberculosis control in the Union is under the Department of Public Health. A special branch was created in 1945 for dealing with the disease. The Public Health Act 1919 (as amended) contains a special provision for refunding to local authorities who are made responsible for provision of facilities and for the treatment of tuberculosis of a part of the expenditure incurred, in erecting and maintaining institutions and in treating communicable tuberculosis cases. Where the hospital has been erected at his request, to serve a large area, the Minister may refund the whole of this nett cost. Provision is also made for the limit of £10,000. in refunds to local authorities to be exceeded when approved expenditure on tuberculosis is undertaken. Health education and propaganda have been delegated to the South African Red Cross Society which has appointed a Health Education Committee. Voluntary Associations are encouraged (under the Public Health Act) by provision for grants in aid. A summary of unofficial activities for the control of tuberculosis in this country was given in a paper by Marais (1936). Among the voluntary associations interested are the Witwatersrand Anti-Tuberculosis Association, the Cape Provincial Tuberculosis Prevention Committee, the Natal Anti-Tuberculosis Association, the Association for the Prevention of Consumption, and various local authorities' anti-tuberculosis committees.

According to the Secretary of Public Health (Allan 1945) 92 beds were available for tuberculosis in the Union in 1924. On the 30th June, 1944 the position had improved and the following accommodation was available:— For Europeans 589 beds, for Non-Europeans 1,803 beds. He stated that further schemes were in hand to provide beds for 135 Europeans and 705 Non-Europeans. Various other plans were under discussion and it was hoped to take over certain military hospitals and camps which would provide at least another 3,000 beds.

The National Health Services Commission of 1914, using the generally accepted standard of a bed for every fatal case of
tuberculosis per annum, estimated that there should be 15,000 Non-European beds made available for treatment of Non-European cases. Dormer (1943) however, bearing in mind the reluctance of natives to be hospitalized and the fact that only 20 per cent of natives are diagnosed early enough for treatment to be effective, estimates that 4,800 beds would be sufficient to cope with the need. Of these he suggests that 1,600 should be in the Cape Province, 1,200 in Natal, 1,600 in the Transvaal and 400 in the Orange Free State. According to Dormer's estimation therefore it will be noted that there is a shortfall of beds available for isolation and treatment amounting to 3,000.

The service available for treatment of natives including those suffering from tuberculosis are today grossly inadequate. Most natives when ill never see a medical man either because of inaccessibility of their dwellings or because they are unable to afford a visit from a European doctor. Many South African practitioners have urged the necessity of a medical service for natives in their territories (Gear, Gale, Allan, Burton, Tonkin).

In 1939, the Minister for Public Health laid down the policy of establishing a chain of health clinics throughout the native territories. It was proposed to establish one such clinic at Polela, as a model, and to expand the service as the clinics proved their value. This clinic was to have six satellite clinics, each of which was to be attended by a medical officer on one day a week. £12,000. was voted by parliament towards this end in 1940. (Gear 1941)

Dormer (1943) has stated that 70 per cent of tuberculosis cases diagnosed in Durban hospitals have advanced bilateral pulmonary disease.

Gillman and Gordon (1942) discuss the fate of 150 pulmonary tuberculotics admitted to the Non-European hospital Johannesburg the previous year, who did not die in hospital. They found that 45 cases had been discharged by physicians, 36 cases refused further hospital treatment and only 17 cases were transferred to other institutions for further treatment. Of the 133 cases returning to their homes only 7 were found to have negative sputum. The average duration of hospitalization was 30.1 days; 20 per cent of cases were hospitalized
for more than 90 days; 26.5 per cent of cases remained in hospital for less than 10 days. They found the average length of stay of tuberculous patients in American hospitals to be 164 days and in Nelspoort and King George V Sanatorium the average duration of hospitalization during 1940 was 166 and 153 days respectively.

Macvicar (1932) states that in his hospital native tuberculosics are retained for an average of a month to educate them in preventive methods. Most of his cases are rapidly progressive in type.

From 1936 to 1943 the Public Health expenditure on tuberculosis amounted to about £750,000. The amount spent by the Provinces totalled about £170,000, while a quarter of a million had been spent in capital expenditure. (L.S. Williams 1945).

The capital cost of tuberculosis beds in the Union works out at approximately £500 per bed for Europeans, through lessening amounts for Europeans and Asiatios to £100 per bed for the Bantu. This, explains Cluver (1945) is not because of deplorable race discrimination but because the African is not happy in the sumptuous surroundings demanded by civilized Europeans. He is most successfully treated in his own primitive home surroundings, provided with a diet to which he is accustomed. The maintenance of beds works out at 20/- a day for the European and 2/- a day for the African.

Many suggestions have been made as to the best way of reducing or eliminating the grave problem of tuberculosis in the South African native.

Macvicar (1908) believed that efforts should be directed along two lines: firstly to reduce the spread of tuberculosis and secondly to improve the social conditions of the people: he considered the second to be the more difficult and the more important task. He urged the control of tuberculous natives repatriated from the mines which he believed were as much responsible for spreading the disease as all the other agencies put together. He recommended sanatorium treatment for early cases and general hospital treatment for advanced cases making use of skilled native nurses, and he urged the necessity for health education through schools, universities and native health societies, citing as example the success which had
attended elementary health propaganda among the Red Indians in Canada in reducing the disease by one third and the mortality by one half. He believed that the natives would not ever be able to afford treatment in clinics unless it were free.

The Tuberculosis Commission (1914) laid special emphasis on the fact that measures for combating tuberculosis must begin with the prevention of cases, and that the way to effect this was by improving firstly the conditions under which so large a proportion of the coloured and native population lived in urban areas and secondly the conditions under which they worked in the industrial centres, especially on the mines.

Dormer suggests that the essential measures to combat tuberculosis are to find and isolate the infecting case and to feed all sections of the community adequately; as a corollary he adds that no one should be allowed to do sustained physical effort without adequate diet. To implement his first recommendation he suggests mass miniature radiography combined with tuberculin surveys. He believes that education of the patient in hygiene is the most effective public health measure and that a patient who has learnt how not to infect others is already isolated even though he is never admitted to a hospital. Dormer is so strongly convinced of the part that adequate feeding plays in the development of tuberculous disease that he states that if nothing else were done than to feed a nation on a fully adequate diet, tuberculosis would die out in a reasonable number of years.

Ryno Smit (1945) agrees with Maovicar and Dormer in the importance of the economic status of the native. He advocates provision of facilities for isolation in the form of farm colonies and care of the soil and elimination of "cattle barons", in the native reserves. He states the need for accurate vital statistics.

Gale (1945) believes that an adequate public health programme of housing, feeding and health education is of even greater importance than the building of more hospitals and the establishment of more clinics. For tuberculosis is more than a disease, it is "itself a symptom of a disordered socio-economic system".
Dangerfield (1943) proposes the amalgamation of all public bodies, whose aim it is to eradicate tuberculosis, into one national body; its duty would be to co-ordinate all propaganda and it would act in liaison with the Department of Public Health. He rightly points out any scheme which approaches completeness for one race will defeat its own end if it does make similar progress against the disease in other races, due to their close association in domestic service and in industry, which facilitates the transmission of infection from one to another.

It is pointed out by Matthews (1935) that fifty years of knowledge of the infecting organism and several decades of anti-tuberculosis measures leaves about 90% of the adult in most urbanised districts in Europe infected by the tubercle bacillus. Schemes based on this principal are not likely to be any more effective in Africa. The present day conception of effective treatment for tuberculosis demands conditions which in countries such as Zanzibar, do not exist. Even if expensive and elaborate institutions with their specially trained medical and nursing staffs were made available, the mentality of the population would inhibit the success of a form of treatment affecting in detail the general mode of living over a considerable period, and demanding a punctilious attention to small matters beyond the experience and outside the comprehension of the majority. He thinks that by sympathetic adaption of native ideas and methods to the use of modern hygiene, rather than the abrupt substitution of European regulations for native custom success is most likely to be achieved.

Cochrane (1937) and others (Stones 1933, Matthews 1935, Cummins 1939 and Marais 1936) have advocated the tuberculosis village as the ideal type of accommodation for Africans. Cochrane recalls a mission station in Portuguese East Africa where such a village had been installed and his impression was that it was extremely successful. These villages cost little to build and can be pulled or burnt down in whole or in part if they are badly infected. Where but little money is available, these institutions ought to play a valuable part.

As an example of what can be done for patients near their
homes when staff and funds are available, W. Harden Smith quotes the work done by Kibongoto Tuberculosis Hospital at Kilimanjaro. This hospital acts as a separate unit and associated with it is a chain of dispensaries on the mountain which are partly conducted in collaboration with mission dispensaries. Cases are detected at the dispensaries, and when considered advisable they are sent to the main hospital. At the hospital artificial pneumothorax and other surgical procedures are carried out in suitable cases who may be sent home to continue their treatment, including refills by visiting members of the hospital staff at the dispensaries. Results are definitely encouraging and many apparently arrested cases may be seen; these cases have a considerable propaganda value.

At Kibongoto, a village settlement for open cases who are able to do light work has been established, but it is new and still in the experimental stage.
Adequate explanation of the behaviour of tuberculosis in the native is an extremely difficult subject, but this has not deterred the proffer of several different theories in which their champions emphasize one or other of the various factors. These theories may be divided into those which deal with environmental influences and those which have to do with constitutional factors involved in resistance or that power of withstanding the attack of the tubercle bacillus which the organism possesses apart from the effects of the infection itself.

The view that the status of tuberculosis among natives is due to their escape from primary infection in early life is generally believed not to hold, since tuberculin surveys show that the incidence of infection among natives at all ages closely approximates that of Europeans. Further it is known that primary infection occurs also in Europeans and when it does it proceeds just as mildly as it does in childhood.

As to heredity, it is a well-established fact that animals of different species show greatly varying levels of resistance towards tuberculosis infection, quite apart from environmental factors. Guinea pigs for example show very little resistance, whilst that of rats is high.

It has been asserted that different human races show a varying resistance or susceptibility to tuberculosis. Koch believed in the idea of an "inherited predisposition" to infection, a theory which received support from the statistical investigations of Karl Pearson and later warmly upheld by Maynard (1912). Sanarelli (quoted by Lyle Cummins 1939) propounded a theory of "inherited resistance" to infection, the quality being less marked in persons showing a liability to the disease than in those capable of escaping it.

The chief advocate of the idea of racial susceptibility are Pinner and Caspar; they believe that "there exists a true "racial (genotypic) difference between the two races, which "confers high resistance on one and low resistance on the other "race". They are"impressed by the necessity of considering the
likelihood of true genotypic difference. A genotypic low racial resistance, if this is the explanation of the tuberculosis in natives, dooms him to a continual high morbidity and mortality except for the changes that can be brought about in living conditions and by lessened exposure to infection.

The possibility of hereditary transmission of specially low or specially high resistance to tuberculosis has been postulated. These properties are held to be constant or at least very slowly changing features of races. The relatively immune races are believed to have acquired this characteristic through long experience with tuberculosis, which the non-resistant race has not had. In this way there has arisen a process of natural selection; those who succumb to the disease are the weakest and those who survive are the strongest; the qualities which the strong possess are transmitted to their progeny and in due course all members of the group will have inherited the constitutionally fixed ability to resist the disease. The relative amount of such resistance among races will depend upon the relative length of exposure to the infection. Natives and other races who have known the disease a comparatively short time have not yet had the opportunity to acquire the full resistance that the forces of natural selection are considered to have brought to most of the white races. The changes that are accomplished by this mechanism must be measured in terms of generations and are of necessity slow in being established, yet in the comparatively short history of exposure of American negroes (Lewis 1942), a marked alteration in the type of disease can clearly be demonstrated, as in the South African native during the last 20 years. (Sutherland Strachan 1945).

Anderson (1928) on the basis of mortality rates and age incidence in the racially mixed population of Mauritius, concludes that a race unexposed to tuberculosis and which then has a continued contact with tuberculosis "begins to acquire resistance within thirty years, has already acquired it to "and appreciable extent in 50 years, and in more than one- "hundred and less than two hundred years has developed
"resistance to the full degree".

Apparently as time goes by the range of variation in racial resistance in man is narrowing. The tuberculosis of Negroes, Polynesians, Eskimos and other groups with former astounding tuberculosis mortality, appears to be far less acute in general in its type today.

Rich (1938) believes that the European has an ability to develop acquired immunity more rapidly and effectively than the black as a result of infection. The concept postulates not so much the inheritance of immunity as the inheritance by European races of a power to respond to infection in a more favourable way than the native.

Grasset (1929) believes that the aptitude to create immunity is hereditarily transmissible as a fixed character; this reactive faculty, notwithstanding the fact that there can be no occasion for its operation, is however transmitted as a latent character from generation to generation although it is subject to considerable individual variation in the same species.

The influence of environmental factors especially nutrition, housing and sustained physical effort.

There appears to be general agreement that environmental factors determine or influence the onset of disease and its type but there is a wide margin of variation in the importance attached to it.

Tuberculosis has always been and still is far more widespread among those sections of the population whose economic situation is bad than among the better placed sections. The lower the income the higher the mortality from tuberculosis. This inverse relation between incidence of tuberculous disease on the one hand and standard of living on the other is one of the few tangible facts to be found in the whole study of tuberculosis (Ustvedt 1942). Whitney in America found the tuberculosis mortality rates for unskilled workers to be 185, for skilled workers 66 and for professional men 26 per 100,000. In South Africa the figures were 77.9 for unskilled workers, 57.4 for skilled workers and 25.4 per 100,000 for professional men (both quoted by Dormer 1943). In Copenhagen the tuberculosis mortality rates
were found to be 51 per 100,000 living in the upper class, 95 in the middle class and 132 in the working class (Ustvedt 1942).

During the last century the tuberculosis mortality rate has shown a steady decline in most civilized countries. Dorrer 1943 denies that this downward trend can be ascribed to our efforts at treatment and segregation as it commenced long before the cause of the disease was discovered. He believes the prime factor has probably been the improvement in quantity, diversity and quality of foodstuffs and the decrease in the sustained physical effort in nations as a whole.

The Tuberculosis Research Committee (1932) came to the conclusion that primitive communities exhibit a marked susceptibility to tuberculosis when they encounter it first and that although the infection may be fairly well tolerated under the natural tribal conditions, this susceptibility is fraught with extreme danger when exposure to infection is accompanied by a sudden change in occupation, food, housing and mode of life generally.

That this does not always apply and when it does is subject to special conditioning factors is illustrated by the finding of Kahn (1936) among the Bush Negroes of Dutch Guiana, who show a low rate of infection as revealed by tuberculin testing (2.34 per cent) and a great rarity of clinical disease. Kahn believes that although isolation of the group which has lived under the natural conditions to which they were accustomed in Africa before transportation to America as slaves may account for the unusual finding, the most probable explanation of this unusual situation is the favourable circumstances of their lives. In the calamitous epidemic that decimated the Indians of the Canadian plains from 1882 onwards the special conditioning factor was the disappearance of their usual supply of food, the vast herds of buffalo which had until then roamed the plains (Ferguson 1928). From a mortality rate of 9,000 per 100,000 at first the rate rapidly dropped "by its own weight" to 1,000 in 1907 and 270 in 1932. From 1822 to 1848 the death rate from tuberculosis for negroes was no greater than that for Europeans in Charlestown S.C., though from the date of the American Civil
war onwards the black showed a markedly greater tuberculosis death rate; many were homeless and there was a greater economic upheaval amongst the blacks following the Civil War. Since then the mortality rate for the negroes has been roughly twice that of the Europeans in America. (Bushnell 1920).

The native race has by force of circumstances been impelled, somewhat precipitately, to change its manner of living from that of a pastoral and warlike people to one of industrialism with increasingly close contact with Europeans and to some extent with urbanisation. This transformation in their lives has been accompanied by a considerable decline in their relative economic status and the availability of protective foods.

Fox (1939) considers that the simple diet of many of the natives living in the reserves is capable of producing and maintaining good nutrition but that the margin of safety is a small one. He believes that their simple diet is being undermined by the "extremely destructive methods of native agriculture which are converting once fertile areas into semi-desert". The factors leading to a slow and steady deterioration in the nutrition of the Native Reserves are overpopulation, overstocking and overgrazing intensified by periodical droughts, and the inability of the native to adjust himself sufficiently quickly to the changes that are taking place in his environment. He believes that the problems of nutrition in urban areas arise mainly from "lack of purchasing power as well as from ignorance".

In a survey of school children in the Transkei, it was found that 39.4 per cent have milk, 28.3 per cent eat beans, 14.6 per cent eat meat, 9.2 per cent have pumpkins and only 0.4 per cent ever eat green vegetables. (Ryno Smit 1945).

In a survey of the districts of Umtata, Ngqueleni, Libode and Mganduli, it was found that among 8,000 kraals 28.8 per cent of families had no cattle, a further 30 per cent derived little or no benefit from their cattle either in milk or in ability to plough or haul; in the district of Cala, if the cattle were evenly distributed (which they are not) every person would receive only one third of a pint of milk daily; in this district
too, provided the people ate all the cattle that died of starvation, redwater and gallsickness, in addition to all those slaughtered, each person would receive 24 lbs. of meat per year; the average yield of maize per acre in the Transkei is 1.19 bags per annum. (Ryno Smit 1945).

Peter Allan (1924) was impressed with the part played by insufficient food in adding to the liability of infected persons to develop clinical tuberculosis, and the tendency for tuberculosis incidence and mortality to fall when good and sufficient nourishment is available.

The Tuberculosis Research Committee (1932) considered that the better feeding of natives in Portuguese East Africa may offer an explanation of the fact that these natives produced 22 per cent less of the acute types of tuberculosis on the mines than did the natives from British South Africa.

Ramsbottom (1905) practising in the Orange Free State believed that the chief agent in the spread of tuberculosis "next to the bacillus itself" was poverty.

Crenstein 1939 stresses the importance in tuberculosis of economic status and more particularly inadequate nutrition.

Gale (1945) maintains the importance of maintaining full nutrition to prevent tuberculous disease in a tuberculized country. He finds that the death rate during the war has risen slightly among Europeans and Indians and steeply among natives and the coloured population and believes this is a sure indication which groups in the population are suffering most from the socio-economic consequences of war.

Allan (1927) after an extensive experience with Portuguese natives on the Witbank coal mines is of the opinion that racial factors play a secondary part and that "general hygiene" plays a dominant part in the causation of disease.

In 1909 the general condition and "treatment of natives" "(on the coal mines) was indescribable; underfed, over-crowded, "overworked, pay tickets stopped for weeks, and continuously and "brutally assaulted by miners, they were worse treated than cattle".

The tuberculosis wastage for 1000 per annum in 1909 was
23.5 in 1926 it had fallen to 4.6.

Matthews (1935) noted that the diet of Zanzibar natives was poorly balanced and that there was a common tendency to deficiency in protein, mineral salts and vitamins A and B. The incidence rate of tuberculosis in Zanzibar and the types of disease found, more or less paralleled those occurring in South Africa.

When the Factory and Education Acts were introduced and became effective in England, the incidence of this type of tuberculosis fell to a notable extent; previous to this the mortality rate had been a very serious matter but it has since ceased to be a matter of national importance (Rept. Sub. Res. Comm. 1932). Under good social conditions, especially as regards nutrition, the infant mortality can be reduced to remarkably low levels. (Ustvedt 1942).

Macvicar (1908) states that if racial predispositions be a factor in the spread of tuberculosis among the native races of South Africa, then in his opinion, "it can safely be relegated to a secondary place". Its prevalence was in direct ratio to the squalor and each grade of society had its own limit of possible tubercular infection. He quotes Jonas (Am. Journ. Med. Sciences 1906) who says of the negroes of the Southern States who suffer more from phthisis than the whites, "the racial element is now regarded as a negligible quantity, their environment being the principal factor which favours tuberculosis". Baldwin (quoted by Bushnell 1920) believes the tuberculosis of natives to be that of a tuberculized race, just as is that of the whites. "It is not safe to assume that the difference in mortality is due to "racial susceptibility, for even a superficial study of conditions "discloses bad housing, improper food, ignorance of disease, and "lack of medical attention to a degree that raises the question "whether whites, subject to the same influences, would not suffer as "much."

Although the tuberculosis rate among British and Dominion prisoners of war in German hands was not above the normal incidence, it has been extremely high among other allied
prisoners. The main difference has not been in housing but in food the British and Dominion prisoners of war received 40 lbs. of carefully balanced supplementary food from the Red Cross Societies per month. (T.W.B. Osborn 1945).

Denmark during the early period of the Great War was exporting most of the butter, eggs and milk which she produced to the Allies and the tuberculosis death rate continued to rise as it did in other belligerent countries. When the blockade made it virtually impossible to export these products and they were consumed locally, the death rate from tuberculosis fell whilst it continued to rise in the other belligerent countries until after the war. The "primitive" type of tuberculosis was frequently encountered in Austria after the Great War. (Dormer 1943). The tuberculosis death rate in England and Wales for 1941 shows an increase of 12.1 per cent over the 1938-39 figure; for Scotland the increase was 20 per cent. Dormer attributes this to rationing, overcrowding and increase in physical effort.

In Natal, Dormer (1943) found that the disease incidence in the Native Reserves was below 0.25 per cent, whilst in the urban areas it rose to six times this figure (1.5 per cent) in spite of both sections of the community being almost equally tuberculized; of the Reserve native 20 per cent have an adequate and 80 per cent inadequate diet; in the urban areas the native diet was found to be "almost completely inadequate". On the basis of these facts Dormer believes that malnutrition is the conditioning factor in the acute type of disease seen in the native and sustained physical effort with fatigue, to be the precipitating factor. He does not attach any significance to racial factors per se. In his opinion, if Europeans were exposed to the same degree of malnutrition and sustained physical effort when they were highly allergic as the natives constantly are, they would succumb to the identical type of disease.

These observations show that defective nutrition may have a very considerable effect on the source of tuberculosis in any given individual or race, for in the tuberculous patient it may accelerate
the tempo of the disease and in the infected person it may increase the likelihood of disease. The importance of nutrition seems to be so great that it would appear reasonable to regard it as the most vital of all factors underlying the association between a low standard of life and the high incidence and rapid progression of tuberculous disease.

Influence of housing: Willcocks (1938) believes that the chief factor influencing the incidence of tuberculosis is close contact in small dark ill-ventilated huts where it is the custom for whole families to sleep together.

Dormor (1943) on the other hand finds that crowding gives the opportunity for infection but not necessarily for disease. In this he is supported by Ustvedt (1942) who believes overcrowding to be of doubtful influence in the development of tuberculous disease.
The industries of South Africa employ annually nearly a million natives. In 1942, 355,614 natives were employed on the Rand Gold Mines. This labour force is drawn virtually from the whole of Africa south of the equator.

Large numbers of natives are constantly absent from the territories working for various periods elsewhere in the Union of South Africa. In the Umtata area in 1927 of 141,903 male tax-payers, 39,572 or 27% got travelling passes to look for work outside the territory; of these 72.3% went to work in the gold mines. (Peter Allan in Rept. of Tub. Res. Comm. 1932).

The natives are recruited in the various Territories, medically examined, and sent in batches to the Witwatersrand Native Labour Association Compound in Johannesburg. There is a comprehensive medical service to cater for the needs of the native labour force. When the natives reach the Witwatersrand Native Labour Depot, they are re-examined and any showing signs suggestive of tuberculosis, e.g. "pleurisy with or without effusion, thickened pleura, un-resolved pneumonia, "localized crepitations or silicosis" are detained in the hospital for observation and investigation.

In 1932 (Rept. of Tub. Res. Comm.) from 170,000 to 180,000 recruits were examined annually, and the number presented for examination daily varied from 300 to 1,200 with a staff of 6 whole-time medical officers. The labour force has been practically doubled since then.

Examination is mainly stethoscopic and there is universal criticism of the efficiency of this method in identifying the early case of tuberculosis. (Dangerfield 1943, Dorrer 1943, Ustvedt 1942). Dangerfield (1943) states that there is an average of 200 stethoscopic examinations on a medium sized mine and that other conditions than tuberculosis, such as early pneumonia, complicated influenza, bronchitis, pleurisy and crepitations of non-tuberculous origin, are more frequently the prize of the medical officer's search.

Each mine has a wholetime mine medical officer concerned
only with the health of the native miners. When the native reaches the mine to which he has been allocated, he is again examined by the mine medical officer.

Each mine (or a group of mines in close proximity) has a mine hospital. Hospital beds are provided in the ratio of 2½ per cent of the average number employed. Some are staffed by European female nursing sisters, others by male nurses. Experience has shown that except in very minor ailments and injuries, restoration to health is delayed in the case of natives treated as out patients. Broadly speaking 40 per cent of cases at any one time are medical, and the majority of such cases are suffering from respiratory diseases. (Rept. Tub. Res. Comm.)

The terms of service vary with the natives coming from different territories. Some are employed on a monthly basis, some on a three monthly basis, and others contract to serve between 180 and 313 working days. At the termination of their contracts, native miners are again examined by the medical officer and if found to be free of tuberculosis or silicosis, they are repatriated. The rate of change of the mine native native population varies on the different mines between 80 per cent and 115 per cent per annum. It is estimated that at any one time 25 per cent of the native compliment are boys under 25 years, 70 percent between 25 and 40, and not more than 10 per cent over the age of 40. The duration of employment is 67 per cent over one year and under five years, and 10 per cent over five years.

The diet scale as laid down by the government regulation includes mealie meal (24 oz.) bread (6 oz.) beans or peas (3 oz.) meat 6.85 oz. soup meat (1.7 oz.) peanuts (2 oz.) sugar (1 oz.) vegetables (5 oz.) salt (0.3 oz.) cocoa (0.25 oz.) kaffir beer (6 oz.) (minimum daily allowance), giving approximate calorific value of 4,385. The usual practice is to give a small meal before going on duty and a large one on returning. Bread is issued at the time of going on shift.

Of 20,000 natives selected at random, subject to having done at least 6 months service and being discharged as apparently fit, the average weight on entry was 132.4 lbs. and on discharge
135.7 lbs. 26.4 per cent had lost an average of 4.13 lbs., while 7.7 per cent had maintained a stationary weight and 65.9 per cent had gained an average of 6.58 lbs.

A native must be weighed every 6 weeks during the time of his employment. Any native who is found to have lost 5 lbs. between two consecutive weighings, or a total of 6 lbs. or more in three consecutive weighings is subjected to a special clinical examination including if necessary detention in hospital. The efficiency of these examinations is indicated by the fact that out of 2,217 consecutive cases of pulmonary tuberculosis (not including tuberculosis with silicosis) which had been certified on the gold mines of the Witwatersrand, 1,006 were diagnosed during illness in hospital, 504 as a result of clinical medical examination outside the hospital, and 707 at periodical weighings. The value of weighing is indicated by the finding that out of 2,054 consecutive cases of pulmonary tuberculosis, unconnected with silicosis, 1,714 had lost weight, 404 gained and 136 remained stationary in consecutive weighings. (Rept. Tub. Res. Comm. 1932).

As a result of queries from the Miner's Phthisis Bureau being passed on by the Gold Producers Committee to the Mine Medical Officers, every native showing losses of even one or two pounds at each of two consecutive periodical weighings, was subjected to a special medical examination over a period of three months, with the result that for a 200 per cent increase in examinations done, the number of tuberculosis cases detected at periodical weighings rose from 26.25 per cent to 31.71 per cent. (Dangerfield 1943).

Underground work is arduous and working conditions are sometimes difficult with a relatively high air humidity and temperature. Dangerfield (1943) believes the native is rendered more liable to develop tuberculosis by the unusual conditions of feeding and living, the large numbers housed together in compound rooms, the close contact in cages and the strain of hard work in humid underground conditions.

Virtually 70 per cent of Native Miners arriving for duty
at the gold mines are tuberculin positive. (See. Section dealing
with tuberculin).

INCIDENCE OF TUBERCULOUS DISEASE:

In 1914 when Gorgas submitted his report on the sanitation
of the gold mines, he pointed out that the wastage from tuberculosis
alone in 1912 had been 10.87 per 1,000. He recommended scattering
the natives in their living quarters by allowing more floor space
and by subdividing the large compounds. He further emphasized the
necessity for the careful routine examination of the sick and ex-
clusion of tuberculous cases from the mines. In 1915 the incidence
of tuberculosis and silicosis was 13.1 per thousand. This had
dropped to 7.2 per thousand in 1930. There has been a progressive
decline in the prevalence of tuberculosis of the lungs on the gold
mines from 4.50 per 1,000 in 1928 to 2.54 in 1942. Tuberculosis
other forms i.e. tuberculous glands, peritonitis, miliary tuber-
culosi and joint tuberculosis, principally, also show a decline
from 1.93 per 1,000 in 1928 to 1.14 per 1,000 in 1942. Silicosis
with tuberculosis shows a great improvement - 1.17 per 1,000 in 1928
to 0.23 per 1,000 in 1942. (A. Miller 1943).

The annual report of the Miner's Phthisis Bureau gives
the "production rate" of tuberculosis (pulmonary) for European Miners
for 1927 - 28 as 2.19 per 1,000 and natives as 3.85 per 1,000.

The Tub. Res. Comm. (1932) found evidence suggesting that
certain groups of natives coming from parts most isolated from out-
side contacts with civilization were more susceptible than the others.

The incidence rate appears to vary with the years of
service. Mavrogordato found (Rept. Res. Comm 1932) that the first
year on the mines, whether on first engagement or re-engagement, was
the great danger zone, and that the liability to contract tuber-
culosi was far greater on first engagement than on re-engagement,
that natives who ran the gauntlet of the first year were in a good
position for some time, but that after the close of the fifth year,
the tuberculosis rate started to rise as the baneful effects of
duration of exposure to mining conditions overcame the beneficial
effects of acclimatization to these conditions: the silicotic
element played a large part in the rise of the tuberculosis rate after the close of the fifth year.

Natives known to have over 5 years continuous service on the gold mines have a prevalence rate of 32 per 1,000 (Rept. Tub. Res. Comm. 1932).

Lyle Cummins and L.G. Irvine, examining the clinical and radiological records of 762 cases found that "new" mine natives tended to develop a very acute and very severe type of disease, whereas long service natives inclined towards the more chronic type of disease. In a series of 500 cases each of natives with tuberculosis fit for repatriation, and those unfit for repatriation, the average duration of service of the former was 3 years 10 months, and for the latter 2 years and 6 months in those dying on the mines. (Rept. Tub. Res. Comm. 1932).

The examination of mine soil and air was undertaken at the suggestion of Prof. Lyle Cummins in an endeavour to find out to what extent the mines themselves were infective (Rept. Tub. Res. Comm. 1932). That infection with tuberculosis can occur underground had already been shown by Nivrogordato (1926). He found tuberculosis in 3 out of 46 rats kept underground for varying periods, but in no instance for more than 18 months. Acid-fast bacilli were observed in 48 out of 100 samples of soil examined (biological tests were negative in every instance) and in 3 out of these 14 samples of air examined. Watkins-Pitchford (1916) found 15.2 per cent of sputa from underground workings to be tuberculous but the diagnosis was based only on bacterioscopic findings. 7 out of 33 specimens of sputum examined from underground (i.e. 20%) showed the presence of acid-fast bacilli but biological testing proved only 1 to be tuberculous. (Rept. Tub. Res. Comm. 1932).

All cases of tuberculosis detected on the gold mines are sent to the W.N.L.A. Hospital for examination by the Miners Phthisis Medical Bureau in terms of the Miners Phthisis Act. On completion of the formalities of compensation which last about a fortnight, the natives are sent to their homes. A number of natives in an advanced stage of the disease are unable to undertake the journey and are detained and cared for until death supervenes. "The early
cases seem to improve and put on weight but the natives clamour to return to their kraals and refuse to stay in hospital long enough for adequate treatment". (Rept. Tub. Res. Comm. 1932).

Compensation is provided to the sufferer provided that he has been in continuous underground employment for at least one month and that his disease has been detected by the Bureau within six months after he has ceased to be employed underground. Thence, again if he is fit to travel, he is repatriated to his home in the territories.

Dangerfield (1943) condemns the rapid evacuation of these natives to their homes. He says that no effort is made to give them the benefit of any special treatment and believes that travelling in their ill state further reduces any chance of recovery. The authorities have always contended that a native who is suffering from tuberculosis knows the prognosis is hopeless and his one desire is to return to his home. (Rept. Tub. Res. Comm. 1932).

Miller (1943) admits that very little effort is made to dissuade tuberculous natives from seeking repatriation.

In the early days of gold mining on the Rand before the medical services had been co-ordinated, there was a high death rate on the mines. Between July 1903 and June 1904 274 deaths occurred in 567 natives admitted to hospital suffering from phthisis. Pulmonary tuberculosis was responsible for 5.40 per cent of all deaths in that year (Macvicar 1908). Of 3,046 recorded cases of tuberculosis on the mines for the three years 1926 - 29, 610 or 20 per cent died in the mine hospitals. (Rept. Tub. Res. Comm. 1932). In 1942 the case mortality had dropped to 11.8 per cent (Dangerfield, 1943).

Deaths from tuberculosis on the gold mines show a maximum incidence in the summer months (Rept. Tub. Res. Comm. 1932).

Attention has been focussed on the tuberculotic natives repatriated to the territories for the probable role they play in disseminating infection among their tribes. The Tuberculosis Commission (1914) believed that owing to the extent to which the disease occurred on the mines, together with the frequently changing personnel, the mining industry was "one of the most important of
"all factors in the cause and diffusion of the disease among the "native population". Peter Allan (Rept. Tub. Res. Comm. 1932) gave it as his considered opinion that the part played by them in disseminating infection was "small". In the same report one learns that about 25 per cent of tuberculosics seen at the Holy Cross Mission in Pondoland contracted the infection on the Rand gold mines or from relatives who had returned from the mines.

Peter Allan (1924) followed a series of 112 natives repatriated from the gold mines with active tuberculosis and found that whilst 49 per cent had died within a year, 25 per cent recovered under kraal conditions, without any skilled treatment, sufficiently to work again. In 1926 and 1927, 60 per cent of 207 native tuberculosics repatriated to the Transkei had died after two years. At the end of the two years a further 10 per cent were unfit for any work, 26 per cent were doing light work, and 4 per cent were back at heavy work.

Forty per cent of 348 natives repatriated from 1926 to 1928 died in the first year. (Peter Allan in Rept. of Tub. Res. Comm. 1932). In 1942 repatriation totalled 869 tuberculosics including 74 tuberculosics with silicosis.

Of 168 cases of tuberculosis repatriated and admitted to the Macvicar Tuberculosis Hospital, Lovedale, 61 were discharged as quiescent or much improved with negative spueta, 20 were improved with positive spueta, and 58, who were admitted with advanced disease died within a few days of admission.

The rapidly fatal type of disease which is observed in native miners, as also the more chronic type observed in long term service natives in association with silicosis, has already been commented on in the sections dealing with pathology and clinical manifestations. Dormer (1943), Cluver (1945), Peter Allan (1945 and Williams (1945) all emphasize the necessity for a course of training for native recruits to industry prior to their employment. They believe it is particularly necessary in the newly recruited African worker who has come from a life of leisure or ease and malnutrition in the native reserves. Dangerfield (1943) advises the introduction of mass miniature radiography and tuberculin
testing of all recruits. Willcocks (1938) has warned against the employment of natives showing positive reactions, with vesiculation to tuberculin testing, in industry.
RESPONSE OF THE NATIVE TO TREATMENT.

There exists a common impression that no treatment is of much avail in the native tuberculous. The type of rapidly progressive disease observed on the gold mines makes a poor case for treatment. The majority of native miners presenting symptoms for the first time already suffer from advanced lesions.

In the general native population there exists a considerable degree of malnutrition and this militates against successful localisation of the disease. Economic factors play an important part in determining at what stage of the disease the patient will seek treatment. The Bantu's low level of economic security forces him to remain at work for as long as possible and it is found difficult to keep native patients on long treatment for this very reason.

Heimann (1936) writing of the advanced type of case requesting treatment states that no treatment is of much avail in these late and hopeless cases. In the earlier cases where the disease was unilateral and artificial pneumo-thorax was attempted, he found the mentality of the people was against prolonged treatment as they discontinued it precipitately after a few refills.

It is Lyle Cummin's opinion that the native lacks natural immunity to tuberculosis which insures the success of operative procedure like artificial pneumo-thorax and thoracoplasty and yet it is a matter of common observation that natives in their natural environment in the native reserves who have contracted the disease often make good recoveries.

Wicht, of Cape Town Isolation Hospital obtained results which were worth while and proved that the native is fair field for trial (Lyle Cummins 1939).

At Rietfontein Hospital during the year 1943-44, 109 cases were treated; of these 50 cases died during that year and 16 were discharged to their homes.

During the year ending June, 1941, 60 successful pneumo-thorax inductions, 22 phrenic operations and three thoracoscopies with adhesions section were performed at the Ns. Cord Zulu Hospital in Durban (Annual Rept. Deptl. Public Health 1941).
In Dormer's experience (1943) he has found that native children with advanced disease respond to immobilization plaster jackets and a high protein intake.

Willoocks (1938) working in Tanganyika has noted encouraging results in early cases with pneumothorax treatment. He emphasises the necessity for early diagnosis.

Matthews (1935) in Zanzibar was able to prove to his satisfaction that native tuberculosis "did benefit materially "even under conservative treatment".

Ferguson (1932) treated a number of cases of tuberculosis adenitis conservatively and they made good recoveries.

The Tuberculosis Research Committee (1932) also noted that natives who had developed tuberculous cervical adenitis during service on the mines, frequently returned after one or two years with well healed scars and an apparently stable tuberculous complex as they were then able to fulfill a contract of service and remain in good health.

Macfadyen and Horne (1937) found that cases of tuberculous peritonitis had a good prognosis and appeared to respond well to aspiration and injection of oxygen intra-peritoneally. All their patients were poorly nourished and were given a high protein diet. Heimmann (1936) similarly noted a good response in these cases to laparotomy.

Brock (1933) emphasised the limitations of pneumothorax in the American negro. In his experience the results of this treatment are much in favour of the European. Cuttler, Rodgers and Cippes (1934) (quoted by Grasset 1944) insist on the need for early collapse therapy in negroes, delayed treatment, according to them, proving disastrous. Fischer (1937) (quoted by Grasset 1944 believes in collapse therapy in suitable cases in the American negro. He advises against its use in acute rapidly spreading cases and adopts it only in those showing a certain degree of chronicity. Chadwick (1933) is a strong believer in intervention in the treatment of tuberculosis in the negro. He believes it is a waste of time and money to attempt to control tuberculosis in the negro without first removing him from his home.

Many reports especially from French North Africa and
Canada have noted favourable results from the prophylactic use of B.C.G., but so far it has not been used in the South African native. Dormer (1943) is chary about recommending it for use as he feels there is not yet sufficient information on the type of disease following its administration.

Grasset (1944) has reported on the use of tubercle endotoxoid prepared by him. From the pathological point of view, a fibrotic transformation was observed accompanied by a clearing in more recently infiltrated areas. Of a series of 47 unselected cases treated at the non-European Hospital, Johannesburg (1939-42), suffering from acute, and in most cases, advanced forms of pulmonary tuberculosis (39 with bilateral disease, 40 with cavitation, and all with positive sputa), 32 benefitted from endotoxoid treatment and showed fibrotic changes of varying degrees. Of 14 patients who died in this series during treatment, 9 showed either radiological or postmortem evidence of fibrotic changes of the lesions including two cases of miliary tuberculosis.
THE CLINICAL MANIFESTATIONS OF TUBERCULOSIS IN SOUTH AFRICAN NATIVE.

That the clinical form of Tuberculosis and its pathological anatomy are unique in the African had been occasionally noted before. Borrel (1920) by the clarity and completeness of his description of the disease in Senegalese soldiers in France aroused interest in what he called tuberculosis in "primitive" or "virgin" soil. The African troops that were called to French Army Service during the world war came from a part of Africa where tuberculosis while not completely absent was uncommon. In his studies of the interior, the natives showed positive tuberculin reactions of 4 to 7 per cent. In the villages he found 20-30 per cent. In continental France amongst his own people there was a frequency of 60 to 90 per cent. At the time of mobilizations the Senegalese soldiers showed a positive rate of 4 to 5 per cent positive reactions. After being in a training camp for about a year, the men rapidly came down with an infection having symptoms entirely confusing to the physicians accustomed to seeing tuberculosis in the French whites. Borrel divided the course of the disease into two periods. During the initial period lasting from one to three months, there might be at first no outward symptoms or physical changes. At this time enlarged lymph glands may be found in the chest. A lymph gland in the supraclavicular region could easily be seen or palpated in about 70 per cent of cases. He emphasized the diagnostic importance of this enlarged supraclavicular gland in an otherwise healthy appearing individual. Later in the initial period the patient began to show indication of the infection. There was a change in the happy facial expression characteristic of the well Senegalese. The limbs became soft and flabby. Muscular tone was lost as well as the lustre of the healthy skin. There was a loss of weight, and a slight fever developed. Borrel called this the lymphatic stage because of a massive involvement of the lymph glands, particularly the tracheo-bronchial group; huge densities at the hilar regions were noted on X-Ray.

The second period was more definitely recognisable
as tuberculosis because it followed the usual course of generalised
Pleuritis, peritonitis and meningitis appeared at times.
The outstanding finding was the marked involvement of the lungs with
caseous pneumonia. There was little fever which increased slightly
toward the end. Neither was there tachycardia. The sputum was nearly
always negative for tubercle bacilli. The second period lasted from
1 to 3 months and the disease was uniformly fatal. Post-mortem
examinations proved that the infection was primarily of the lymph
glands and secondarily of the lungs. There was never any indication of
a fibrous reaction. The sequence of events thus reconstructed was
"primary chancre" in bronchial mucosa, invasion of lymph glands,
extension to the lungs, and finally a bacillaemia during which, as
Borrel picturesquely writes "it snows" tubercles. A complete lack of
resistance is thus indicated and Borrel rightly compares the condition
to tuberculosis in the infant or in experimentally infected guinea pigs
or rabbits.

The lack of an acquired or inherited resistance may not be
the whole explanation of the behaviour of tuberculosis in the Senegalese
soldiers. From every day experience it is known that first infection
need not be serious. The period of greatest sensitivity to the tubercle
bacillus is in the first few years of life. During this time many
individuals receive their initial dose of organisms without any apparent
harm; even primitive Africans may have primary infections without dire
results. It may quite reasonably be assumed that the Senegalese troops
in spite of the claims of the French for the hygienic conditions of the
continental military camps were exposed to massive primary infection
under adverse conditions of hard labour, possibly inadequate diet and an
unadjusted army life giving rise to this unusual form of tuberculosis.

It is noteworthy that if these cases were recognisable
in the first or "glandular" stage and placed at rest on an adequate diet,
they regained their health and 50 per cent were able to be repatriated.

As early as 1895 Symonds in Kimberley had noted a type of
disease in the Bantu which conformed clinically to Borrel's disease
"of the infant".

The Tuberculosis Research Committee (1932) came to the
conclusion that the type of disease occurring in native miners was of
the childhood type with a generalised infection of the lymphatic glands. They believe that the comparative inability of the native of any age to localize tuberculosis permanently is a much more important manifestation than his occasional ability to modify the course of the disease. The only natives who showed a significantly large incidence of European adult disease were a group of 200 tuberculo-silicotics who frequently showed extensive pulmonary fibrosis and cavity formation. On the whole the natives showed a type of disease not entirely conforming to Borrel's description, but intermediate between "primitive" disease and European adult disease.

Tuberculosis is inclined to stay within the lymphatic glands and not to break through the barriers of the capsules unless it is led to do so either by virtue of its extreme numerical strength or through its being driven on by the influences that go with hard work or unfavourable associates. (Cummins 1939).

A. Sutherland Strachan who has had a very extensive experience of the pathology of native tuberculosis, states (1945) that in his opinion there has been a change in the picture presented during the last 20 years from that of childhood or "primitive" type to a tuberculosis more closely resembling that of a tuberculised community, although the most commonly discovered type of lesion in his experience is still the exudative type.

Dormer and his co-workers (1943) in a careful survey of 10,000 natives in Natal found that in the Reserves, natives show 0.25 per cent of disease of three types:

(a) The delayed disease characteristic of Europeans;
(b) The infants had the typical, disseminated disease of all infants.
(c) The progressive primary disease or severe delayed disease in the native who had returned diseased from industrial employment.

In the mission reserves the disease rate rose to 0.75 per cent with the same types manifested in the same proportion as in the reserves. In the peri-urban areas disease rate rose to 1.5 per cent. The type of disease was found to be progressive primary disease (70 per cent) and severe delayed disease (30 per cent). In the urban areas where the disease rate rose to 1.5 to 2 per cent the type of
disease was similar to that found in the peri-urban areas.

Allan (Rept. Tub. Comm. 1932) also noted the incidence of a delayed type of tuberculosis in the native reserves.

Willcocks (1938) found a preponderance of cases of acute "gallopping" consumption in Tanganyika natives. Fibrosis was rarely noted except in the middle aged. Bilateral infiltrations were far commoner than in Europe and the lower lobes were frequently involved. Miliary spread occurred often. On the whole, the appearances, while not so gross as those seen in a "virgin" race, were not so fibrotic as those common in civilized races. He concludes that these natives therefore "lie midway between the completely primitive races and our relatively resistant selves".

F.J. Allan (1929) tuberculin tested natives recruited to the Witbank Collieries and found that during the first six months of service 37 per cent were positive; in the second six months 52 per cent and after one year 60 per cent were positive, while only very few showed evidence of tuberculosis clinically. He came to the conclusion that the primary infection can occur in the native adult without any clinical manifestations of tuberculosis.

A. Sutherland Strachan (1945.) examined most of the lungs of native miners removed at autopsy and reported on by the Tuberculosis Research Committee (1932). These lungs showed almost constantly the presence of calcified foci in the situation where the Ghon focus usually occurs. In his experience, natives may live for a considerable time after the healing process had taken place. He found that the manifestations of tuberculosis occurring in natives showing a calcified Ghon focus corresponded very closely to those occurring in non-immunized individuals.

Willcocks (1938) noted that Africans expectorate tubercle bacilli at an earlier period than do Europeans for a comparable stage of the disease. Opie in American negroes has also noted this: "Amongst the Europeans a third of all patients in any one year have had tubercle in the sputum, and among negroes a half".

**RELATIVE FREQUENCY OF THE VARIOUS CLINICAL TYPES OF TUBERCULOSIS.**

Macvicar (1908) found phthisis in 72.6 per cent and regional adenitis in 17.3 per cent of 483 cases of tuberculosis seen
by him in the Ciskei. He quotes Mc Cord of Durban as having seen 540 cases among 6,000 native patients of whom 55 per cent had phthisis and 37 per cent scrofula. He noted that tuberculous meningitis appeared to be less common among the natives than it was in Britain, but that this may be due to faulty registration. Among 300 consecutive admissions to Macvicar's hospital in 1927 there were 74 cases of tuberculous of whom 55 were pulmonary, 5 of spinal, 3 of abdominal tubercle and 1 of the genito-urinary tract.

The following table shows the incidence of the deaths from tuberculous disease in the Johannesburg General Hospital (non European section).

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>NUMBER</th>
<th>PERCENTAGE OF TOTAL T.B. CASES</th>
<th>DEATHS IN HOSPITAL</th>
<th>PERCENTAGE OF ADMISSIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lungs</td>
<td>1065</td>
<td>65.1</td>
<td>513</td>
<td>48</td>
</tr>
<tr>
<td>Miliary Tuberculosis</td>
<td>40</td>
<td>2.0</td>
<td>35</td>
<td>87.5</td>
</tr>
<tr>
<td>Meningitis</td>
<td>51</td>
<td>3.0</td>
<td>46</td>
<td>90</td>
</tr>
<tr>
<td>Abdominal</td>
<td>60</td>
<td>3.7</td>
<td>31</td>
<td>52</td>
</tr>
<tr>
<td>Spine</td>
<td>134</td>
<td>9.3</td>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>Other Bones and Joints</td>
<td>64</td>
<td>4.0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Glands</td>
<td>174</td>
<td>10.4</td>
<td>21</td>
<td>12</td>
</tr>
<tr>
<td>Disseminated Tuberculosis</td>
<td>32</td>
<td>2.0</td>
<td>23</td>
<td>72</td>
</tr>
<tr>
<td>TOTAL</td>
<td>1620</td>
<td>-</td>
<td>671</td>
<td>40</td>
</tr>
</tbody>
</table>

It was not determined whether the bone, joint and gland tuberculosis was human or bovine. (du Toit 1939).

Gillman and Gordon (1942) review the types of infection occurring in 421 cases seen in the non-European section of the Johannesburg General Hospital during 1940. Of a total of 431 cases 317 or 73.6 per cent were cases of pulmonary tuberculosis; of these 2 had unilateral disease without cavitation, 32 had unilateral disease with cavitation or bilateral disease without cavitation, and 263 had extensive bilateral disease with cavitation; 114 cases had non-pulmonary tuberculosis.
Willcocks (1935) writing of inhabitants of Tanganyika records that there exist a large number of lesions in the lower and middle zones of the lungs.

<table>
<thead>
<tr>
<th>AGE GROUP</th>
<th>UPPER AND MIDDLE ZONES</th>
<th>LOWER &amp; MIDDLE ZONES</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-15</td>
<td>4</td>
<td>14 or 77%</td>
</tr>
<tr>
<td>6-15</td>
<td>39</td>
<td>36 or 41%</td>
</tr>
<tr>
<td>16-25</td>
<td>57</td>
<td>52 or 41%</td>
</tr>
<tr>
<td>26-50</td>
<td>95</td>
<td>108 or 51%</td>
</tr>
<tr>
<td>Over 50</td>
<td>10</td>
<td>17 or 63%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>205</td>
<td>227 or 52%</td>
</tr>
</tbody>
</table>

This suggests that lesions in the lower lobes are much commoner in these natives cases than in Europeans. In 1935 the same author records the results of 446 X-ray examinations in Tanganyika natives; of these 250 showed tuberculous infiltration of the lungs. Bilateral disease was present in 68 per cent and unilateral in 37 per cent. Cavitation was observed in 111 cases, but fibrosis round the cavities was rarely found. Calcified nodules were demonstrated in 206 persons submitted to X-rays. In 105 cases calcification was found without other evidence of disease. Fibrosis was recognised in 34 per cent of the cases of definite tuberculosis. In 106 contacts 60 (57 per cent) showed in the films actual tuberculous infiltration, calcification or pleurisy. Of 242 contacts examined by him in 1935 39 per cent showed evidence of disease. He concludes that the contact "stands head and shoulders above his fellows in the risk he runs of acquiring the disease".

Of 275 cases of tuberculosis diagnosed by Matthews in Zanzibar in 1935, 269 were pulmonary cases, four were cases of cervical adenitis, and there was one case each of tuberculous peritonitis and spinal caries.

The admissions from abdominal, gland, bone and joint tuberculosis during the eight years previous to 1932 to Macvicar's hospital averaged 61 per annum, the highest figure 62, occurring in 1928 at the end of a long drought; of these spinal cases averaged 16. At the time of writing (1932) he had in hospital 45 cases of spinal tuberculosis, 5 of the hip and knee joints 2 cervical gland
and 3 of abdominal tuberculosis. During the years 1927 to 1935 inclusive 180 children 16 years and under and 90 adults 17 years and over, suffering from bone and joint tuberculosis were admitted to the Victoria Hospital Lovedale (Macvicar 1935). Age incidence was maximum from 2 to 4 years and the majority of patients were males. An analysis of the 180 cases occurring in children shows 141 spine cases, 18 of the hip joint, 13 of the knee joint. The thoracic spine was most frequently involved in tuberculous disease of the vertebrae; of the 141 cases 41 showed paraplegia of the lower limbs. Admissions from bone and joint tuberculosis constituted 20 per cent of the total admission from tuberculosis and just under 3.5% of admissions from all causes.

Grasset (1944) observes that bone tuberculosis in natives does not generally run the rapid course observed in pulmonary lesions.

The Tuberculosis Research Committee (1932) found that joint tuberculosis was fairly common in native miners but that disease of the wrist and ankle joints was only occasionally encountered.

**LENGTH OF SURVIVAL**

Once started, the evolution of the disease appears to be more rapid than in the European. The average duration of survival of cases of tuberculosis sent from the mines to the Witwatersrand Native Labour Association hospital varied from death on the day of admission to a month or 6 weeks, averaging out of 16 days (Rept. Tub. Res. Comm. 1932). Cases were observed among recruits from the East Coast especially, who had been admitted to hospital a few days after arrival with broncho-pneumonia, and who died of acute miliary tuberculosis in three weeks.

The figures submitted by four mine hospitals show that for 145 consecutive cases of death from tuberculosis the average survival period was 28 days. Practically the only cases retained in mine hospitals are those who are too sick to move. (Rept. Tub. Res. Comm. 1932). Dangerfield (1943) reviews 60 deaths from tuberculosis in native miners occurring soon after ceasing work underground.
3 cases died within one week of ceasing work, 9 in the second week, 6 in the third week, 7 in the fourth week, 18 in the fourth to the eighth week, 7 in the 8th to 12th week and 8 in the 12th to 16th week. Of the 60 deaths, 44 occurred in the mine hospitals (being presumably too seriously ill to move) and the remainder in the Central hospital awaiting repatriation. The author remarks that "these fatal results are in no way extraordinary to anyone familiar "with tuberculosis". The majority he believes, were due to acute tuberculous broncho-pneumonia or acute generalized tuberculosis, both of which are rapidly fatal.

The published articles referring to cases in the Native Reserves give a considerably longer duration of the disease. Peter Allan (Rept. Tub. Comm. 1932) found a number of cases in the Transkei where evolution of the disease was rapid, nevertheless his opinion is that "much more frequently the evolution is slow, "extending over a period of two years or longer". Of 37 fatal cases of tuberculosis seen by Maovicar at Victoria Hospital, Lovedale, 18 died within 6 months of contracting the disease, 10 within 6 to 12 months, 5 within 1 to 2 years, 3 within 2 to 3 years and 1 within 3 to 4 years.

Of 246 cases of pulmonary tuberculosis seen by Matthews (1935) in Zanzibar only 23 had suffered from the disease for more than 2 years. The average duration of the disease in those who died proved to be less than twelve months. The average period between the appearance of symptoms and death was eight months in males and eleven months in females. Of 152 cases 35 per cent died within between 2 and 10 months.

Brock (1933) found in his experience that the duration of the disease in American negro patients averaged 4 months, while in whites it averaged 10 months.

Rogers (1921) (quoted by Lewis, 1942) reported that for 75 negroes the average duration of fatal tuberculosis was 1 year and 1 month; for 75 white it was 1 year and 6 months.

Pinner and Casper (1932) (quoted by Lewis 1942) found the disease fatal for 47 negroes with an average of 324 days, while
for 96 whites they found it fatal of an average of 995 days.

**GLANDULAR TUBERCULOSIS.**

Enlarged glands in the cervical region are of common occurrence in South African natives. It is not certain what proportion is due to tuberculosis. A large percentage of coloured and poor European children in the Cape Nutrition Survey showed enlarged cervical glands. The incidence was so high that it was thought unlikely that they could be caused by tuberculosis. They were spread so widely through the neck that it was thought unlikely that they could be caused by tonsil sepsis, or to infection of the scalp secondary to pediculi. Brook (1942) finds it difficult to know how much weight to attach to such a stigma. There is no evidence that it is caused by malnutrition and yet it is not found among the best nourished children. In a survey of 800 Bantu school children in three urban areas and six rural areas, S.L. Kark (1944) found palpable cervical glands in 76.35 per cent of boys and 70.80 per cent of girls.

Ryno Smit (1945) is of the opinion that every child suffering from "glands" in the Native Reserves is notified as "Glandular" tuberculosis, in spite of the fact that there are numerous other causes of glandular enlargement.

Between 1920 and 1933 Burton (1934) examined 33,308 male natives between the ages of 18 and 45 for a native labour recruiting agency: 102 or 0.3% were rejected for enlarged glands of the neck. The glands were situated in the triangles of the neck and the sub-occipital region. Septic infection from teeth, tonsils, and vermin on the scalp accounted for a number of cases in which a tendency to abscess formation was present. Other cases had the typical appearance of tuberculosis with periadenitis, causing adhesion to the cervical fascia and sinus formation after caseation had set in. Even in the latter he was impressed by the good physique of those affected. He thinks syphilis may have accounted for some cases.

The Tuberculosis Research Committee (1932) requested
a number of South African doctors to cite their experience with glandular tuberculosis.

Macvicar in response to an enquiry as to fate of local glandular disease stated:— "In some cases the infection seems to spread to other organs, but in the majority of cases tuberculous disease remains limited to the glands and most make a good ultimate recovery. We see many people in good health with the scars of old tuberculous gland abscesses."

Dr. Peter Allan writes:— "In the Transkei I saw many natives with tuberculosis of the neck. They did not appear to be generalized, in fact, I saw several old gentlemen of 70 years of age with well marked typical cicatrices who told me that they had swollen glands which had broken down 40 years previously; after discharging for some time the glands healed up. One might reasonably compare these cases with the European child, except that the native seems to develop the condition in early adult life."

Dr. A.I. Girdwood, Chief Medical Officer - W.N.L.A. who has an unrivalled experience of the diseases of the S.A. native, has noted that natives with primary tuberculous glands of the neck do not do well on the mines. The glands invariably get bigger and eventually break down and suppurate in spite of the treatment. These cases do not, as a rule develop pulmonary or generalized tuberculosis. On the other hand natives with old healed tuberculous scars of the neck do remarkably well. It is exceptional for the disease to recur or for tuberculosis of the lungs to develop during their period of contract underground. He feels justified in saying that the mine native reacts to local glandular tuberculosis as does the European child.

If enlarged glands are discovered in labour recruits they are promptly repatriated; it was observed that many cases that had left the mines with enlargement of the cervical glands returned there for work a year or two later with a few healed scars being in excellent physical condition. Of 471 native miners developing tuberculosis after previous tuberculin testing, local
glandular infection was encountered in 100 cases. Of these 85 cases arose in the 376 positive reactors (22 per cent), 15 in 49 hyper-reactors (30 per cent) and 15 in 95 negative reactors (16 per cent). The Tuberculosis Research Committee (1932) concludes that the rarity or absence of epidemic spread of acute tubercular disease in isolated communities exposed to occasional infection is perhaps to be explained in terms of the tendency to spontaneous arrest of cases in the glandular stage, as noted by Borrel, when placed at rest and suitably dieted.
THE INCIDENCE OF BOVINE TUBERCULOSIS AMONG ANIMALS IN SOUTH AFRICA.

As appears to be the case with the native races of man, so with the native cattle, tuberculosis was rare or unknown until infected stock was introduced from other countries. The danger to indigenous stock appears to be in direct proportion to the degree of contact with foreign cattle to which they are exposed.

According to Macvicar (1908) bovine tuberculous infection was very rare among native cattle in the Eastern Province, the Transvaal, Basutoland and Southern Rhodesia. It was prevalent in the Western Province, being introduced chiefly by cattle imported from Madagascar of whom more than 80% were infected. In 1906 out of a consignment of 64 such cattle landed at Durban, no fewer than 42 reacted to tuberculin, many of them being severely affected with the disease.

As to the early history of bovine tuberculosis in the Cape Province, the chief veterinary surgeon of the Union, Mr. C.E. Gray, is quoted as stating that until 1905 he held the opinion "that for all practical purposes this disease did not exist in South Africa". It was not until 1904 according to J.D. Borthwick that the prevalence of tuberculosis in dairy herds of the Cape began to attract serious attention. (Rept. Tub. Res. Comm. 1932).

For many years the western province of the Cape supplied colonial breeding stock, much of it of the Friesland breed, to the rest of South Africa, and there is evidence that by this means the disease has been distributed in widely different parts of the Union. (Tuberculosis Commission 1914).

In 1928 Paine and Martinaglia found cases of tuberculosis occurring in wild animals living under natural conditions in the Albany District of the Cape. Two species (Kudu, Strepsiceros, and the Cape Duiker, Sylvia-capri grimmli) were found to be infected, the organisms isolated in every case being of characteristic bovine type. The infection must presumably have been derived from cattle although the incidence among the cattle in that district was very low.
In Bloemfontein the percentage of tuberculous cattle slaughtered in 1929 was 0.38 per cent per annum. In Cape Town of 8,348 cows slaughtered during 1929, 67 or 0.80 per cent showed tuberculous lesions. In Durban the average incidence of tuberculosis in slaughtered cattle was 0.053% per annum between 1921 and 1929. Of milk samples tested approximately 5% showed the presence of tubercle bacilli, and 15% of the dairy herds were positive reactors to tuberculin testing (1929). In East London only three tuberculous slaughter cattle were encountered for the three years preceding 1929. This is attributed to the fact that most of them were veld-reared animals. In Johannesburg during the period 1910-1929 when nearly 2 million animals were slaughtered, 0.079 per cent were tuberculous. During the same period there were three quarters of a million pigs slaughtered and of these 0.319% were tuberculous. In Pietermaritzburg the average incidence of tuberculosis in slaughtered cattle for 1929 was 0.065% and for pigs 1.53%. For the three years preceding 1929, 0.046% of cows slaughtered were tuberculous; and for the year 1929, in Pretoria 0.176 of slaughter cattle and 0.67% of pigs.


These figures represent a low incidence of bovine tuberculosis in slaughter cattle.

Between 1930 and 1940 a total of 178 dairy cows were found to be clinically tuberculous in dairies supplying milk in Johannesburg. In the majority of these cases lesions were present in the udders and tubercle organisms were being excreted in the milk. (Martinaglia in Annual Rept. of Director of Abattoir and Live Stock Market Dept. Johannesburg 1940). Of 38 milk samples in Johannesburg in 1940 only 1 was found to contain tubercle bacilli (Pullinger ibid 1940).

The testing of dairy herds in Natal has disclosed high percentages of animals to be infected. Thus of 152 animals tested on account of the discovery of cases of tuberculosis, 28.2 per cent were positive reactors and tuberculosis was subsequently proved by autopsy; of 56 tested without suspicion of tuberculosis 25% gave positive reactions.
Lamprocht (1944) reviewing the health of cattle in the Transkei stated that a few years previously 1,500 head of cattle had been tuberculin tested with negative results; only odd cases of disease had been noted amongst carcases inspected at the Umtata abattoir. Tuberculosis of pigs on the other hand was fairly common; of 3,100 pigs slaughtered at Umtata during the previous five years 99 were found to be suffering from tuberculosis (type unspecified).

Allan in 1924 was of the opinion that tuberculosis was widespread amongst dairy herds, especially in the Western Province where tuberculin testing yielded up to 73.9% positive reactors in one herd.
BOVINE TYPE TUBERCULOSIS CAUSING DISEASE IN HUMANS

Harvey Firie (Rept. of Tub. Res. Comm. 1932) examined bacilli isolated from 100 cases of tuberculosis in mine natives and every one was of the human type. Dormer (1943) claims that only three cases of bovine infection in human being have been recorded in South Africa. In Natal he and his associates typed 200 cultures of organism obtained from human sputum, bone abscesses, joints, and neck glands, and all were of the human type. Willcocks (1938) found in Tanganyika that of 70 strains of tubercle bacilli isolated from natives of the territory, only two were of the bovine type. Matthews in 1935 found that in Zanzibar "the predominant type of causal organism seems to be of the human type." It would appear therefore from the scanty investigations that have been undertaken, that the role of bovine tuberculosis in producing human disease is a relatively minor one.
PATHOLOGY OF NATIVE TUBERCULOSIS.

Bushnell (1920) summarized the features of "primitive" tuberculosis as follows: - a general involvement of the lymph glands as great packets of enormously enlarged caseated and suppurating organs; involvement of the lung with caseous broncho-pneumonia; caseous labor pneumonia or a gelatinous pneumonia; primary involvement of the serous membranes; tuberculosis of the liver, spleen, kidneys, myocardium, pericardium and often the endocardium; acute intestinal ulcers and finally terminal miliary tuberculosis of the lungs.

As early as 1895 H. Symonds practising in Kimberley, remarked on the salient features of the pathology of native tuberculosis. "The condition found in the lungs is more that described as acute pneumonic phthisis, large caseating masses being found, involving often whole lobes of the lung with immense caseating bronchial glands. The difference (from the European) lies in the fact that similar large caseating masses are found in other parts of the body especially the spleen and often the liver, kidneys and pericardium, and nearly always general tuberculous peritonitis with ascites". He observed that "phthisis as a rule runs a much more acute course in the native".

In the Report of the Tuberculosis Research Committee (1932) on a number of autopsies performed in mine natives, 538 cases were found to have died of tuberculosis. Numerous cases were encountered which concern to Bushnell's description of primary tuberculosis, a general infiltration of the lymph glands, manifesting itself often as great packets of enormously enlarged caseating and suppurating glands. Caseation was very common, but more common than suppuration was some hardening of the glands, microscopic examination showing a mixture of caseation, some fibrosis and considerable proliferation of endothelial cells, suggesting "the occurrence of tuberculous infection not in a perfectly "natural" condition, but in a subject slightly though very imperfectly immunized". A terminal miliary spread was commonly seen when there was extensive caseation in one
in one of the gland groups. Tuberculous lobar pneumonia (or gelatinous pneumonia) was rather rare in the series, only two characteristic cases being seen, to which could be added 6 or 7 cases of confluent broncho-pneumonia involving the whole or nearly the whole of one lobe; of the whole series of 538 cases dying from tuberculosis 1 per cent showed tuberculous nodular myocarditis and 15 per cent tuberculous pericarditis, as compared to Zone Clark's figures (quoted by Bushnell 1920) from a series of 452 autopsies amongst the West Indian negroes at Panama, showing 3.3 per cent and 14 per cent respectively. In the same series the figures for the mine natives for involvement of the spleen, liver and kidneys were 61.52 and 28 per cent as compared to Clark's figures 58.52 and 42 per cent respectively. Probably a considerable number of cases had an evolution similar to that seen by Borrel in Senegalese troops in France. The majority of these cases occurred in the cases seen in the Mine Medical Officer's series, the records shown of some cases which died within a month of their arrival on the Rand, and of the others who had been working up to within a few days of their death. Borrel states that the initial lesions almost always occur in the glands draining the upper air passages (90 per cent); this was found to be a comparative rarity in the mine series. Borrel found the seat of the primary lesion to be in the lungs in not more than 5 per cent of cases whereas in the mine series the figure was over 30 per cent. It was at times difficult to state which was the primary lesion - the heavily involved tracheo-bronchial glands or the caseous lungs. The number of cases showing pulmonary cavities must be taken as indicating that many of the mine series of cases ran a somewhat longer course than did the majority of Borrel's. Only 6 of 241 cases mainly with thoracic lesions showed evidence of the formation of fibrous tissue round their cavities to an extent at all comparable to ordinary European phthisis. That healing or arrest of primary pulmonary lesions does occur was evidenced by the findings amongst 62 cases of death from conditions other than tuberculosis of 5 cases showing such features. Borrel also distinguished a group of cases where the lesions, secondary to an initial glandular focus, were predominantly in the serous sacs. This could be abundantly paralleled
in the mine series. In a mine series of 97 cases where the lesions were mainly extrathoracic, only 6 appeared to have originated as an invasion by the abdominal route. In 75 it was felt that the spread had occurred definitely from the tracheo-bronchial glands. In the mine series of 15 cases showing bone or joint lesions, only 2 appeared not to be obviously primarily glandular. "To sum up, it may be said that the acute, progressive tuberculosis as seen mainly in the younger natives early in their mining career although partly "natural" tuberculosis, is somewhat modified and has reached a stage intermediate between the completely "natural" tuberculosis of virgin soil and the "modified" tuberculosis of the European adult". Five cases recorded in the mine series showed the first gross lesion in the spleen. Unusual features in the acute cases were five instances of tuberculous involvement of the pancreas and two possible cases of primary involvement of the spleen where the lesions were entirely confined to this organ. A series of 200 cases of tuberculosis with silicosis are reviewed; more than half the cases fell into the age group 40-49 and fully two thirds into the age period 30-49; the average length of mining service was 6 years. It was noted that there was much fibrous tissue formation in the lungs and that tuberculosis was comparatively frequently limited to the lungs, although it may have spread to other organs affected by silicosis more than half the cases showed cavities and only 37 developed miliary tuberculosis. With a few exceptions tuberculo-silicosis furnished the only cases of "chronic" tuberculosis seen in the total series. In the elderly native, apart from the influence of silicosis, tuberculosis tends to become generalized practically as much as in the younger native. Nine cases of oesophageal fistulae were noted in relation to breaking down tuberculo-silicotic tracheo-bronchial glands.

Two hundred postmortems on the mine natives (half from the Witwatersrand Native Labour Association Hospital and half from the mine hospitals) were compared with 200 postmortems performed at the City of London Hospital for Diseases of the Heart and Lungs, Victoria Park, London. The following points are commented on:— Glands: cervical glands were rarely involved in the native as compared to the
European, but there was a strikingly more common involvement of the retrosternal and diaphragmatic, and portal and retroperitoneal glands in the native. Lungs and Pleurae: Lung lesions were more extensive as well as more constantly present in the European. Scarring, fibrosis, pleural adhesions, caseation and cavities, are all obviously more striking features in the European. Definite caseous deposits were more common in the native. Heart and Pericardium: of 35 European cases only one was an actual case of tuberculous pericarditis, the others being cases of excess fluid or of slight adhesions. In the native series there were 31 cases of tuberculous pericarditis or myocarditis and 6 cases of adhesions. Abdominal organs: Involvement of the liver and spleen was very much more common in the native and in the spleen especially the degree of involvement was much greater. Tuberculous peritonitis was also more common in the native. Laryngeal involvement was present in 43 of the European series but was observed in only 2 natives, possibly owing to the more acute nature of illness. Amyloid disease was noted in 13 Europeans but was not present in any native.

A comparison of 25 cases of tuberculosis in Cape Coloured people (University of Cape Town) with the series of 200 native and 200 European cases showed the type of tuberculosis much nearer that of the native, but intermediate between them. Of the 25 cases, 15 were under the age of 15 and 10 over that age. All the cases with chronic lesions of the lungs occurred in the group over 15 years of age. The suggestion is that amongst adult Cape Coloured people the disease is of the European adult type.

The percentage involvement of various organs in 338 consecutive cases of acute tuberculosis in the gold mines was noted to be: (241 cases in which the lesions were mainly thoracic and 97 mainly extrathoracic).

<table>
<thead>
<tr>
<th>Glands</th>
<th>%</th>
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<tbody>
<tr>
<td>Cervical</td>
<td>16%</td>
</tr>
<tr>
<td>Clavicular</td>
<td>21%</td>
</tr>
<tr>
<td>Diaphragmatic</td>
<td>31%</td>
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<tr>
<td>Pancreatic</td>
<td>48%</td>
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<tr>
<td>Axillary</td>
<td>1</td>
</tr>
<tr>
<td>Inguinal</td>
<td>0.3%</td>
</tr>
</tbody>
</table>

Tracheo 68%
Bronchial 68%
Lower retroperitoneal 30%
Lungs:

Caseation
Caseation with excavation
Milia only
25) 81%
30)
26)

Pleurae:

Definite tuberculous pleurisy 32%

Heart and Pericardium:

Definite tuberculous pericarditis or myocarditis
17% Adhesions, possibly tuberculous 4%

Spleen
Liver
Kidney
Suprarenals
Peritoneum
Intestine
Brain and Meninges
Bone or Joint
Genitalia
70%
61%
31%
10%
31%
21%
2.5%
4%
1.5%


During 1922-1928 inclusive 1,402 autopsies were performed on native mine workers of a total of 1,718 deaths mainly in the medical wards (W.O. Fischer 1929); of these 302 cases were found in which the principal cause of death was tuberculosis, i.e. 21.5 per cent of all autopsies;

42 had General T.B.
48 had Pulm. T.B.
30 had Pulm. T.B. and silicosis
22 had abdominal T.B.
2 had T.B. of the kidney
3 had T.B. pericarditis
7 had T.B. meningitis
2 had Tuberculosis of the Brain
2 had T.B. abscess of liver
2 had T.B. of the vertebrae
136 had Miliary T.B.

The diseased lungs (256 cases) revealed in 104 cases 40.8 per cent) the lesions of chronic phthisis, and in 152 cases (59.2 per cent) those of the acute form. The chronic form was in 30 cases associated with more or less pronounced silicosis; of the acute cases 91.5 per cent were of the caseating broncho-pneumonic type, whilst 8.5 per cent belonged to the lobar type of tuberculous pneumonia.

A remarkable feature of the majority of cases of miliary tuberculosis was the great enlargement of the spleen, the organ attaining in 56 cases (41.1 per cent) a weight of 500 grammes and
more; in 6 cases (5.9 per cent) even more than 1,000 grammes. The size of the tubercles in the spleen ranged from scarcely visible white spots to the size of a bean or even a walnut. Nearly all the cases occurred in young people.

Reviewing 561 consecutive autopsies performed on mine natives, subsequent to his first series, W.O. Fischer (1932) found a slightly reduced number of deaths due to tuberculosis (16.04 per cent) as compared to 21.5% previously. Acute miliary tuberculosis was found in 50 per cent; this was due to the fact that all cases of tuberculosis are repatriated if they are able to travel and that the cases which come to autopsy are predominantly fulminating.

In 1,000 consecutive postmortems performed on natives in Nairobi, Kenya, by Vint (1936) 132 cases were found to have died of tuberculosis. The organs were affected in the following percentages: Lungs 94, Liver 82, Spleen 43, Thoracic and Mesentric glands 42, Kidneys, 30, Cerebral and Meningeal 29, Heart 12, Bones and Joints 2; and all the above organs were found to be affected in 5 per cent of cases; 67 of the 132 cases suffered from miliary tuberculosis, cavity formation was found in the lungs in 37 cases. It was rare to find microscopic evidence of reactionary fibrosis round pulmonary tuberculous lesions. The postmortem findings were those of a low grade septicaemia resembling acute miliary tuberculosis in European children. There were 4 cases of tuberculosis of the intestines and mesentric glands without a primary lesion in the lungs. In the remainder of the 1,000 cases where tuberculosis was not the cause of death, 46 showed active tuberculosis and 14 healed lesions.

A. Sutherland Strachan (1945) who has had a very extensive experience of tuberculosis in natives, states that the lesions are commonly of the exudative type but that he has noted during the last 20 years that there appears to be developing more of the proliferative type of reaction. The typical lesions occurring in the American negro are massive lymph node caseation, massive exudative lesions which do not respect the normal anatomical boundaries of organs and a type of generalized tuberculosis without the formation of miliary tubercles but with irregularly scattered nodular, exudative foci. (Lewis 1942).
BANTU MORTALITY RATES FOR TUBERCULOSIS.

The European death rate per 100,000 shows a steady decline from 45.93 in 1920 to 33.16 in 1943 for tuberculosis (all forms). In the absence of vital statistics it is not possible to give Bantu mortality rates.

The estimated number of non-European deaths from tuberculosis in 1942 was 15,000 (Union Health Dept. Annual Report 1942). This would place the rate between 180 and 210 per 100,000.

In 1941 the mortality rate in Indians was five times, in the Bantu seven times and in coloured ten times the European death rate. (Dormer 1943). According to this estimation the rate for natives would be in the vicinity of 240 per 100,000.

S. Lyle Cummins (1939) quotes a prevalence rate of 2,907 per 100,000 and a mortality rate of 2,219 for 100,000 for the South African Labour Corps in France during the Great War.

The Transvaal colliery figures for tuberculosis for 1926-1927 show a prevalence of 383 per 100,000 with a case mortality of 26 per cent. (Rept. T.B. Res. Comm 1932). The medical Officer for Cape Town in his annual report, returns the non-European mortality from tuberculosis for 1926 at 430 per 100,000.

The Tuberculosis Officer for Durban returns the death rate from tuberculosis at 160 per 100,000 for 1925 with a prevalence rate of 358 per 100,000. The death rate in Pietermaritzburg for the years 1927-1932 for pulmonary tuberculosis for Europeans was 2.7 and for natives 9.3 per cent of all deaths during this period. (Anning 1933).

The incidence of notified cases and the mortality rates for natives in the Johannesburg municipality for all forms of tuberculosis in 1940 per 100,000 population were 259 and 162 respectively; for 1941, 285 and 175; for 1942, 386 and 178; for 1943, 381 and 190; for 1944 the mortality rate per 100,000 was 231. It is worthy of note that there has been an increase of forty per cent in the mortality rate since the first year of the war.

Of the position of tuberculosis in the United States, Louis I. Dublin (1920), statistician to the Metropolitan Life Ins. Coy., New York, wrote:- "Tuberculosis of the lungs is the most
important cause of death among the coloured people. The death rate is more than twice as high among the insured negroes as among the white policy holders. The disease is a veritable scourge among young negroes. At the ages between 10 and 14 years, the tuberculosis death rate among the coloured boys is eleven times as high as it is among the white boys of the same age. Coloured girls at the same age period show a death rate eight times greater than white girls. Tuberculosis is pre-eminently a disease of young persons but it is especially so among coloured people". And in 1924:— "The pessimism which prevailed 25 years ago with regard to the future of the negroes, is no longer even remotely justified. A race still living in many areas under primitive conditions of sanitation and often from hand to mouth is today enjoying a life expectancy just about that of the white people of America only 30 or 40 years ago. The negroes are only a generation behind the achievement of the white people of this country".

In the United States the death rate of coloured people reached its maximum figure in the 1850's, amounting certainly to between 600 and 700 per 100,000, and probably 900 per 100,000. Immediately after the Civil War in a disrupted disorganised South it was difficult for both whites and negroes to obtain gainful occupations. The general sufferings of reconstruction days hit the coloured people in their most vulnerable spot, their susceptibility to tuberculosis. From the time of these high mortality figures to the present time, there has been a remarkable decline in deaths from tuberculosis among the negroes. While the curve of mortality showing this decrease roughly parallels the decline of the curve for whites there is actually a slight lag in the fall of the coloured curve. It has been predicted that this breach will continue to increase until the white mortality curve begins to flatten out as the disease approaches extinction at which time the coloured rates will make greater relative changes. Prior to 1925 tuberculosis was the leading cause of death among the negroes. Since then it has been exceeded by heart disease. In white people tuberculosis now stands sixth place among diseases, being exceeded by heart disease, cancer, nephritis, cerebral haemorrhage and pneumonia. The general average
death rate in 1934 for negroes was in the United States 146.4 per 100,000 as compared to 45.1 per 100,000 for whites, the ratio between the two being 3.2 : 1. (Lewis (1942).
INCIDENCE OF TUBERCULOUS INFECTION AS DETERMINED
BY TUBERCULIN SURVEYS.

Previous to 1912 the extent of tuberculous infection among
the African natives was known only by the clinical evidence of the
disease. Since one of the outstanding characteristics of tuber-
culos is in most natives is the lack of outward signs, even in the
presence of massive disease, clinical observation alone is insuffi-
cient to determine the extent of infection. It was Calmette who
probably more than anyone else popularised the tuberculin reaction.
He pointed out its usefulness as a tool both in the practice and in
the investigation of tuberculosis and introduced it in making tuber-
culos surveys in Africa. He and his colleagues in this way covered
most of the French African Colonies and disclosed the variable
amounts of tuberculosis in natives and the relation of these amounts
to certain factors. Their work was followed by many similar surveys
in other parts of the continent.

It is doubtful whether all the tuberculin surveys made
among the natives of Africa are comparable, because the same dilution
of tuberculin was not used in each case nor was there a uniform
method of carrying out the test. Moreover old tuberculin deterior-
ates rapidly in the tropics and this fact might have vitiated some
of the results. (Lewis 1942).

Willcocks (1938) believes that tuberculin rates provide
good indices not only of the amount of infection but also of the
amount of actual tuberculous disease.

Calmette (1912) (quoted by Lyle Cummins 1939) using the
von Pirquet test found 80 - 90 per cent positive reactors in West
African natives, 3.4 per cent in the Ivory Coast, 7 to 9.3 per cent
in Madagascar, in Reunion after long and intimate admixture with
the French, 5 per cent positive reactors, and in French North Africa
52.8 per cent reactors.

Ziemann (1912) (quoted by Lyle Cummins 1939) found 4.4
per cent of Bantu soldiers and 1.6 per cent of women and children
from various tribes in German South West Africa to be positive
reactors. Of 80 natives from the interior all save one who had
been a soldier were negative.
Muller (1914) (quoted by Lyle Gumms 1939) found that 200 of 600 natives in a hospital were positive, at Tanga, German East Africa, in the midst of a population where Indians were well represented.

Nagga, Bay and Shukry (1938) found that Egyptians are as heavily tuberculized as are the English and Welsh.

Maynard (1912) found that of 544 native labourers arriving at the Witwatersrand mines, 129 Nyasa boys gave 4 per cent positive reactions to Calmette's ophthalmic tuberculin test; and of 415 Mozambique natives 1.7 per cent gave a positive reaction, an average of 2.4 per cent. None of these boys had previously been on the mines. Of 115 tropical natives examined who had previously been on the mines 15 per cent gave a positive result, coinciding exactly with the figure obtained by Calmette in his native France. Of 131 natives returning home after one trip to the mine 19.8 per cent gave positive results.

Working among the Dinkas of Bahr-el-Ghazal, Burrows (1935) found 32.7 per cent positive reactors in 3,662 persons tested.

Matthews (1935) found 100 per cent of adult natives in Zanzibar to react positively.

Willcocks (1935) found that of 1,037 natives of all ages tested in Tanganyika Territory 47 per cent were positive. In a later survey (1938) he found the positive rate varied from 46 to 80 per cent with the highest incidence along the coastal areas and lowest inland.

Peter Allan (Rept. of Tub. Res. Comm. 1932) found that of 513 natives of all ages tested at Lusikisiki (coastal) 81 per cent were positive reactors; at Butterworth near the coast, of 3,933, 76 per cent were positive; at Matatiele of 1,211 tested 40 per cent reacted positively and in Basutoland of 851 tested 50 per cent were positive.

Generally speaking at 5 years of age a third gave positive reactions; at 10 years slightly more than a half reacted positively, and over 25 years 90 per cent had encountered their primary infection. The southern group of natives showed a higher tuberculization rate
than the northern. Positives were even in two sexes except for girls who showed a higher rate between ages of 5 and 15 years, a difference which was soon made up when the young men left their homes to look for work.

Macvicar in 1928 tuberculin tested 728 pupils at Lovedale College and found 77 per cent of them to be positive reactors.

At the instigation of the Tuberculosis Research Committee (1932) 93,979 natives applying for work on the Witwatersrand gold mines were tuberculin tested with varying strengths of intra-dermal old-tuberculin. It was found that 65 per cent were positive reactors. Among all the natives tested 566 developed tuberculosis (all forms), 452 cases being observed in the positive reactors and 114 cases in the negative reactors, representing 738 and 347 per hundred thousand respectively. Of 3,879 hyper-reactors 60 developed tuberculosis and of 57,236 reactors 391 developed the disease, representing 1,547 and 683 per one hundred thousand respectively. Among the hyper-reactors there was a tuberculosis rate of 10.3, among the ordinary positives 6.5, and among the negatives a rate of 2.9 per thousand. They concluded that the incidence rate was more than twice as high in the positive as it was in the negative group; that the more strongly positive the reaction to tuberculin, the greater was the liability to disease; and that the negative reactors had the highest resistance to disease. The rapidly fatal type of generalised tuberculosis occurred most frequently in the negative reactors.

In a large survey carried out by Dormer and his colleagues in Natal in 1935 and reported in 1943 it was found that in the Natal Native Reserves 40 - 50 per cent of Natives were positive reactors and the incidence of active disease was 0.25 per cent; the corresponding figures for Mission Reserves were 50-60 and 0.80, for peri-urban areas 70 and 1-1.5, and for urban areas 70-80 and 1.5 - 2.

At 5 years of age 55 per cent of natives were found to react positively, at 10 years 46 per cent, at 15 years 60 per cent, at 20 years 60 per cent and at 25 years 82 per cent.

Surveys of population groups with tuberculin and X-Ray have been made in sufficient numbers to show fairly adequately the comparative frequency of infection among the natives and white. In
general the difference between the two races has not been significant.
In most cases the native rate has been slightly higher than that of
the whites, but the excess is not of the same order as the excess of
native deaths over white deaths.

HYPERSensitivity TO TUBERCULIN.

There is much evidence that tested in the same way Africans
are more sensitive to tuberculin than Europeans. For example
Willcocks (1935) points out that in normal Londoners a reduction in
strength of tuberculin from 1:1,000 to 1:10,000 resulted in the
reduction in the positive reactions of 25 per cent. In Tanganyika
natives a reduction in strength from 1:5,000 resulted in a reduction
of positive reactions of only 3 per cent. Likewise Allan (Rept. T.B.
Res. Comm. 1932) in South Africa showed that a reduction of the
dilution of tuberculin from 1:5,000 to 1:10,000 made no difference
either in the character of the reactions or in the proportion between
positive and negative tests. Actually the proportion of positive
reactions was fractionally higher with 1:10,000 than 1:5,000. With 1:
100,000 dilution the percentage of positive reactors was reduced about
17 per cent; with 1:1,000,000 dilution there was little difference
in the percentage as obtained with 1:100,000, but there was some
change in the character of the reactions. With a dilution of
1:10,000,000 there was a reduction of 3 per cent from the percentage
obtained with 1:1,000,000 and 24 per cent reductions from that ob-
tained with 1:5,000. In a follow up of hyper-reactors it was found
that there was a disease incidence of 10.3 per thousand whilst in
the ordinary positive reactors the rate was only 6.5 per thousand.

Eagleton (in an unpublished paper quoted to S.Lyle Cummings
1939) early noted the marked reactivity of American Negroes to tuber-
culin.

Willcocks (1935) observed vesiculation in 6.1 per cent
of positive reactors among Tanganyika natives and in 3.2 per cent
of the population tested. In contacts it was as high as 14 per cent.
He quotes D'Aroy Hart as finding vesiculation in 1 per cent of people
examined in England and in 2.3 per cent of his positive reactors.
Cummins (1939) states that the Bantu reaction is one of "sensitivity - allergy" while that of the European is one of "immune - allergy". The native unlike the European does not readily acquire a state of increased resistance coincidently with a rise in allergy (Pinner and Casper 1932 - quoted by S. Lyle Cummins 1939). The Tuberculosis Res. Comm. (1932) remarked that in native miners on the Rand high tuberculo-allergy went with poor resistance to infection and that it placed the subject in a dangerous condition, whereas, in moderation, it may be protective.

The degree of hyper-sensitiveness to tuberculin is not a measure of the degree of immunity nor is it even a measure of the extent of the infection. (S. Lyle Cummins) Immunity indeed, is found to be so low that the pathological picture typically observed in the Bantu is quite unlike that seen in the European.

H.N. Davies (1939) has also observed this hyper-sensitivity in Tanganyika natives and believes that it predisposes to the flaring up of infection into active disease under unfavourable conditions. Willecocks (1938) suggests that it would be wise where tuberculin testing of labour recruits is carried out to prohibit strong reactors from heavy work without a period of close observation.
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