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EPIDEMIOLOGICAL STUDY OF TUBERCULOSIS IN MACASSAR CAMP

BY

ASHRAF MOHAMMED

BSc (Hons)

THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE (EPIDEMIOLOGY)

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SUPERVISOR: DR F.R. PRINSLOO

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DECEMBER 1995

DECLARATION

I the undersigned hereby declare that the work contained in this thesis/study project is my own work (original) and has not previously in its entirety or in part been submitted at any university for a degree.

SIGNATURE: .....

DATE: 30 NOVEMBER 1995

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"Reading maketh a full man,
conference a ready man,
and writing an exact man."

—— *Francis Bacon*

ABSTRACT
(ENGLISH & AFRIKAANS)

EPIDEMIOLOGICAL STUDY OF TUBERCULOSIS IN MACASSAR CAMP

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"The joy I felt at the prospect before me of being the instrument destined to take away from the world one of its greatest calamities ..... was so excessive that I sometimes found myself in a kind of reverie."

Drewitt FD (1931)
Life of Edward Jenner
Longmans, Green & Co.,
London.

ABSTRACT

EPIDEMIOLOGICAL STUDY OF TUBERCULOSIS IN MACASSAR CAMP

By: Mr Ashraf Mohammed

The aim of this study was to determine and evaluate the prevalence of TB infection, active TB cases and the risk factors associated with TB infection in Macassar Camp in Macassar (about 40 km from Cape Town on the False Bay coast, with a population of 369). The study design of this epidemiological study was a cross sectional study with a descriptive and an analytic component.

A comparison between the Mantoux, TB ELISA and X-ray screening tests was performed first. A description of the origin, discovery, characteristics and pathology associated with Mycobacterium tuberculosis as well as the development of the TB epidemic on a global, national and local level, is given. TB was first described to give a South African perspective of the TB epidemic and both the "Virgin Soil" and "Non-Virgin Soil" theory of TB was reviewed.

Secondly, the TB infection rate in Macassar Camp and the risk factors as well as the determinants of TB infection with regards to overcrowding, ventilation, primary food subsistence level rating (PFSL), social class and employment status were evaluated.

The third aspect of the study compares prevalence/incidence rates of TB to clinical diagnosis with regards to the

symptomatology, radiographs, sputum microscopy, bacteriology and Mantoux test. Lastly the Mantoux test was compared with the TB ELISA test with regards to diagnosis of infection, in new and past confirmed TB cases.

The first part of the survey involved the measurement of openable window area and the floor area of each Camp dwelling (to determine if ventilation was within required limits), during the administration of a household questionnaire which was designed to determine the number of occupants, rooms, income, food expenditure per household in the Camp. A personal questionnaire was administered to all Macassar Camp residents to elicit information on demography, knowledge and attitudes to TB, history of past TB, TB contacts, alcohol intake and smoking habits, occupation and BCG status.

The Mantoux test were performed on consenting Camp residents in addition to the collection of 5 ml of blood for the TB ELISA tests. The Camp residents heights and weights were recorded prior to the miniature mass chest radiographs being taken. The 'TB suspects' sputa were collected for the microscopy and bacteriological examination. A review of the clinical records of TB patients in the Macassar/Stellenbosch area was also undertaken.

The response rate to the household questionnaire was 60 from

63 (95,2%) dwelling units. Whereas the response rate to the personal questionnaire was 296 (80,2%). As for the Mantoux and TB ELISA tests the response rate was 209 (56,6%). Of the 60 dwelling units, 43 (71,7%) were calculated (according to Batsons Index) to be crowded and 16 (26,7%) dwelling units had an overall ventilation of less than 5% (below the required regulation).

There were significantly ($p<0,005$) more male than female smokers and only 78 (34,2%) of the residents regarded themselves as non-smokers. A similar trend was noted with regards to the alcohol intake of the residents, where only 86 (37,7%) regarded themselves as teetotallers, with significantly more ($p=0,003$) male than female alcohol consumers. Females scored significantly ($p=0,002$) better than the males with regards to TB knowledge and awareness.

Only 199 (67,2%), residents indicated that they had had BCG vaccination. Of the 296 residents responding to the survey, there were 83 children aged 14 years or less. And only 74 of these children were confirmed to have been vaccinated with BCG, resulting in a 89,2% BCG coverage.

Two (4,7%) of the 43 children aged 14 years or less were determined to be malnourished on the basis of Z-scores (below -2SD) taking into account height for age as well as weight for height.

However, on reviewing the Z-scores based on weight for age, there were 3 (7,0%) children determined to be malnourished. Amongst those aged 15 years or older, 37 (22%) residents were to be malnourished, based on the Body Mass Index (BMI standard: men <20; women <19).

There was no significant difference amongst those residents that were Mantoux positive and malnourished as compared to those that were Mantoux negative and not malnourished. However, there were significantly more residents who were Mantoux positive and below the PFSL as compared to those residents who were Mantoux negative and above the PFSL and (p=0,009; RR=1,92; 95% CI=1,13-3,26; n=209). The effect of occupational status on infection (Mantoux positive) was significant. Thus those residents that were unskilled and partly skilled were at greater risk of infection than those that were skilled (p=0,12; RR=2,68; 95% CI=1,13-6,35;n=114)

There were 67 (32,1%), residents that were confirmed to be infected based on the Mantoux test. The overall annual risk of infection (ARI) of TB in the Camp (all ages) was 1,8 and 0,4 (lowest) in the age group of 0-9 years and 1,8 (highest) in the age group 20-29 years. There were significantly more Mantoux positives amongst females than males (p=0,035; RR=1,55; 95% CI=1,02-2,36; n=209). The 'gender factor' was even more pronounced in females aged less than 15 years (p=0,009; RR=4,77; 95% CI=1,18-19,27; n=43).

Furthermore, it was confirmed that 'TB contacts', had a significantly higher chance of being infected as compared to those who were not classified as 'TB contacts' (p=0,007; RR=1,98; 95% CI=1,27-3,08; n=196). In children aged less than 15 years who were classified as 'TB contacts', risk of infection was even more pronounced as compared to those children who were not 'TB contacts' (p=0,027; RR=3,25; 95% CI=1,45-7,27;n=43).

The outcome of Mantoux results were significantly different among female residents who experienced 1 or more miscarriage as compared to those females who did not have a miscarriage. Mantoux positive females who were matched for age and race with 2 females who were Mantoux negative showed that there was a significant difference in comparison to those who were Mantoux negative and did not have miscarriages at all (p=0,023; RR=2,38, 95% CI=1,28-4,42; n=66).

Those residents that were subjected to overcrowded dwelling units as well as exposed to overall ventilation of less than 5%, had a significantly greater number of infected residents than those that were not subjected to this combined factor (p=0,016; RR=2,82; 95% CI=1,12-7,06; n=57). This study also showed that regular participation in sport/exercise appeared to have had a protective effect (RR<1) against TB infection (Mantoux positive) as compared to residents who were 'not exposed' to regular sport/exercise (p=0,025; RR=0,53; 95% CI=0,29-0,97; n=164).

There were 9 (2,4%) Camp residents that had been treated for TB prior to this study. Of the 275 mass miniature X-rays performed, 37 were regarded as 'TB suspect' and 32 were requested to have X-rays repeated at the clinic in addition to sputum microscopy and culture resulting in 6 cases of active TB (including 1 TB relapse case) being confirmed. Based on these results the prevalence of PTB for 1988, in the population of Macassar Camp was 1 355/100 000.

The number of infected individuals detected by the TB ELISA tests was 20 (9,6%) as compared to the 67 (32,1%) detected by positive Mantoux tests. Comparison of TB ELISA and Mantoux tests with regards to the infection rate, denoted poor agreement (Kappa statistic = 0,070). The TB ELISA tests gave a 94,5% specificity and a 100% sensitivity for newly confirmed PTB cases. There was no correlation between TB ELISA and Mantoux tests.

With regards to screening for TB, the TB ELISA as screening test, gave the highest positive predictive value (45,5%), with a 100% sensitivity at a 94,5% specificity, when compared to the X-ray (positive predictive value of 25,0% with a 100% sensitivity at 93,0% specificity); and Mantoux test (positive predictive value of 4,5% with a 60% sensitivity at 68,6% specificity).

In conclusion, it can unequivocally be stated that there is a TB epidemic raging in the Western Cape and the incidence

in Macassar/Firgrove is amongst the highest in the Western Cape Province. Factors such as household contact, overcrowding & poor ventilation, living below the primary food subsistence level, malnutrition, smoking, unskilled work and gender (female) were some of the factors associated with the residents vulnerability to TB infection.

With advent of the HIV pandemic, infection and development of disease will take place more rapidly. With more than half of the South African population harbouring dormant Mycobacterium tuberculosis and/or MOTTS, the number of TB cases will increase, as has been noted since 1986 when the effects of AIDS were first noted in South Africa. The problem could be compounded by patients receiving irregular or inadequate TB treatment, resulting in an increase of MDR-TB patients. Pressure at hospitals and clinics could result in patients with resistant Mycobacterium tuberculosis not being treated with the proper supervision thus further spreading the infection and paving the way for a deadly TB epidemic.

If a concerted effort is not made immediately to combat TB on a nationwide basis, the new South Africa will be ill-prepared to deal with these problems. This will result in new South Africa facing a disaster and a national emergency of the magnitude not yet imaginable.

ABSTRAK (Afrikaans)

EPIDEMIOLOGIESE STUDIE VAN TUBERKULOSE IN DIE MACASSARKAMP

Deur: Ashraf Mohammed

Die doel van die studie was om die voorkoms van TB-infeksie, aktiewe TB-gevalle en die risiko faktore, geassosieer met TB-infeksie, te bepaal en te evalueer. Macassar is ongeveer 40 km vanaf Kaapstad aan die Valsbaai-kus met 'n bevolking van 369. Die studie-ontwerp van hierdie epidemiologiese studie was 'n dwarssnit met 'n beskrywende en analitiese komponent.

'n Vergelyking tussen die Mantoux, TB ELISA-en X-straal-siftingtoetse is eers uitgevoer. 'n Beskrywing van die oorsprong, ontdekking, kenmerke en patologie geassosieer met *Mycobacterium tuberculosis*, asook die ontwikkeling van die TB epidemie op 'n globale, nasionale en plaaslike vlak, word gegee. TB word eers beskryf om Suid Afrikaanse perspektief van die TB- epidemie te gee, en beide die "Virgin Soil"-en "Non-Virgin Soil"-teorië van TB hersien.

Ten tweede, is die voorkoms van TB-infeksie in Macassarkamp en die risiko faktore, sowel as die determinate van TB-infeksie met die oog op oorbewoning, ventalasie, skatting van die primêre voedsel onderhoudsvlak (PFSL), maatskaplike klas en beroepstatus, ge-evalueer.

'n Derde aspek van die studie vergelyk die voorkomssyfer van TB met kliniese diagnose m.b.t. simptomatologie,

X-straalplate, sputum mikroskopie, bakteriologie en die Mantoux-toets. Laastens is die Mantoux-toets vergelyk met die TB ELISA-toets met betrekking tot diagnose van infeksie in nuwe en vorig bevestigde TB gevalle.

Die eerste dedeelte van die opname het die meting van die oopmaakbare venstergebied en die vloeroppervlak van elke Kampwoning behels, om te vas te stel of ventilasie binne die vereiste spesifikasies was. Dit was geadministreer deur 'n huishoudelike vraelys te verskaf om die getal inwoners, kamers, inkomste en voedseluitgawe te bepaal. 'n Persoonlike vraelys is ook aan alle Macassar kamp-inwoners verskaf om inligting te verkry t.o.v. demografie, kennis van en houding t.o.v. TB, geskiedenis van vorige TB, TB-kontakte, alkohol-inname en rookgewoontes, bewoning en BCG-status.

Die Mantoux-toets is, met hulle toetstemming, uitgevoer op inwoners van die Kamp; 5 ml bloed is getrek vir die TB ELISA-toetse. Kamp-inwoners se lengte en gewig is opgeteken voor die miniatuur-borskars X-straalfoto's geneem is. Sputum van die TB 'verdagte' is geneem vir mikroskopiese en bakteriologiese ondersoeke. 'n Oorsig van die kliniese rekords van TB pasiënte in die Macassar/Stellenbosch gebied is ook onderneem.

Antwoordsyfers op die huishoudelike vraelys was vir 60 uit

63 (95,2%) huishoudings, terwyl die antwoordsyfers van die persoonlike vraelyste 296 (80,2%) was. Vir die Mantoux-en TB-ELISA toetse was die antwoordsyfers 209 (56,6%). Uit die 60 wooneenhede was 43 (71,7%) as oorbevolk bereken (na die Batson Indeks), en 16 (26,7%) wooneenhede het a totale ventilasie van minder as 5% gehad (onder die vereiste regulasie).

Daar was beduidend meer rokers onder mans as vrouens ($p<0,005$) en net 78 (34,2%) van die inwoners het hulself as nie-rokers beskou. Dieselfde neiging is gesien ten opsigte van alkohol- inname onder inwoners waar net 86 (37,7%) hulself as geheelonthouers beskryf terwyl daar beduidend meer manlike ($p=0,003$) as vroulike alkoholverbruikers was. Vrouens ($p=0,002$) het beter as mans vertoon t.o.v. TB kennis en bewustheid.

Net 199 (67,2%) van die inwoners het aangedui dat hulle BCG-inenting gehad het. Van die 296 respondente was 83 kinders van 14-jarige ouderdom en jonger. Net 74 van hierdie kinders was bevestigde BCG-inentings (89,2% BCG-dekking).

Twee (4,7%) van die 43 kinders met ouderdom 14 jaar of jonger was ondervoed op die Z-teller basis (onder -2SD), waar hoogte en gewig vir ouderdom in ag geneem is.

In die ondersoek van die Z-teller basis van gewig vir ouderdom was daar egter 3 (7,0%) kinders wat ondervoed was

Van die inwoners van 15 jaar en ouer, het 37 (22%) aan wanvoeding gely, gebaseer op die Liggaamsgewig-Indeks.("Body Mass Index" - BMI standaard: mans <20; vrouens <10).

Daar was geen beduidende verkil tussen inwoners wat Mantoux-positief getoets en aan wanvoeding gely het nie, in verlyking met Mantoux-negatief getoetse inwoners wat nie aan wanvoeding gely het nie. Daar was egter beduidend meer inwoners wat Mantoux-positief getoets en onder die PFSL was, in vergelyking met Mantoux-negatiewes en bokant die PFSL (p=0,009); RR=1,92; 95% CI-1,13-3,26; n=209). Die effek van beroepstatus of infeksie (Mantoux-positie) was beduidend. Inwoners wat ongeskoold en gedeetelik geskoold was, het 'n groter risiko vir infeksie gehad as geskoolde inwoners (p=0,12; RR=2,68; 95% CI=1,13-6,36,n=114).

Sewe en sestig (32,1%) van die inwoners was positief vir die Mantoux-toets. Die algehele jaarlikse risiko vir infeksie (ARI - annual risk of infection) vir TB in die Kamp (alle inwoners) was 1,8 en 0,4 (laagste) in die ouderomsgroep 0-9 jaar, en 1,8 (hoogste) in die ouderdomsgroep 20-29 jaar. Daar was beduidend meer Mantoux-positiewes onder vrouens as mans (p=0,035; RR=1,55; 95% CI-1,02-2,36; n=209). Die geslagsfaktor was selfs meer betekenisvol in vrouens jonger as 15 jaar (p=0,009; RR=4,77; 95% CI=1,18-19,27; n=43).

Verder was dit bevestig dat TB-kontakte 'n beduidende hoër

kans gehad het om geïnfekteer te raak in vergelyking met nie-geklassifiseerde TB kontakte (p=0,007; RR=1,98; 95%CI=1,27-3,08; n=196). In kinders jonger as 15 jaar wat as TB-kontakte geklassifiseer is, was die risiko vir infeksie meer uitgesproke in vergelyking met kinders wat nie TB-kontakte was nie (p=0,027; RR=3,25; 95% CI=1,45-7,27; n=43).

Resultate van die Mantoux-toets was beduidend verskillend onder vroulike inwoners wat een of meer miskrame gehad het, teenoor vrouens wat geen miskrame gehad nie. Mantoux-positiewe vrouens wat vir ouderdom en ras gemeet is en vergelyk is met twee vrouens, negatief getoets, vir die Mantoux-toets, het 'n beduidende verskil aangetoon in vergelyking met Mantoux-negatiewes en die wat geen miskrame gehad het nie (p=0,023; RR=3,23; 95% CI=1,28-4,42; n=66).

Inwoners oderwerp aan oorbewoonde behuisings-eenhede, asook blootgestel aan totale ventilasie van minder 5%, het beduidend meer geïnfekteerde inwoners gehad as as dié wat nie onderwerp was aan hierdie gekomineerde faktor nie (p=0,016; RR=2,38; 95% CI=1,12-7,06; n=57). Hierdie studie het ook aangetoon dat gereelde deelname aan sport/oefening 'n beskermende effek (RR<1) teen TB infeksie (Mantoux-positief) gehad het, teenoor inwoners wat 'nie blootgestel' was aan gereeld sport/oefening nie (p=0,025; RR=0,53; 95% CI=0,29-0,97; n=164).

Nege (2,4%) inwoners van die Kamp is voorheen vir TB getoets. Van die 275 miniatuur X-straal-ondersoeke wat uitgevoer is, was 37 beskou as 'TB verdagtes' en 32 is gevra om weer X-straal-ondersoeke by die kliniek te ondergaan, sowel as sputum mikroskopiese en-kultuurondersoeke, wat 6 bevestigde gevalle van aktiewe TB opgelewer het (ingeslote 1 TB terugval-geval). Gebaseer op hierdie resultate was die prevalensie vir PTB vir 1988 (vir inwoners van Macassarkamp) 1 355/100 000).

Die getal geïnfekteerde individue opgespoor deur die TB ELISA-toetse was 20 (9,6%) teenoor die 67 (32,1%) opgespoor deur positiewe Mantoux-toets. 'n Swak ooreenkoms (Kappa statistiek = 0,070) is aangetoon met vergelyking tussen TB ELISA-en Mantoux-toetse m.b.t. die infeksiekoers. TB ELISA-toetse het 94,5% spesifisiteit aangetoon en 'n 100% sensitiwiteit vir nuutbevestigde PTB-gevalle. Daar was geen korrelasie tussen TB ELSA en Mantoux-toetse nie.

Wat sifting vir TB betref, het die TB ELISA die hoogste positiewe voorspelbare waarde (45,5%) met 'n 100% sensitiwiteit en 94,5% spesifisiteit, aangetoon; teenoor die X-straaltoetse (positiewe voorspelbare waarde van 25,0% met 'n 100% sensitiwiteit by 93,0% spesifisiteit), en Mantoux-toets (positiewe voorspelbare waarde van 4,5% met 60% sensitiwiteit by 68,6% spesifisiteit).

Ten slote kan ondubbelsinnig verklaar word dat daar 'n TB

-epidemie in Wes-Kaap woed, en dat die insidensie in Macassar/Firgrove van die hoogste in die Wes-Kaapprovinsie is. Faktore soos huishoudelike kontak, oorbewoning en swak ventilasie, wanvoeding, 'n lewensbestaan onder die primêre voedselonderhoudsvlak, rook ongeskoolde werk en geslag (vroulik), was sommige van die faktore geassosieer met die inwoners se vatbaarheid vir TB-infeksie.

Met die verskyning van die HIV-pandemie sal infeksie en ontwikkeling van die siekte al hoe vinniger plaasvind. Waar die dormante *Mycobacterium tuberculosis* en/of MOTTS in meer as die helfte van die Suid-Afrikaanse bevolking verskuil is, sal die getalle van TB-gevalle toeneem, soos waargeneem vanaf 1986 toe die effek van VIGS vir die eerste in Suid-Afrika opgemerk is. Die probleem kan gekompliseer word waar pasiënte ongereelde of onvoldoende TB-behandeling ontvang, met gevolglike toename van MDR-TB pasiënte. Druk by hospitale en klinieke kan veroorsaak dat pasiënte met weerstandige *Mycobacterium tuberculosis* nie behandel word onder die nodige toesig nie, sodat die infeksie daardeur verder sal versprei word en die weg baan vir a dodelike TB-epidemie. As 'n gesamentlike poging nie dadelik aangewend word om TB op 'n nasionale basis te bestry nie, sal die nuwe Suid-Afrika onvoorbereid wees om hierdie probleme die hok te slaan. Dit sal 'n gevolglike ramp in die nuwe Suid-Afrika veroorsaak en 'n nasionale noodtoestand van ongeëwenaarde aard.

ACKNOWLEDGEMENT

"True gratitude within the heart
evades the simplest word;
It fills the soul with glow and warmth
and struggles to be heard.

It seeks expression in a smile,
a 'thank you' most sincere;
It strives to pierce formalities,
its purpose high and clear.

Yet it finds fulfillment
and one will know who tries,
In just the firmer clasp of hands,
and grateful shining eyes."

Mabel Jones Gabbott

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DEDICATION

THIS THESIS IS

DEDICATED

TO THE MEMORY

OF

SHAYKH YUSUF

THE FIRST RESIDENT

OF

MACASSAR

WHOSE TRICENTENARY WAS

COMMEMORATED ON 2 APRIL 1994

&

ALSO TO THE FIRST RESIDENT

TO HAVE DIED OF TUBERCULOSIS

IN

MACASSAR CAMP

JUST PRIOR TO THE COMMENCEMENT OF THIS STUDY

THE
ULTRA-MICROSCOPIC PERSPECTIVE

"All that I have seen teaches me
to trust the Creator
for all I have not seen."

Anonymous

Mycobacterium tuberculosis observed by means of an electron microscope. Magnification X 100,000

SOURCE:

Crispen, RG; Biological & ultrastructural characteristics of Mycobacterium tuberculosis cells & cell components. Northwestern University, Medical School, PhD thesis, 1967.

LIST OF TABLES

Now go, write it before them in
a table, and note it in a book.

—— *Isaiah 30:8*

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LIST OF FIGURES

"I saw to my astonishment that they had the appearance and all the other characters of the mysterious cultures."

Robert Koch

NOTE

This statement by Robert Koch, was made, in the lecture where he enunciated his famous postulates, and in the context of his discovery, stated that the avian and mammalian tubercule bacilli were different. Because the morphology of the two types of tubercule bacteria was the same, he earlier on assumed it was the same organism.

Source:

Koch R. (1890). "Bacteriology & its Results." Lecture delivered at First General Meeting of the Tenth International Medical Congress, Berlin. Trans. TW Hime. Bailliere, Tindall & Cox, London.

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LIST OF APPENDIXES

Sermons on brevity and chastity are about equally effective. Verbal promiscuity flows from poverty of language and obesity of thought, and from an unseemly haste to reach print — a premature ejaculation as it were.

Eli Chernin

LIST OF APPENDIXES

CHAPTER 1
INTRODUCTION

"Read (Proclaim)!
In the name of thy Lord & Cherisher,
Who created – created Man,
Out of a mere clot of congealed blood.
Read (Proclaim)!
For thy Lord is Most Beneficent.
Who has taught the use of the pen,
Has taught Man, that which he knowth not."

Al Quran
Sura 96: 1–5

CHAPTER 1

INTRODUCTION

SUMMARY

This chapter commences by introducing the causative organism (*Mycobacterium tuberculosis*) of TB. It briefly outlines the history of Man's control of pestilence and disease (even some of the most formidable diseases). Man has even successfully eradicated some of these diseases such as small pox.

TB has been associated with Man since time immemorial and yet continues to flourish in many developing countries. With the advent of HIV/AIDS pandemic, the previous decline of TB incidence in some of the developed and developing countries has actually been reversed in the past few years. TB is a major cause of disease and death in South Africa and PTB accounts for 56% of all notifiable medical conditions and is major health problem especially amongst the Black and Coloured communities. The pending TB epidemic in the Western Cape was already noted in 1908 and by 1991 in the Western Cape the TB prevalence rate had reached an all time high of 1 134 per 100 000 population (in certain suburbs)
TB is preventable and curable. Why then, has the TB problem remained unsolved for so long in South Africa?

Perhaps the answer lies in a more direct focus on contributing factors such as malnutrition, stress and socioeconomic status that may have been associated with TB infection, the development of open infectious cases of TB, aspects of active case finding and case holding and a well motivated programme of TB treatment for the successful implementation of an effective and efficient TB Control Programme. Furthermore the key to the conquest of TB lies in the strategy whereby emphasis should be placed also on the dormant *Mycobacterium tuberculosis* in healthy individuals Approximately 10 million South Africans are infected with this pathogen. An estimated 15% of the pool of infected persons, could develop active TB during their lifetime.

The best single indicator for the evaluation of the trend of TB in developed and the developing countries is the annual rate of TB infection.

The way forward would be the Primary Health Care Model which is comprehensive and intersectoral. Finally this chapter concludes by motivating reasons for the study in Macassar.

CHAPTER 1

INTRODUCTION

1 THE CAUSAL AGENT OF TUBERCULOSIS (TB)

More than a century ago, Robert Koch isolated and identified *Mycobacterium tuberculosis* as the causal agent of tuberculosis (TB). TB is now a curable and often preventable disease as can be noted by the decline in the TB epidemics from many developed countries. Present available control measures should be able to sufficiently alter the course of the TB epidemic in South Africa to lead to a rapid decline in the incidence of TB. Why then has this problem, which can be solved, continued to mushroom into such a serious problem in South Africa? **[14]**

Infection with *Mycobacterium tuberculosis* may or may not lead to the development of active TB. After infection, a primary complex is formed within 4-12 weeks which may heal or may become calcified but still retain viable bacteria. These dormant bacilli may reactivate when the immune system is compromised by immunosuppressant conditions such as malnutrition, stress, or overcrowding which are mainly a result of the socioeconomic status of the individual.

Perhaps, in South Africa there has been an over-emphasis of medical science which has diverted attention from the real underlying causes of ill health such as poverty. This has been further compounded by numerous other factors, which have undermined attempts to control TB.[20]

2 MANKIND VERSUS INFECTIOUS DISEASE

Pestilence of many sorts has brought death and misery to mankind throughout history. Over the last 100 years however, most formidable infectious diseases have been brought under control. An example of not only control but total eradication of an infectious disease is smallpox. However, TB formerly known as "The Great White Plague", defies modern medical science and continues to flourish especially in developing countries.[1]

One can take the liberty of renaming TB: "The Last of the Great Plagues" and one which is likely to increase in seriousness within the next few years especially with the advent of the AIDS pandemic and the HIV interaction with TB.

3 AN OVERVIEW OF TB

TB has been associated with human settlements since time immemorial. As yet there is no evidence

of where and when Man first became afflicted with TB. However, there is evidence of TB in Man, during the Neolithic period.[1,2,3] In the Western World this dreaded disease (TB) had become an insignificant problem until the current HIV/AIDS epidemic. However, TB remains a major threat in all developing countries and is one of the world's most prevalent infectious diseases. Of the 3 million or more people dying every year of TB, more than 80% are from developing countries. With the advent of HIV/AIDS pandemic and its interactions with TB, the previous decline of TB incidence in some of the developed and developing countries (such as USA and Tanzania) has actually been reversed in the past few years.

A report presented on World Health Day, 1964, acknowledged that TB was a major problem in the developing countries and it was estimated then that this disease was claiming more than 10 million TB victims and resulting in excess of 3 million deaths annually.[4] Further 1 out of every 150-200 persons were suffering from infectious TB in a population of 2 000 million from the developing countries (predominantly Latin-America, Africa and Asia). This was not only a serious risk to all contacts but increased the

infected pool with long term consequences now being observed on an increasing scale compounded by factors such as the lack of basic health facilities mentioned in the Declaration of Alma-Ata.

The TB problem in South Africa was compounded by the large scale influx of tuberculotic immigrants into the country thus increasing the TB infected pool. And according to Dr Scholtz in his book, [8] which promoted South Africa as a climatic cure for TB: "... this might in the near future seriously affect the population of South Africa ...." [5,6,7]

4

MORTALITY & MORBIDITY TB IN SOUTH AFRICA

TB is a major cause of disease and death in South Africa. In 1988, there were 58 502 (which includes all population groups) notified cases of pulmonary tuberculosis (PTB) in South Africa. [9] PTB accounts for 56% of all notifiable medical conditions and was a major health problem amongst the Black and Coloured communities. [10] In the same year there were 6 956 (all races) registered TB deaths. In 1987 a study showed that there was a rising incidence of TB (amongst the Coloureds and Blacks) in the Western Cape. [11,12,13]

These morbidity and mortality figures for TB are reported despite the promise of the Alma-Ata Declaration and the fact that TB is potentially preventable and curable provided that the disease is diagnosed in the early stages. Why then, has the TB problem remained unsolved for so long in South Africa? [14]

The present TB morbidity and the mortality figures in South Africa (Western Cape) are regarded as one of the highest in the world. As far back as 1906, TB was causing great concern and alarm. At a conference convened in 1906 in Cape Town, by Medical Officers of Health, [15] the gravity of the TB problem was noted and the danger threatening the Black and Coloured population.

Nearly ninety years later, we are still a long way from solving the TB problem in this country. A far cry, from a catchy slogan I once spotted as graffiti in a township:

"FREEDOM = NO TB DEATHS
WILL THE AFRICAN NATIONAL CONGRESS (ANC)
DELIVER THE GOODS?"

This slogan is vaguely reminiscent of the phrase that was popularized by Dr Basil Dormer, in the middle of this century: "A meal a day will keep TB

away." Somehow, as we are about to enter the 21st century, this phrase has undergone a metamorphosis to suit the present 'climate' in the country. Only time will tell what the next stage of the 'metamorphosis' of this phrase will be in years to come.

TB, for many of the underprivileged and poor who form the majority of the South African population is considered a dreaded deadly disease reminiscent of the attitude to the TB epidemic during the Industrial Revolution. The stigma attached to this disease within the community and attitudes of employers have further compounded this problem. It is evident that these attitudes can be attributed to ignorance, poor level of education and the socioeconomic and political situation in the country.

Robert Koch, the discoverer of the causal agent (*Mycobacterium tuberculosis*) of TB aptly summed up for posterity the TB morbidity and mortality:[16] "If the number of victims which a disease claims is the measure of its significance, then all diseases, particularly the most dreaded infectious diseases, such as bubonic plague, Asiatic cholera, must rank far behind TB."

Today, with the advent of the AIDS pandemic, Koch's statement, made more than 100 years ago remains true. Bubonic Plague (Black Death) of the 14th century; the Great Plague of London (1664-65); and the plague pandemic (1896-1917) will look like child's play due to the TB mortalities as a result of AIDS. Those who are infected with the tubercle bacillus which in most individuals remains dormant, (Appendix A1), will have a high risk of endogenous reactivation into active TB in the event of HIV infection which is the prelude to AIDS. Thus HIV will take up the role of the 'agent provocateur' in the already beleaguered communities afflicted with the TB epidemic.

This will have a far reaching implications and impact on the TB Control Programme which is presently struggling to cope with the TB epidemic in most developing countries. The current data has already indicated a vastly increased mortality rate in the HIV/TB patients.[17]

CAUSES OF TB

Despite tremendous growth in knowledge and technological advances in the areas of diagnosis, treatment and prevention,[18] we have failed to control TB in South Africa. Perhaps the answer to

our failure in the TB Control Programme in this country, lies in the fact that we have desperately tried to combat the effect of this disease rather than "attacking the root cause" of TB, which has been rampant in this country for more than a century.

To attribute the worldwide TB epidemics to *Mycobacterium tuberculosis* alone would be a gross underestimation of a more complex and an intricate problem, that all developing countries and to some extent developed countries have to contend with. Following infection, TB has a varying incubation period and the majority of those infected may never develop this disease at all. This suggests that there are other variables such as malnutrition, stress and socioeconomic status that contribute to the development of open infectious cases of TB. In South Africa, Apartheid[20] was an additional factor in the unabated attack of this microscopic organism named *Mycobacterium tuberculosis*, on humanity.

The problem of TB in South Africa, was put in its proper holistic perspective by the former Medical Officer of Health (MOH) of Cape Town in 1988, Dr Reg Coogan, who was interviewed on South African Television, (SABC) on the then NETWORK programme,

on the topic of TB:[21] "Tuberculosis is a socioeconomic related disease. It's 'caused' by poor living conditions, by overcrowding, by damp housing, by malnutrition, by strain ..... Until one eliminates these socioeconomic causes, there is no medical cure of TB. All doctors can do is treat the actual cases of TB and try to prevent the spread. One will not eliminate the disease without eliminating the 'cause' in the first instance."

However, for those persons who have developed TB through infection reactivation and/or relapse, the aspects of active case finding and case holding and a well motivated programme of treatment are very important to the successful implementation of an effective and efficient TB Control Programme.

An excellent example of the treatment of TB as a preventative measure as well can be noted from Dormer's innovative idea in 1959. Dr Basil Dormer in 1959, encouraged tuberculous mothers to breastfeed their babies thus protecting their infants with INH from the day of their birth.[19] This innovative manner of treatment of the mother and baby resulted in a preventative measure as well and saving lives of countless infants.

EPIDEMIOLOGICAL STUDY OF TUBERCULOSIS IN MACASSAR CAMP

BY

ASHRAF MOHAMMED

BSc (Hons)

THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE (EPIDEMIOLOGY)

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DEPARTMENT OF COMMUNITY HEALTH

AT THE UNIVERSITY OF STELLENBOSCH

SUPERVISOR: DR F.R. PRINSLOO

CO-SUPERVISOR: PROF P.R. DONALD

DECEMBER 1995

DECLARATION

I the undersigned hereby declare that the work contained in this thesis/study project is my own work (original) and has not previously in its entirety or in part been submitted at any university for a degree.

SIGNATURE: .....

DATE: 30 NOVEMBER 1995

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"Reading maketh a full man,
conference a ready man,
and writing an exact man."

—— *Francis Bacon*

ABSTRACT
(ENGLISH & AFRIKAANS)

EPIDEMIOLOGICAL STUDY OF TUBERCULOSIS IN MACASSAR CAMP

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"The joy I felt at the prospect before me of being the instrument destined to take away from the world one of its greatest calamities ..... was so excessive that I sometimes found myself in a kind of reverie."

Drewitt FD (1931)
Life of Edward Jenner
Longmans, Green & Co.,
London.

ABSTRACT

EPIDEMIOLOGICAL STUDY OF TUBERCULOSIS IN MACASSAR CAMP

By: Mr Ashraf Mohammed

The aim of this study was to determine and evaluate the prevalence of TB infection, active TB cases and the risk factors associated with TB infection in Macassar Camp in Macassar (about 40 km from Cape Town on the False Bay coast, with a population of 369). The study design of this epidemiological study was a cross sectional study with a descriptive and an analytic component.

A comparison between the Mantoux, TB ELISA and X-ray screening tests was performed first. A description of the origin, discovery, characteristics and pathology associated with Mycobacterium tuberculosis as well as the development of the TB epidemic on a global, national and local level, is given. TB was first described to give a South African perspective of the TB epidemic and both the "Virgin Soil" and "Non-Virgin Soil" theory of TB was reviewed.

Secondly, the TB infection rate in Macassar Camp and the risk factors as well as the determinants of TB infection with regards to overcrowding, ventilation, primary food subsistence level rating (PFSL), social class and employment status were evaluated.

The third aspect of the study compares prevalence/incidence rates of TB to clinical diagnosis with regards to the

symptomatology, radiographs, sputum microscopy, bacteriology and Mantoux test. Lastly the Mantoux test was compared with the TB ELISA test with regards to diagnosis of infection, in new and past confirmed TB cases.

The first part of the survey involved the measurement of openable window area and the floor area of each Camp dwelling (to determine if ventilation was within required limits), during the administration of a household questionnaire which was designed to determine the number of occupants, rooms, income, food expenditure per household in the Camp. A personal questionnaire was administered to all Macassar Camp residents to elicit information on demography, knowledge and attitudes to TB, history of past TB, TB contacts, alcohol intake and smoking habits, occupation and BCG status.

The Mantoux test were performed on consenting Camp residents in addition to the collection of 5 ml of blood for the TB ELISA tests. The Camp residents heights and weights were recorded prior to the miniature mass chest radiographs being taken. The 'TB suspects' sputa were collected for the microscopy and bacteriological examination. A review of the clinical records of TB patients in the Macassar/Stellenbosch area was also undertaken.

The response rate to the household questionnaire was 60 from

63 (95,2%) dwelling units. Whereas the response rate to the personal questionnaire was 296 (80,2%). As for the Mantoux and TB ELISA tests the response rate was 209 (56,6%). Of the 60 dwelling units, 43 (71,7%) were calculated (according to Batsons Index) to be crowded and 16 (26,7%) dwelling units had an overall ventilation of less than 5% (below the required regulation).

There were significantly ($p<0,005$) more male than female smokers and only 78 (34,2%) of the residents regarded themselves as non-smokers. A similar trend was noted with regards to the alcohol intake of the residents, where only 86 (37,7%) regarded themselves as teetotallers, with significantly more ($p=0,003$) male than female alcohol consumers. Females scored significantly ($p=0,002$) better than the males with regards to TB knowledge and awareness.

Only 199 (67,2%), residents indicated that they had had BCG vaccination. Of the 296 residents responding to the survey, there were 83 children aged 14 years or less. And only 74 of these children were confirmed to have been vaccinated with BCG, resulting in a 89,2% BCG coverage.

Two (4,7%) of the 43 children aged 14 years or less were determined to be malnourished on the basis of Z-scores (below -2SD) taking into account height for age as well as weight for height.

However, on reviewing the Z-scores based on weight for age, there were 3 (7,0%) children determined to be malnourished. Amongst those aged 15 years or older, 37 (22%) residents were to be malnourished, based on the Body Mass Index (BMI standard: men <20; women <19).

There was no significant difference amongst those residents that were Mantoux positive and malnourished as compared to those that were Mantoux negative and not malnourished. However, there were significantly more residents who were Mantoux positive and below the PFSL as compared to those residents who were Mantoux negative and above the PFSL and (p=0,009; RR=1,92; 95% CI=1,13-3,26; n=209). The effect of occupational status on infection (Mantoux positive) was significant. Thus those residents that were unskilled and partly skilled were at greater risk of infection than those that were skilled (p=0,12; RR=2,68; 95% CI=1,13-6,35;n=114)

There were 67 (32,1%), residents that were confirmed to be infected based on the Mantoux test. The overall annual risk of infection (ARI) of TB in the Camp (all ages) was 1,8 and 0,4 (lowest) in the age group of 0-9 years and 1,8 (highest) in the age group 20-29 years. There were significantly more Mantoux positives amongst females than males (p=0,035; RR=1,55; 95% CI=1,02-2,36; n=209). The 'gender factor' was even more pronounced in females aged less than 15 years (p=0,009; RR=4,77; 95% CI=1,18-19,27; n=43).

Furthermore, it was confirmed that 'TB contacts', had a significantly higher chance of being infected as compared to those who were not classified as 'TB contacts' (p=0,007; RR=1,98; 95% CI=1,27-3,08; n=196). In children aged less than 15 years who were classified as 'TB contacts', risk of infection was even more pronounced as compared to those children who were not 'TB contacts' (p=0,027; RR=3,25; 95% CI=1,45-7,27;n=43).

The outcome of Mantoux results were significantly different among female residents who experienced 1 or more miscarriage as compared to those females who did not have a miscarriage. Mantoux positive females who were matched for age and race with 2 females who were Mantoux negative showed that there was a significant difference in comparison to those who were Mantoux negative and did not have miscarriages at all (p=0,023; RR=2,38, 95% CI=1,28-4,42; n=66).

Those residents that were subjected to overcrowded dwelling units as well as exposed to overall ventilation of less than 5%, had a significantly greater number of infected residents than those that were not subjected to this combined factor (p=0,016; RR=2,82; 95% CI=1,12-7,06; n=57). This study also showed that regular participation in sport/exercise appeared to have had a protective effect (RR<1) against TB infection (Mantoux positive) as compared to residents who were 'not exposed' to regular sport/exercise (p=0,025; RR=0,53; 95% CI=0,29-0,97; n=164).

There were 9 (2,4%) Camp residents that had been treated for TB prior to this study. Of the 275 mass miniature X-rays performed, 37 were regarded as 'TB suspect' and 32 were requested to have X-rays repeated at the clinic in addition to sputum microscopy and culture resulting in 6 cases of active TB (including 1 TB relapse case) being confirmed. Based on these results the prevalence of PTB for 1988, in the population of Macassar Camp was 1 355/100 000.

The number of infected individuals detected by the TB ELISA tests was 20 (9,6%) as compared to the 67 (32,1%) detected by positive Mantoux tests. Comparison of TB ELISA and Mantoux tests with regards to the infection rate, denoted poor agreement (Kappa statistic = 0,070). The TB ELISA tests gave a 94,5% specificity and a 100% sensitivity for newly confirmed PTB cases. There was no correlation between TB ELISA and Mantoux tests.

With regards to screening for TB, the TB ELISA as screening test, gave the highest positive predictive value (45,5%), with a 100% sensitivity at a 94,5% specificity, when compared to the X-ray (positive predictive value of 25,0% with a 100% sensitivity at 93,0% specificity); and Mantoux test (positive predictive value of 4,5% with a 60% sensitivity at 68,6% specificity).

In conclusion, it can unequivocally be stated that there is a TB epidemic raging in the Western Cape and the incidence

in Macassar/Firgrove is amongst the highest in the Western Cape Province. Factors such as household contact, overcrowding & poor ventilation, living below the primary food subsistence level, malnutrition, smoking, unskilled work and gender (female) were some of the factors associated with the residents vulnerability to TB infection.

With advent of the HIV pandemic, infection and development of disease will take place more rapidly. With more than half of the South African population harbouring dormant Mycobacterium tuberculosis and/or MOTTS, the number of TB cases will increase, as has been noted since 1986 when the effects of AIDS were first noted in South Africa. The problem could be compounded by patients receiving irregular or inadequate TB treatment, resulting in an increase of MDR-TB patients. Pressure at hospitals and clinics could result in patients with resistant Mycobacterium tuberculosis not being treated with the proper supervision thus further spreading the infection and paving the way for a deadly TB epidemic.

If a concerted effort is not made immediately to combat TB on a nationwide basis, the new South Africa will be ill-prepared to deal with these problems. This will result in new South Africa facing a disaster and a national emergency of the magnitude not yet imaginable.

ABSTRAK (Afrikaans)

EPIDEMIOLOGIESE STUDIE VAN TUBERKULOSE IN DIE MACASSARKAMP

Deur: Ashraf Mohammed

Die doel van die studie was om die voorkoms van TB-infeksie, aktiewe TB-gevalle en die risiko faktore, geassosieer met TB-infeksie, te bepaal en te evalueer. Macassar is ongeveer 40 km vanaf Kaapstad aan die Valsbaai-kus met 'n bevolking van 369. Die studie-ontwerp van hierdie epidemiologiese studie was 'n dwarssnit met 'n beskrywende en analitiese komponent.

'n Vergelyking tussen die Mantoux, TB ELISA-en X-straal-siftingtoetse is eers uitgevoer. 'n Beskrywing van die oorsprong, ontdekking, kenmerke en patologie geassosieer met *Mycobacterium tuberculosis*, asook die ontwikkeling van die TB epidemie op 'n globale, nasionale en plaaslike vlak, word gegee. TB word eers beskryf om Suid Afrikaanse perspektief van die TB- epidemie te gee, en beide die "Virgin Soil"-en "Non-Virgin Soil"-teorië van TB hersien.

Ten tweede, is die voorkoms van TB-infeksie in Macassarkamp en die risiko faktore, sowel as die determinate van TB-infeksie met die oog op oorbewoning, ventalasie, skatting van die primêre voedsel onderhoudsvlak (PFSL), maatskaplike klas en beroepstatus, ge-evalueer.

'n Derde aspek van die studie vergelyk die voorkomssyfer van TB met kliniese diagnose m.b.t. simptomatologie,

X-straalplate, sputum mikroskopie, bakteriologie en die Mantoux-toets. Laastens is die Mantoux-toets vergelyk met die TB ELISA-toets met betrekking tot diagnose van infeksie in nuwe en vorig bevestigde TB gevalle.

Die eerste dedeelte van die opname het die meting van die oopmaakbare venstergebied en die vloeroppervlak van elke Kampwoning behels, om te vas te stel of ventilasie binne die vereiste spesifikasies was. Dit was geadministreer deur 'n huishoudelike vraelys te verskaf om die getal inwoners, kamers, inkomste en voedseluitgawe te bepaal. 'n Persoonlike vraelys is ook aan alle Macassar kamp-inwoners verskaf om inligting te verkry t.o.v. demografie, kennis van en houding t.o.v. TB, geskiedenis van vorige TB, TB-kontakte, alkohol-inname en rookgewoontes, bewoning en BCG-status.

Die Mantoux-toets is, met hulle toetstemming, uitgevoer op inwoners van die Kamp; 5 ml bloed is getrek vir die TB ELISA-toetse. Kamp-inwoners se lengte en gewig is opgeteken voor die miniatuur-borskars X-straalfoto's geneem is. Sputum van die TB 'verdagte' is geneem vir mikroskopiese en bakteriologiese ondersoeke. 'n Oorsig van die kliniese rekords van TB pasiënte in die Macassar/Stellenbosch gebied is ook onderneem.

Antwoordsyfers op die huishoudelike vraelys was vir 60 uit

63 (95,2%) huishoudings, terwyl die antwoordsyfers van die persoonlike vraelyste 296 (80,2%) was. Vir die Mantoux-en TB-ELISA toetse was die antwoordsyfers 209 (56,6%). Uit die 60 wooneenhede was 43 (71,7%) as oorbevolk bereken (na die Batson Indeks), en 16 (26,7%) wooneenhede het a totale ventilasie van minder as 5% gehad (onder die vereiste regulasie).

Daar was beduidend meer rokers onder mans as vrouens ($p<0,005$) en net 78 (34,2%) van die inwoners het hulself as nie-rokers beskou. Dieselfde neiging is gesien ten opsigte van alkohol- inname onder inwoners waar net 86 (37,7%) hulself as geheelonthouers beskryf terwyl daar beduidend meer manlike ($p=0,003$) as vroulike alkoholverbruikers was. Vrouens ($p=0,002$) het beter as mans vertoon t.o.v. TB kennis en bewustheid.

Net 199 (67,2%) van die inwoners het aangedui dat hulle BCG-inenting gehad het. Van die 296 respondente was 83 kinders van 14-jarige ouderdom en jonger. Net 74 van hierdie kinders was bevestigde BCG-inentings (89,2% BCG-dekking).

Twee (4,7%) van die 43 kinders met ouderdom 14 jaar of jonger was ondervoed op die Z-teller basis (onder -2SD), waar hoogte en gewig vir ouderdom in ag geneem is.

In die ondersoek van die Z-teller basis van gewig vir ouderdom was daar egter 3 (7,0%) kinders wat ondervoed was

Van die inwoners van 15 jaar en ouer, het 37 (22%) aan wanvoeding gely, gebaseer op die Liggaamsgewig-Indeks.("Body Mass Index" - BMI standaard: mans <20; vrouens <10).

Daar was geen beduidende verkil tussen inwoners wat Mantoux-positief getoets en aan wanvoeding gely het nie, in verlyking met Mantoux-negatief getoetse inwoners wat nie aan wanvoeding gely het nie. Daar was egter beduidend meer inwoners wat Mantoux-positief getoets en onder die PFSL was, in vergelyking met Mantoux-negatiewes en bokant die PFSL (p=0,009); RR=1,92; 95% CI-1,13-3,26; n=209). Die effek van beroepstatus of infeksie (Mantoux-positie) was beduidend. Inwoners wat ongeskoold en gedeetelik geskoold was, het 'n groter risiko vir infeksie gehad as geskoolde inwoners (p=0,12; RR=2,68; 95% CI=1,13-6,36,n=114).

Sewe en sestig (32,1%) van die inwoners was positief vir die Mantoux-toets. Die algehele jaarlikse risiko vir infeksie (ARI - annual risk of infection) vir TB in die Kamp (alle inwoners) was 1,8 en 0,4 (laagste) in die ouderomsgroep 0-9 jaar, en 1,8 (hoogste) in die ouderdomsgroep 20-29 jaar. Daar was beduidend meer Mantoux-positiewes onder vrouens as mans (p=0,035; RR=1,55; 95% CI-1,02-2,36; n=209). Die geslagsfaktor was selfs meer betekenisvol in vrouens jonger as 15 jaar (p=0,009; RR=4,77; 95% CI=1,18-19,27; n=43).

Verder was dit bevestig dat TB-kontakte 'n beduidende hoër

kans gehad het om geïnfekteer te raak in vergelyking met nie-geklassifiseerde TB kontakte (p=0,007; RR=1,98; 95%CI=1,27-3,08; n=196). In kinders jonger as 15 jaar wat as TB-kontakte geklassifiseer is, was die risiko vir infeksie meer uitgesproke in vergelyking met kinders wat nie TB-kontakte was nie (p=0,027; RR=3,25; 95% CI=1,45-7,27; n=43).

Resultate van die Mantoux-toets was beduidend verskillend onder vroulike inwoners wat een of meer miskrame gehad het, teenoor vrouens wat geen miskrame gehad nie. Mantoux-positiewe vrouens wat vir ouderdom en ras gemeet is en vergelyk is met twee vrouens, negatief getoets, vir die Mantoux-toets, het 'n beduidende verskil aangetoon in vergelyking met Mantoux-negatiewes en die wat geen miskrame gehad het nie (p=0,023; RR=3,23; 95% CI=1,28-4,42; n=66).

Inwoners oderwerp aan oorbewoonde behuisings-eenhede, asook blootgestel aan totale ventilasie van minder 5%, het beduidend meer geïnfekteerde inwoners gehad as as dié wat nie onderwerp was aan hierdie gekomineerde faktor nie (p=0,016; RR=2,38; 95% CI=1,12-7,06; n=57). Hierdie studie het ook aangetoon dat gereelde deelname aan sport/oefening 'n beskermende effek (RR<1) teen TB infeksie (Mantoux-positief) gehad het, teenoor inwoners wat 'nie blootgestel' was aan gereeld sport/oefening nie (p=0,025; RR=0,53; 95% CI=0,29-0,97; n=164).

Nege (2,4%) inwoners van die Kamp is voorheen vir TB getoets. Van die 275 miniatuur X-straal-ondersoeke wat uitgevoer is, was 37 beskou as 'TB verdagtes' en 32 is gevra om weer X-straal-ondersoeke by die kliniek te ondergaan, sowel as sputum mikroskopiese en-kultuurondersoeke, wat 6 bevestigde gevalle van aktiewe TB opgelewer het (ingeslote 1 TB terugval-geval). Gebaseer op hierdie resultate was die prevalensie vir PTB vir 1988 (vir inwoners van Macassarkamp) 1 355/100 000).

Die getal geïnfekteerde individue opgespoor deur die TB ELISA-toetse was 20 (9,6%) teenoor die 67 (32,1%) opgespoor deur positiewe Mantoux-toets. 'n Swak ooreenkoms (Kappa statistiek = 0,070) is aangetoon met vergelyking tussen TB ELISA-en Mantoux-toetse m.b.t. die infeksiekoers. TB ELISA-toetse het 94,5% spesifisiteit aangetoon en 'n 100% sensitiwiteit vir nuutbevestigde PTB-gevalle. Daar was geen korrelasie tussen TB ELSA en Mantoux-toetse nie.

Wat sifting vir TB betref, het die TB ELISA die hoogste positiewe voorspelbare waarde (45,5%) met 'n 100% sensitiwiteit en 94,5% spesifisiteit, aangetoon; teenoor die X-straaltoetse (positiewe voorspelbare waarde van 25,0% met 'n 100% sensitiwiteit by 93,0% spesifisiteit), en Mantoux-toets (positiewe voorspelbare waarde van 4,5% met 60% sensitiwiteit by 68,6% spesifisiteit).

Ten slote kan ondubbelsinnig verklaar word dat daar 'n TB

-epidemie in Wes-Kaap woed, en dat die insidensie in Macassar/Firgrove van die hoogste in die Wes-Kaapprovinsie is. Faktore soos huishoudelike kontak, oorbewoning en swak ventilasie, wanvoeding, 'n lewensbestaan onder die primêre voedselonderhoudsvlak, rook ongeskoolde werk en geslag (vroulik), was sommige van die faktore geassosieer met die inwoners se vatbaarheid vir TB-infeksie.

Met die verskyning van die HIV-pandemie sal infeksie en ontwikkeling van die siekte al hoe vinniger plaasvind. Waar die dormante *Mycobacterium tuberculosis* en/of MOTTS in meer as die helfte van die Suid-Afrikaanse bevolking verskuil is, sal die getalle van TB-gevalle toeneem, soos waargeneem vanaf 1986 toe die effek van VIGS vir die eerste in Suid-Afrika opgemerk is. Die probleem kan gekompliseer word waar pasiënte ongereelde of onvoldoende TB-behandeling ontvang, met gevolglike toename van MDR-TB pasiënte. Druk by hospitale en klinieke kan veroorsaak dat pasiënte met weerstandige *Mycobacterium tuberculosis* nie behandel word onder die nodige toesig nie, sodat die infeksie daardeur verder sal versprei word en die weg baan vir a dodelike TB-epidemie. As 'n gesamentlike poging nie dadelik aangewend word om TB op 'n nasionale basis te bestry nie, sal die nuwe Suid-Afrika onvoorbereid wees om hierdie probleme die hok te slaan. Dit sal 'n gevolglike ramp in die nuwe Suid-Afrika veroorsaak en 'n nasionale noodtoestand van ongeëwenaarde aard.

ACKNOWLEDGEMENT

"True gratitude within the heart
evades the simplest word;
It fills the soul with glow and warmth
and struggles to be heard.

It seeks expression in a smile,
a 'thank you' most sincere;
It strives to pierce formalities,
its purpose high and clear.

Yet it finds fulfillment
and one will know who tries,
In just the firmer clasp of hands,
and grateful shining eyes."

Mabel Jones Gabbott

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DEDICATION

THIS THESIS IS

DEDICATED

TO THE MEMORY

OF

SHAYKH YUSUF

THE FIRST RESIDENT

OF

MACASSAR

WHOSE TRICENTENARY WAS

COMMEMORATED ON 2 APRIL 1994

&

ALSO TO THE FIRST RESIDENT

TO HAVE DIED OF TUBERCULOSIS

IN

MACASSAR CAMP

JUST PRIOR TO THE COMMENCEMENT OF THIS STUDY

THE
ULTRA-MICROSCOPIC PERSPECTIVE

"All that I have seen teaches me
to trust the Creator
for all I have not seen."

Anonymous

Mycobacterium tuberculosis observed by means of an electron microscope. Magnification X 100,000

SOURCE:

Crispen, RG; Biological & ultrastructural characteristics of Mycobacterium tuberculosis cells & cell components. Northwestern University, Medical School, PhD thesis, 1967.

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Now go, write it before them in
a table, and note it in a book.

—— *Isaiah 30:8*

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"I saw to my astonishment that they had the appearance and all the other characters of the mysterious cultures."

Robert Koch

NOTE

This statement by Robert Koch, was made, in the lecture where he enunciated his famous postulates, and in the context of his discovery, stated that the avian and mammalian tubercule bacilli were different. Because the morphology of the two types of tubercule bacteria was the same, he earlier on assumed it was the same organism.

Source:

Koch R. (1890). "Bacteriology & its Results." Lecture delivered at First General Meeting of the Tenth International Medical Congress, Berlin. Trans. TW Hime. Bailliere, Tindall & Cox, London.

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LIST OF APPENDIXES

Sermons on brevity and chastity are about equally effective. Verbal promiscuity flows from poverty of language and obesity of thought, and from an unseemly haste to reach print — a premature ejaculation as it were.

Eli Chernin

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INTRODUCTION

"Read (Proclaim)!
In the name of thy Lord & Cherisher,
Who created – created Man,
Out of a mere clot of congealed blood.
Read (Proclaim)!
For thy Lord is Most Beneficent.
Who has taught the use of the pen,
Has taught Man, that which he knowth not."

Al Quran
Sura 96: 1–5

CHAPTER 1

INTRODUCTION

SUMMARY

This chapter commences by introducing the causative organism (*Mycobacterium tuberculosis*) of TB. It briefly outlines the history of Man's control of pestilence and disease (even some of the most formidable diseases). Man has even successfully eradicated some of these diseases such as small pox.

TB has been associated with Man since time immemorial and yet continues to flourish in many developing countries. With the advent of HIV/AIDS pandemic, the previous decline of TB incidence in some of the developed and developing countries has actually been reversed in the past few years. TB is a major cause of disease and death in South Africa and PTB accounts for 56% of all notifiable medical conditions and is major health problem especially amongst the Black and Coloured communities. The pending TB epidemic in the Western Cape was already noted in 1908 and by 1991 in the Western Cape the TB prevalence rate had reached an all time high of 1 134 per 100 000 population (in certain suburbs)
TB is preventable and curable. Why then, has the TB problem remained unsolved for so long in South Africa?

Perhaps the answer lies in a more direct focus on contributing factors such as malnutrition, stress and socioeconomic status that may have been associated with TB infection, the development of open infectious cases of TB, aspects of active case finding and case holding and a well motivated programme of TB treatment for the successful implementation of an effective and efficient TB Control Programme. Furthermore the key to the conquest of TB lies in the strategy whereby emphasis should be placed also on the dormant *Mycobacterium tuberculosis* in healthy individuals Approximately 10 million South Africans are infected with this pathogen. An estimated 15% of the pool of infected persons, could develop active TB during their lifetime.

The best single indicator for the evaluation of the trend of TB in developed and the developing countries is the annual rate of TB infection.

The way forward would be the Primary Health Care Model which is comprehensive and intersectoral. Finally this chapter concludes by motivating reasons for the study in Macassar.

CHAPTER 1

INTRODUCTION

1 THE CAUSAL AGENT OF TUBERCULOSIS (TB)

More than a century ago, Robert Koch isolated and identified *Mycobacterium tuberculosis* as the causal agent of tuberculosis (TB). TB is now a curable and often preventable disease as can be noted by the decline in the TB epidemics from many developed countries. Present available control measures should be able to sufficiently alter the course of the TB epidemic in South Africa to lead to a rapid decline in the incidence of TB. Why then has this problem, which can be solved, continued to mushroom into such a serious problem in South Africa? **[14]**

Infection with *Mycobacterium tuberculosis* may or may not lead to the development of active TB. After infection, a primary complex is formed within 4-12 weeks which may heal or may become calcified but still retain viable bacteria. These dormant bacilli may reactivate when the immune system is compromised by immunosuppressant conditions such as malnutrition, stress, or overcrowding which are mainly a result of the socioeconomic status of the individual.

Perhaps, in South Africa there has been an over-emphasis of medical science which has diverted attention from the real underlying causes of ill health such as poverty. This has been further compounded by numerous other factors, which have undermined attempts to control TB.[20]

2 MANKIND VERSUS INFECTIOUS DISEASE

Pestilence of many sorts has brought death and misery to mankind throughout history. Over the last 100 years however, most formidable infectious diseases have been brought under control. An example of not only control but total eradication of an infectious disease is smallpox. However, TB formerly known as "The Great White Plague", defies modern medical science and continues to flourish especially in developing countries.[1]

One can take the liberty of renaming TB: "The Last of the Great Plagues" and one which is likely to increase in seriousness within the next few years especially with the advent of the AIDS pandemic and the HIV interaction with TB.

3 AN OVERVIEW OF TB

TB has been associated with human settlements since time immemorial. As yet there is no evidence

of where and when Man first became afflicted with TB. However, there is evidence of TB in Man, during the Neolithic period.[1,2,3] In the Western World this dreaded disease (TB) had become an insignificant problem until the current HIV/AIDS epidemic. However, TB remains a major threat in all developing countries and is one of the world's most prevalent infectious diseases. Of the 3 million or more people dying every year of TB, more than 80% are from developing countries. With the advent of HIV/AIDS pandemic and its interactions with TB, the previous decline of TB incidence in some of the developed and developing countries (such as USA and Tanzania) has actually been reversed in the past few years.

A report presented on World Health Day, 1964, acknowledged that TB was a major problem in the developing countries and it was estimated then that this disease was claiming more than 10 million TB victims and resulting in excess of 3 million deaths annually.[4] Further 1 out of every 150-200 persons were suffering from infectious TB in a population of 2 000 million from the developing countries (predominantly Latin-America, Africa and Asia). This was not only a serious risk to all contacts but increased the

infected pool with long term consequences now being observed on an increasing scale compounded by factors such as the lack of basic health facilities mentioned in the Declaration of Alma-Ata.

The TB problem in South Africa was compounded by the large scale influx of tuberculotic immigrants into the country thus increasing the TB infected pool. And according to Dr Scholtz in his book, [8] which promoted South Africa as a climatic cure for TB: "... this might in the near future seriously affect the population of South Africa ...." [5,6,7]

4 **MORTALITY & MORBIDITY TB IN SOUTH AFRICA**

TB is a major cause of disease and death in South Africa. In 1988, there were 58 502 (which includes all population groups) notified cases of pulmonary tuberculosis (PTB) in South Africa. [9] PTB accounts for 56% of all notifiable medical conditions and was a major health problem amongst the Black and Coloured communities. [10] In the same year there were 6 956 (all races) registered TB deaths. In 1987 a study showed that there was a rising incidence of TB (amongst the Coloureds and Blacks) in the Western Cape. [11,12,13]

These morbidity and mortality figures for TB are reported despite the promise of the Alma-Ata Declaration and the fact that TB is potentially preventable and curable provided that the disease is diagnosed in the early stages. Why then, has the TB problem remained unsolved for so long in South Africa?[14]

The present TB morbidity and the mortality figures in South Africa (Western Cape) are regarded as one of the highest in the world. As far back as 1906, TB was causing great concern and alarm. At a conference convened in 1906 in Cape Town, by Medical Officers of Health,[15] the gravity of the TB problem was noted and the danger threatening the Black and Coloured population.

Nearly ninety years later, we are still a long way from solving the TB problem in this country. A far cry, from a catchy slogan I once spotted as graffiti in a township:

"FREEDOM = NO TB DEATHS
WILL THE AFRICAN NATIONAL CONGRESS (ANC)
DELIVER THE GOODS?"

This slogan is vaguely reminiscent of the phrase that was popularized by Dr Basil Dormer, in the middle of this century: "A meal a day will keep TB

away." Somehow, as we are about to enter the 21st century, this phrase has undergone a metamorphosis to suit the present 'climate' in the country. Only time will tell what the next stage of the 'metamorphosis' of this phrase will be in years to come.

TB, for many of the underprivileged and poor who form the majority of the South African population is considered a dreaded deadly disease reminiscent of the attitude to the TB epidemic during the Industrial Revolution. The stigma attached to this disease within the community and attitudes of employers have further compounded this problem. It is evident that these attitudes can be attributed to ignorance, poor level of education and the socioeconomic and political situation in the country.

Robert Koch, the discoverer of the causal agent (*Mycobacterium tuberculosis*) of TB aptly summed up for posterity the TB morbidity and mortality:[16] "If the number of victims which a disease claims is the measure of its significance, then all diseases, particularly the most dreaded infectious diseases, such as bubonic plague, Asiatic cholera, must rank far behind TB."

Today, with the advent of the AIDS pandemic, Koch's statement, made more than 100 years ago remains true. Bubonic Plague (Black Death) of the 14th century; the Great Plague of London (1664-65); and the plague pandemic (1896-1917) will look like child's play due to the TB mortalities as a result of AIDS. Those who are infected with the tubercle bacillus which in most individuals remains dormant, (Appendix A1), will have a high risk of endogenous reactivation into active TB in the event of HIV infection which is the prelude to AIDS. Thus HIV will take up the role of the 'agent provocateur' in the already beleaguered communities afflicted with the TB epidemic.

This will have a far reaching implications and impact on the TB Control Programme which is presently struggling to cope with the TB epidemic in most developing countries. The current data has already indicated a vastly increased mortality rate in the HIV/TB patients.[17]

CAUSES OF TB

Despite tremendous growth in knowledge and technological advances in the areas of diagnosis, treatment and prevention,[18] we have failed to control TB in South Africa. Perhaps the answer to

our failure in the TB Control Programme in this country, lies in the fact that we have desperately tried to combat the effect of this disease rather than "attacking the root cause" of TB, which has been rampant in this country for more than a century.

To attribute the worldwide TB epidemics to *Mycobacterium tuberculosis* alone would be a gross underestimation of a more complex and an intricate problem, that all developing countries and to some extent developed countries have to contend with. Following infection, TB has a varying incubation period and the majority of those infected may never develop this disease at all. This suggests that there are other variables such as malnutrition, stress and socioeconomic status that contribute to the development of open infectious cases of TB. In South Africa, Apartheid[20] was an additional factor in the unabated attack of this microscopic organism named *Mycobacterium tuberculosis*, on humanity.

The problem of TB in South Africa, was put in its proper holistic perspective by the former Medical Officer of Health (MOH) of Cape Town in 1988, Dr Reg Coogan, who was interviewed on South African Television, (SABC) on the then NETWORK programme,

on the topic of TB:[21] "Tuberculosis is a socioeconomic related disease. It's 'caused' by poor living conditions, by overcrowding, by damp housing, by malnutrition, by strain ..... Until one eliminates these socioeconomic causes, there is no medical cure of TB. All doctors can do is treat the actual cases of TB and try to prevent the spread. One will not eliminate the disease without eliminating the 'cause' in the first instance."

However, for those persons who have developed TB through infection reactivation and/or relapse, the aspects of active case finding and case holding and a well motivated programme of treatment are very important to the successful implementation of an effective and efficient TB Control Programme.

An excellent example of the treatment of TB as a preventative measure as well can be noted from Dormer's innovative idea in 1959. Dr Basil Dormer in 1959, encouraged tuberculous mothers to breastfeed their babies thus protecting their infants with INH from the day of their birth.[19] This innovative manner of treatment of the mother and baby resulted in a preventative measure as well and saving lives of countless infants.

1.3.2.5 Unitarian Theory of TB

The Dualism Theory was refuted by Laennec who proposed the Unitarian point of view more than 60 years before the discovery of the tubercle bacillus in his book:[9] "Des Tuberculosis du Poumon ou de la Phthisis Pulmonaire." He was also responsible for doing away with the 13 categories of Phthisis. Finally it was Schonlein (1839), a professor of Medicine in Zurich who proposed the name 'tuberculosis' substituting it for all forms of Phthisis.

1.3.2.6 The Arab Contribution to Discovery of TB Bacillus

Unfortunately, history has dealt a deplorable blow to many Arab and Muslim scientists. Writers and scientists of developed countries have often directly and/or indirectly ignored the scientific contributions of Arabs and Muslims to the world. In fact, Science (especially Medical Science), was one of the most important contributions of the Arab civilization to the modern world. The tremendous and significant advances in various fields of science made by the Arab civilization in Spain, came to full bloom only after that civilization had disappeared in that country.[64] The Arabs not only translated Greek literature

(especially works of Science), but added their own substantial and significant research knowledge, thus placing Medicine for the first time on a scientific basis. This was due mainly to the fact that Greek Science was mainly based on hypothesis whereas the Arab scientists based their scientific investigation on experimentation and observation.[12]

A Western writer aptly summarizes the significant contributions of Arabs in Spain (1061 AD), to the world:[13] "Europe was darkened at sunset, Cordova shone with public lamps; Europe was dirty, Cordova built a thousand baths; Europe was covered with vermin, Cordova changed it's undergarments daily; Europe lay in mud, Cordova's streets were paved; Europe's palaces had smoked hole in the ceiling, Cordova's arabesques were exquisite; Europe's nobility could not sign its name, Cordova's children went to school; Europe's monks could not read the baptismal service; Cordova's teachers created a library of Alexandrian dimensions."

Thus there can be no doubt of the immense and significant contributions in areas of Health Care and Preventive Medicine, by Abu Bakr Muhammad Ibn Zakariyya Razi (Rhazes 865-925 AD) and Ibn Sina (Avicenna 980-1037 AD). Their works, were in

later centuries translated into Latin.

Long before Fracastorius in 1546 expressed his view on phthisis as one of the many 'contagious diseases' due to 'seeds of contagion', Razi had already experimented with this idea.[14] When he was appointed as the Chief Physician in Baghdad and was asked to select a site for a hospital. His decision for the site of the hospital was based on the experiment whereby he hung up raw meat in various areas of the city and thus chose the site where the meat showed the least signs of putrefaction.[15] He also made contributions in the field of hygiene and public health and children's disease. His postulates on measles and smallpox led to the introduction of vaccination in 1679 against smallpox by the Turks long before Jenner's vaccine against smallpox.

Abu-Ali al-Hussain ibn-Abd-Allah ibn-Sina[15,16] (Avicenna 980-1037 AD from Baghdad) laid the foundations of Greek-Arabic Medicine, in his encyclopaedic work ('Kitab al-Qanun') known as the Canon of Medicine (Appendix C2) or the 'Bible of Medicine' in which he states the contagious nature of phthisis and the spread of disease by water (droplet infection). His Canon of Medicine, which was translated into Latin in the 15th

century by Gerad of Cremona, soon worked its way into a position of prominence in the Medical Literature of the time, displacing works of Galen and Razi and remained a prescribed textbook in the universities of Europe till the late 17th century. [16]

1.3.2.7 Koch's Postulates - 'Golden Age of Bacteriology'

Prior to Koch's discovery of the cause of TB, he established the basis of advances in modern bacteriology and the discovery of many etiological agents of the most important disease in humans, and animals.

These postulates or principles (with regard to infectious diseases) which are still valid to this day are:

(i) A specific organism must be observed in all cases of infectious disease.

(ii) This organism must be isolated and grown in a pure culture.

(iii) Organisms from pure cultures must reproduce the disease in experimental animals.

(iv) It must be possible to recover the organism from animals so infected.

More than 100 years ago, during the period which was termed: "The Golden Age of Bacteriology"

(because great strides were being made in the field of bacteriology), and at the time when TB, was the cause of great morbidity and mortality, Robert Koch announced that he had discovered the causative organism of TB which was named *Mycobacterium tuberculosis* on 24 March 1882. This was the turning point in dealing with this dreaded disease.

Although Robert Koch, discovered the causal agent (*Mycobacterium tuberculosis*) of TB in 1882, this discovery was anticipated during 1865-1868, by a French military surgeon named Villemin who performed experiments by inoculating tuberculous material into animals. However, it must be stressed that the foundation for Koch's historic discovery was set in motion long before Villemin's experiments.

The implications of this finding were that TB was an infectious disease and thus could be cured if the bacillus was destroyed. Robert Koch was not only honoured with the Nobel Prize in 1905 for his contribution in the field of TB research but later was honoured with the title: "The Father of Bacteriology" for his tremendous and significant contributions in the field of a new branch of Science that is today known as Bacteriology.

However, it must be borne in mind that the decline in respiratory TB death rates in England and Wales, had already started long before the discovery of *Mycobacterium tuberculosis*. The reasons for this decline have not yet been fully understood.[69]

1.3.2.8 The Age of Chemotherapy

As the measures in medical science, for the treatment of infectious diseases became more gradually established, the role of the TB researcher became more challenging. From uncertain beginnings and prolonged trial and error, basic principles were evolved in the treatment of TB.

With the discovery of sulphonamides by Domagk in 1935, the Age of Chemotherapy arrived which was initiated by the experimentation of Paul Ehrlich in the first decade of this century and which resulted in the formulation of principles of specific chemical relationships between the pathogens and drugs.[10]

The early post-world War II era brought 3 highly significant developments in the anti-TB campaign which became part of the TB Control Programme of WHO.[11] The anti-TB campaign in the early post World War II period was based on:

(i) the introduction of effective Chemotherapy

(ii) BCG vaccination

(iii) the beginning of a National Health Service in many developed countries.

1.4 HISTORY OF WORLD TB EPIDEMICS

In order to understand the spread of TB and the current disease pattern in South Africa, one has to review the aspects of the disease when it was at its peak in England, prior to the arrival of tuberculotic immigrants on the shores of the Cape.

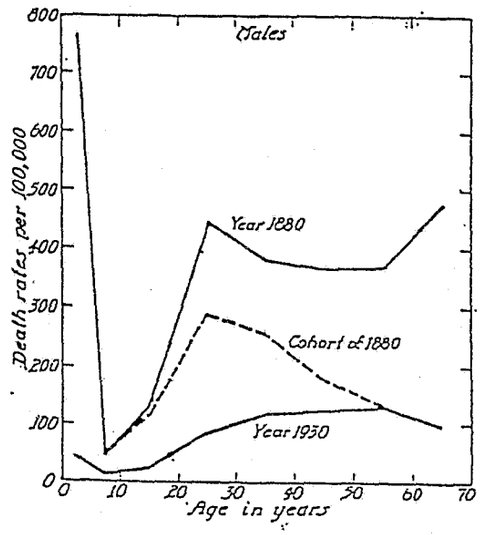
It has been stated that TB is a cyclic epidemic and that the span of the epidemiological curve could last for centuries.[38] The morbidity and mortality of this cyclic curve has a short ascent but a longer descent with a plateau in between. In England, during the 16th century TB had already reached epidemic proportions and reached its peak only in the mid 18th century with the advent of the Industrial Revolution.[39] Various factors of urbanization and industrialization such as the creation of industrial slums, overcrowding, poor sanitation and hygiene, ... , probably exacerbated the TB epidemic.[40] The crude mortality rates were as high 500/100 000 and accounted for 30% of all deaths in British labourers.[41]

By 1844 approximately half of the population of England was infected from TB.[42] The mortality rate in England and Wales had fallen to about 200/100 000 per year. This mortality rate fell significantly to 50/100 000 by the 1940's, prior to the application of chemotherapy. However during the era of the commencement of chemotherapy the mortality rate dropped significantly again to 5/100 000.[43]

In addition to problems arising from the factors of the Industrial Revolution that favoured the TB epidemic, it had become a fashion to be ill with TB in the early 19th century. Alexander Dumas wrote: "In 1823 & 1824, it was a fashion to suffer from the lungs; everybody was consumptive, poets especially; it was a good form to spit blood after each emotion that was at all sensational and to die before reaching age 30".[44] At the time booming sales of lemons were attributed to the fact that poets praised a pale complexion. This resulted in the women drinking lemon juice to kill their appetite and create a wan look.[45] Figure 1 graphically depicts the death rates of all forms of TB among males in 1880 and 1930 in Massachusetts (USA).

Figure 1

Massachusetts death rates from TB (all forms) among males, by age in years 1880 & 1930, and the cohort of 1880



Source:

Comstock, G. Reviews & Commentary: Tuberculosis-A Bridge to Chronic Disease Epidemiology. American Journal of Epidemiology, 1986; Vol 124 No 1, p8.

The spread of this TB epidemic resulted in successive peaks in Western Europe and East Coast of North America in the early part of the 19th century. This TB epidemic, eventually reached Eastern Europe and South America by the late 19th century.

At the beginning of the 20th century, a survey carried out confirmed that almost all the adult population in the European and American cities were tuberculin positive.[42] However, as can be noted from figure 2, the TB death rates began to decline even before the improved living conditions and housing or the advent of chemotherapy or BCG.[1,27,42]

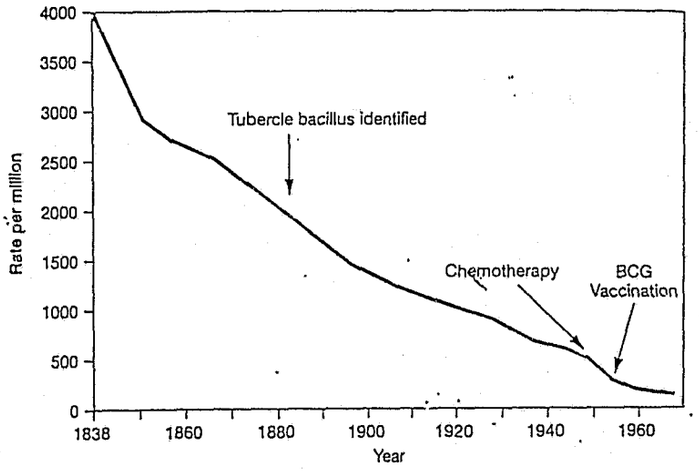
Thus, figure 2 depicts the rapid decline in the in death rates of PTB in the late 19th century in England and Wales like many developed countries at the time. To this day, the reason for the decline in TB mortality in the developed countries, is still a mystery.[1]

TB death rates declined in developed countries before the identification, discovery or isolation of the tubercle bacillus. This very TB epidemic is still raging unabated with high rates still reported in Asia and Africa.

Although the mortality rates have declined, they are still significantly higher when compared to First World countries.

Figure 2

The decline in respiratory tuberculosis death rates in England & Wales since 1838. Standardized to the 1901 population



Source:

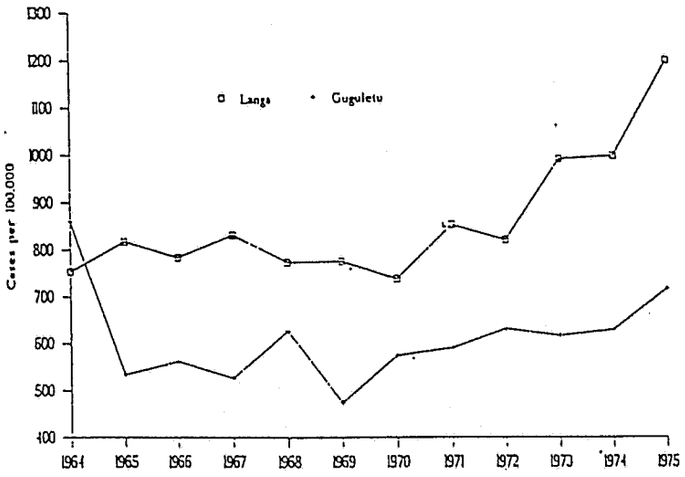
McKeown, T. (1979). The Role of Medicine: Dream, Mirage or Nemesis? Oxford: Basil Blackwell. (Effects 2 world wars not indicated on the graph)

South Africa, like some other countries in the world, has the facets of both developing and developed countries residing in parallel at the same time. The health profile of the various population groups confirms this point especially with regard to TB notification rates.

Figure 3 depicts TB notification rates of Africans in Guguletu and Langa. A similar TB notification rate can be seen for all 'races' in figure 4.

Figure 3

African TB Notifications in Langa & Guguletu (1964 - 1975)



Source:

Packard, R, (1991). White Plaque, Black Labor: Tuberculosis & the Political Economy of Health & Disease in South Africa. p 282

1.4.1 The Progress of the TB Epidemic in South Africa

During the 19th century it was the fashion to seek a 'climatic cure' for TB in South Africa besides country's like USA and Switzerland. Thus, South Africa had become a popular resort for the tuberculotic immigrants in the late 19th century. These immigrants hailed mainly from England and Scotland.**[46a,46b,46c]** The popularity of South Africa as 'tuberculosis climatic cure', occurred

just when the TB epidemic was showing signs of a decline in the developed countries and just before the discovery of the tuberculosis bacillus by Robert Koch in 1882.

At this time of rapid socioeconomic advancement in the West and colonization of South Africa the incidence of TB declined in England, but the 'White Plague', now increased in incidence in the developing countries. This can be noted from the simultaneous decline of mortality in England and the steady increase in mortality rate amongst the Coloureds and Asiatics in South Africa.

Table 1

TB Death Rates per 1 000 Persons

	SOUTH AFRICA			ENGLAND AND WALES
Years	Whites	Coloureds & Asiatics		
1903-1904	1,10	2,91	1,41	1,74 (1903)
1904-1905	1,17	3,08	1,18	1,77 1904)
1905-1906	1,04	5,27	1,38	

Source:

Collins, TFB. History of southern Africa's first TB epidemic. S Afr Med J. 1982; 62 (21), p783.

A further eloquent indication that South Africa was turned into a health resort for tuberculotic

immigrants appeared in the Cape Times in the 1890's:[47] "South Africa, the land of the Highveldt and the Karoo! The land of soaking sunlight and crisp dryness and cool night wind! The land of elevating plains which join the virtues of the desert for which the sick men flee to Egypt with the virtues of the mountain for which they seek Switzerland! The Cape which cures consumption, the Cape which is of Good Hope to all the weak chests, the Cape which offers life and health and a career, to the Englishman suddenly confronted by the modern absolutionist, the doctor with the cold sentence of death or exile."

Many companies capitalized on the fact that South Africa was a 'Tuberculosis Health Resort'. Even the Union Steamship Company benefited from the increased number of passengers to South Africa between 1886 to 1898, with their publication of a book titled:[1] "South Africa as Health Resort." Refer to Appendix C3.

These sanatoria resembled hotels rather than hospitals. The most prestigious and expensive sanatorium was just outside Kimberley built by De Beers Company. There can be no doubt that the large influx of tuberculotic immigrants and the creation of these 'fad' sanatoria resulted not

only in the high incidence of TB amongst the natives but also created an infected pool of which we see the results today in active cases of TB amongst Blacks and Coloureds especially.

The 1914 TB Commission states: "... evidence that the aggregates of consumptives in those Karoo villages formed the foci from which the disease had spread.[46d] Dr Scholtz too noted this phenomena in his book:[47] "... it might in the future seriously affect the population of South Africa - namely the almost daily wholesale influx of phtisical and scrofulous cases, especially of the poorer classes, into this country from Europe."

As the number of tuberculotic immigrants declined in South Africa the mortality rates amongst the Europeans too declined.[48] By 1911 the TB mortality rate as a whole was lower than those in England and Wales - with Cape Town being the exception - having the highest prevalence rates even in Whites.[4e,49] The overall decline in prevalence and mortality rates amongst Whites in South Africa, was according to the 1914 TB Commission, due to the improved standard of living.[4e]

1.4.2 South African TB Epidemic - Contributing Factors

There were many other contributing factors in the spread of the TB epidemic in South Africa besides the 'conversion' of certain areas of South Africa, into a Health Resorts for the 'climatic cure' of TB. The second factor, (in the late 19th & 20th centuries), was and still is; overcrowding and squalor as a result of industrialization and urbanization, mostly among the most exploited susceptible and oppressed groups.[46f,50]

The third contributing factor was the introduction of the migrant labour system, on discovery of gold. The mines played a significant role in the spread of the TB.[51a] In fact a glaring admission of this fact is made in the 1914 TB Commission:[46h] "... the mining industry is one of the most important of all factors in the cause and diffusion of the disease among the native population."

Another study showed that mineworkers had a higher prevalence of TB than their counterparts in their area of origin.[46i] Conditions in the mineshafts were one of the main causes that facilitated the spread of TB between infected and non-infected workers. Table 2 affirms this point.

Table 2

TB Infection Rates & Exposure to Minework (1910 - 1912)

Sample	No. Tested	No. Positive	% Positive
Basuto (mine experience)	222	81	36,0
Basuto (no mine experience)	181	28	15,5
Mozambique (mine experience)	63	11	17,5
Mozambique (no mine experience)	415	7	1,7
Nyassa (mine experience)	52	10	19,0
Nyassa (no mine experience)	129	6	4,0

Source:

The 1914 Tuberculosis Commission, Report, p113-114

The Anglo-Boer War, at the turn of the century led to the fourth contributing factor in the spread of the TB epidemic. The influx of a large number of people, including TB sufferers into the cities due to a downward trend in the economy was further burden to local authorities.[46j]

The fifth factor was, most likely *Mycobacterium bovis* in cattle. The epidemiology of bovine TB in humans due to lack of available data in this country, is controversial indeed. According to Smith,[45] pasteurization of milk in Britain was deliberately blocked for many decades.

In the Cape Colony in 1905, William Robertson, a government veterinary officer confirmed from tests that 60% of the cattle in the Cape Division were affected.[51b] A study in 1911, after a decade of research, concluded that *Mycobacterium bovis* was a serious source of human infection especially among children.[54] The source was believed to be from imported cattle, where 12% of the herds in the Cape were believed to be infected with the tubercle bacilli.[55,46k]

By 1951, Britain had the highest prevalence of bovine TB of any country. This was probably attributed to the fact that doctors in Britain (1929) counselled mothers against boiling milk for their babies and an eminent specialist of that day declared that the pasteurization of milk would sap "natural fertility and strength".[45]

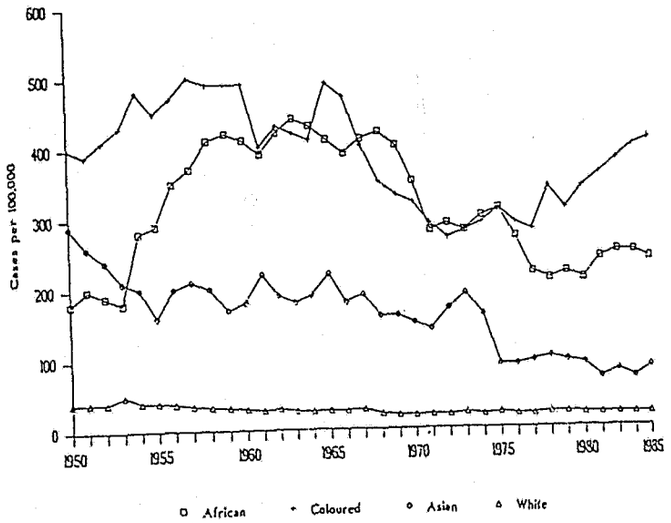
The Cape Act No.16 (Animal Disease Act) of 1893 was amended but the successful implementation was blocked largely due to the vested economic interest of the dairyman and farmer and although isolated efforts were made by the local authorities in the eradication of infected cows, it can be concluded that bovine TB went largely unchecked in this country until after the World War II.[55] By 1947, it was estimated that the

prevalence of bovine TB was between 30%-40% in the dairy cattle of South Africa. Yet, today the isolation of *Mycobacterium bovis* is relatively rare in South Africa.

The sixth contributing factor was undoubtedly Apartheid, which was inextricably linked to state policy based on racial lines which, further fuelled the TB epidemic.[52] Refer to figure 4.

Figure 4

TB Notification Rates, RSA, 1950-1985



Source:

Kustener, HGV, (1979), Tuberculosis Notification: An Update, SA Journal of Science, 1986, 82:386

This state policy of Apartheid can be separated into three historical periods namely:[53]

* pre-1948
* era of segregation (legislation of Apartheid)
* the era of reform

The following main objectives played a role in the South African urbanization policy and significantly impacted on the health of the 'victims' (especially those infected with *Mycobacterium tuberculosis*), in the country: [53]

(i) division of land on racial basis
(ii) need for labour to be regulated & allocated
(iii) need to maintain white political power

1.5 THEORY OF RACIAL SUSCEPTIBILITY TO TB

This is based on the theory that a population that has had very little or no exposure to the disease and the industrial way of life has had no time to acquire resistance. This population is termed the 'Virgin' Population and the belief is that they are more susceptible to the disease. Then there are others that believe that the history of TB in South Africa is due to the same set of political and economic factors that had shaped the history of TB in the West.

The proponents of the 'Non-Virgin' Population theory also believe that the uniqueness of the

South African TB experience is focussed in the manner in which the alignment of the changing political and economic policy evolved into legislative law. These two schools of thought are clearly at odds with each other.

What is now significant is not where the disease originated or who is right, but where is the disease going to and what is being done to halt its spread. Proponents of both theories have one common goal - control and elimination of TB. The following two theories are presented in the spirit of pure academic interest and in patronage with Voltaire's philosophy of: "I disapprove of what you say but I shall defend to death your right to say it."

1.5.1 In "Support of Virgin Soil" Theory

The proponents of this theory feel that the writings of earlier researchers in South Africa provide evidence in support of this theory. The literature available to this effect is: 1914 TB Commission; Dr Livingstone's book - "Missionary Travels & Research in South Africa (1827); Dr Theal's book (1910) - "The Yellow & Dark Skinned People of South Africa" and McVicar's thesis to , to mention but a few.[57]

The current proponents of this theory state the experience of TB in Africans is different from that of Whites and the critics of Apartheid who support this theory, state that this difference is due to Apartheid. This theory should be viewed in the light that limited evidence is available on the health status of Africans prior to the 19th century.

1.5.2 In Support of "Non-Virgin Soil" Theory

The proponents of this theory claim that the rise and fall of TB in England, is reflected in the History of TB and Industrialization, where the incidence of TB is directly linked to changing political and economic interest in England.[51c]

This theory is by no means new. In fact Dubos very aptly expressed the major determinant of the TB epidemic:[58] "TB was perhaps the first penalty that the capitalistic society had to pay for the ruthless exploitation of labour."

The proponents further expand this theory by stating that the decline of the TB mortality rates in Europe, in the late 19th and early 20th century, were not due to modernization, from scientific know-how or any reforms but rather the effects of 'natural selection.'

Thus the proponents have postulated that the decline in TB in England could have been due to the overall resistance produced by the early elimination of genetically susceptible families and the survival of the more resistant families.[51c]

Although the phenomena is a commonly known fact in the natural history of many infectious diseases, TB is a social disease and should be viewed in a socioeconomic context with all its ramifications. The definition of a disease has been aptly summarized by Rosenberg:[59] "A disease is no absolute entity but a complex intellectual construct, an amalgam of biological state and social definitions."

This 'natural selection' theory had been suggested by Maynard in 1912 on the Rand where he noted that the mortality rate amongst experienced mineworkers was lower than the new recruits. He suggested the work of Karl Pearson, which stated that "the resistance of the body was related to the presence or absence of the 'tubercular diathesis', in support of his findings.[60]

In South Africa this theory was further advanced by the impact of the changes in the political and

economic determinants based on race that will have far reaching implications in the future, in the control of tuberculosis in this country. These determinants that have further fueled the TB epidemic were: [51d]

(i) impoverished rural population exposed to sick repatriated workers, thus undermining their ability to resist TB.

(ii) migrant labour system could have delayed the development of resistance to TB.

(iii) the lack of demands by the African labour to push for Labour and Health reforms

Thus it is believed, prior to the 19 century, TB was rare disease in South Africa, and therefore the African population contained a higher percentage of individuals with hereditary susceptibility to TB. In addition to this Africans were exposed to the determinants of Apartheid which further fuelled the TB epidemic.

According to Packard, the reason why the 'Virgin Population' theory was widely accepted and popularized was: [51e] "On the one hand, it has provided defenders of the status quo in South Africa with a means of deflecting attention from the appalling conditions under which Africans lived and worked. On the other hand, it has

offered those who have attempted to challenge the status quo, an equally convenient instrument for highlighting those conditions. In both cases, the 'Virgin Population' theory appears to have been accepted more for its instrumentality than for its basis in historical fact."

2 THE PATHOGENESIS OF TB

2.1 *Mycobacterium tuberculosis*

2.1.1 Bacterial Morphology

These organisms are described in the Bergey's Manual of Determinative Bacteriology.[17] They are rod shaped and size ranges from 0,2 to 0,6 um in breadth and 1 to 5 um in length. They are straight or slightly curved rods occurring singly and occasionally in strands. They stain uniformly or irregularly, often showing banded or beaded forms. Strongly acid-fast and acid-alcohol-fast (AFB) by Ziehl-Neelsen Staining Method (ZN). *Mycobacterium tuberculosis* are obligate aerobes.

2.1.2 Colony Characteristics

Selective Lowenstein-Jensen media is used for the isolation of *Mycobacterium tuberculosis*. After incubation the cultures can be confirmed and speciated. The typical colony of human type strain

is dry, heaped-up, cauliflower-like colony. This type of colony is termed rough, 'eugonic' or 'R' (rough) type. The colour of these colonies is pearly, cream or off-white with luxuriant colony growth. The colonies of *Mycobacterium bovis* on the other hand are flat, smooth and whitish and grow sparsely. These colonies are termed 'dysgonic' or 'S' (smooth) type. However, colony characteristics alone are no sure guide to the identification of *Mycobacteria* species.

2.1.3 Taxonomy, Classification and Types

According to Bergey's Manual of Determinative Bacteriology, *Mycobacteria* belong to the order Actinomycetales which is comprised of the following families:[7]

(i) Mycobacteriaceae,
(ii) Actinomycetacea
(iii) Streptomycetacae.

Mycobacteriacea are divided into 2 groups:

2.1.3.1 Saprophytes and Potential Parasites

Mycobacterium marinum, Mycobacterium fortuitum and Mycobacterium smegmatis are found in the soil and in the plants and are normally recovered from cold blooded animals.

2.1.3.2 Mycobacterial Parasites of Warm Blooded Animals

Examples of these types of species are, which are known to be pathogenic to humans *Mycobacterium bovis*, *Mycobacterium ulcerans* and *Mycobacterium tuberculosis* .

2.1.3.3 The 'Atypical' *Mycobacteria*

These are organisms causing pulmonary disease often indistinguishable from TB. It is these organisms that are termed 'atypical' mycobacteria. Recently these type of mycobacteria have also been termed as 'nontuberculous' or 'environmental' mycobacteria.[65] A classification for these types of mycobacteria was put forward by Runyon in 1959.[18] Increased pathogenicity of atypical mycobacteria has been noted with the advent of the HIV epidemic. The following are the four groups of the Runyon classification for the 'Atypical' Mycobacteria:

- Photochromogen
- Scotochromogen
- Non-chromogen
- Fast-growing mycobacteria

2.2 VIRULENCE AND PATHOGENICITY

Virulence has been defined as the ability of the

bacilli to multiply locally in the tissues of the susceptible host and to spread to other parts of the body, thus producing progressive lesions at the site of entry and elsewhere.[19] In brief, virulence can be summarized as the capacity of the bacilli to survive and multiply in an intracellular environment.[20]

Pathogenicity on the other hand is the capacity of the bacilli to produce anatomical changes within the host. For example the human type tubercle is highly virulent to the guinea pig, causing the multiplication of bacilli within the tissues resulting in progressive and fatal disease.[7] In the case of the rabbit, the same human type tubercle is capable only of producing a lesion at the site of entry and does not cause progressive or fatal disease.

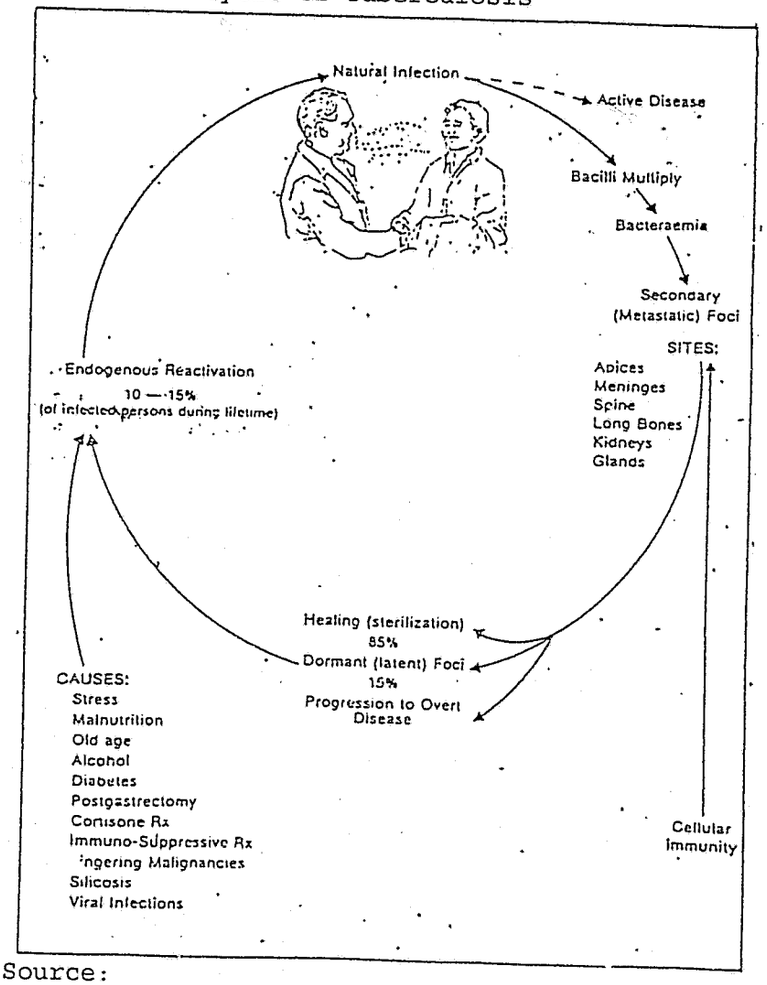
Thus for the rabbit the human type tubercle bacilli is merely pathogenic. The use of the term virulence is not meaningful without mention of certain conditions. Thus it can be said that both virulence and pathogenicity cover the complete complex range of host-parasite relationship.

Figure 5 depicts the cycle of TB, by droplet infection and the progression of infection

into one of the following: healing/sterilization or dormant/latent or progression into overt disease (TB) stage.

Figure 5

The Natural Cycle of Tuberculosis



Source:

Glatthaar, E. Tuberculosis in South Africa. 'Where have we gone wrong?' and a look at the future.' S Afr Med J Special Edition Nov 17 1982 p36-41, Robert Koch Commemorative Conference on TB.

The term virulence of *Mycobacterium tuberculosis* is used in the context of:

(i) the host

(ii) mode of inoculation

(iii) nature & extent of disease observed

(iv) infecting dose of bacilli/viable count of the inoculum

The chemical basis of virulence which is associated with a cord-like serpentine growth in the tubercle bacilli, was suggested as far back 1947 by Middlebrook et al.[21] The *Mycobacteria* that grew in cords had a high virulence capacity which was attributed to the sticky substance (trehalose 6,6'dimycolate) produced by bacteria and which was responsible for the cord formation.[61] Extensive studies have confirmed that while not all cord forming tubercle bacilli are virulent, all virulent tubercle bacilli are cord forming.[22]

Evidence of the role of the cord factor in the production of the disease by virulent tubercle bacilli was presented in 1972 by Kato.[23] He demonstrated that methylated bovine serum albumin produced antibodies specific to the cord factor. After immunizing mice against the cord factor, they were challenged with virulent tubercle

bacilli and it was demonstrated that experimental mice had greater resistance to the TB infection than previously.[24,25] It has been postulated that because the tubercle bacillus is a relatively stable organism that any possible changes to its virulence could not have contributed to secular or regional variations of TB mortality rates.[9]

2.2.1 Pathogenesis

This involves basically three stages:[26]

(i) transmission of infection
(ii) primary infection
(iii) active disease

2.2.1.1 The Sources of TB Infection

(i) 'droplet infection' - TB patient coughs or sneezes droplets containing tubercle bacilli
(ii) food contaminated with tubercle bacilli.
(iii) milk from cattle infected with *Mycobacterium bovis*.

The the major sources of infection are sputum microscopy smear positive cases of PTB who infect new hosts due to expectoration of droplets containing bacilli in the environment. Inhalation of these droplets by an uninfected individual can result in pulmonary infection.

2.2.2.2 Primary Infection

Primary infection takes place when the tubercle bacillus (*Mycobacterium tuberculosis*) penetrates beyond the mucous lining of the respiratory system. Primary infection is a stage in the development of TB but does not necessarily lead to disease.

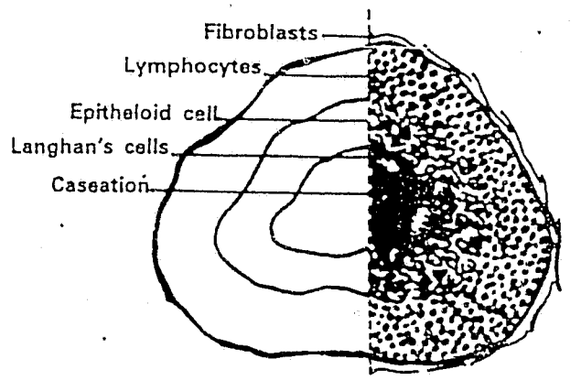
However, when the tubercle bacillus penetrates the alveolar sac it is inevitably engulfed by the macrophages. Unlike other bacilli, these tubercle bacilli not only resist digestion but thrive and multiply within the macrophages. Some of these macrophages containing live tubercle bacilli may be carried to other parts of the lungs or body resulting in extra-pulmonary TB.

After a few weeks, the initial infection leads to a small hard swelling termed a tubercle (nodule). The formation of the tubercle is a result of the clumping of macrophages containing the tubercle bacilli.

Figure 2 illustrates the pathogenesis of TB scar formation indicating the various parts of the tubercle scar as a result of primary infection by this bacilli.

Figure 6

Diagrammatic representation of the structure a tubercle scar



Source:

South African Pharmaceutical Journal,Tuberculosis, May 1983, p 223.

Eventually these macrophages are joined by the T-cells and these clumps grow larger destroying the surrounding lung tissue. As these macrophages die, a caseous area is formed which supports the growth of the tubercle bacilli. At the same time a tough scar begins to form surrounding the tubercle. The purpose of the scar is twofold. Firstly, the scar prevents the further spread of the bacilli and secondly it reduces the amount of oxygen the tubercle receives.

This results in the tubercle bacilli becoming walled in by the scar remaining alive, but inactive and dormant. In many people the Primary Infection produces no symptoms and may go undetected. In some cases symptoms such as fever, rash or nausea may occur.

2.2.2.3 The Disease (Active TB)

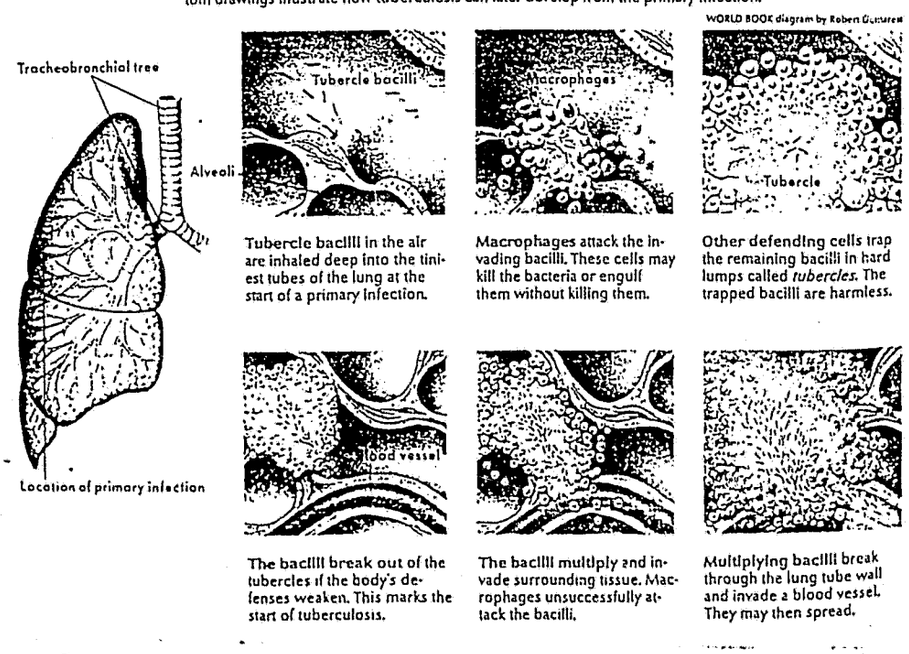
TB may become active if the tubercle bacilli are reactivated or if the person is reinfected. This may occur immediately after Primary Infection. Especially in:
- infants
- children
- elderly people
- ill persons (immunocompromised)
- poor socioeconomic conditions

However, in most cases under epidemic conditions TB develops within 2 years of primary infection exactly what factor or combination of factors triggers the onset of the disease is still today a prime area of research. Endogenous reactivation or exogenous reinfection may occur at any time and it si postulated that when the body's defense mechanism becomes impaired due to other illness, old age or as a result of a second infection of tubercle bacilli, that these factors may play a

role in triggering off the reactivation of the tubercle bacilli leading to active disease. This reactivation results in the rupture of the macrophage and the rapid multiplication of the tubercle bacilli. Cells containing the bacilli may be transported to other parts of the lung, lymph or blood and hence to other organs of the body. This occurs almost solely during primary infection (refer to figure 7).

Figure 7

Stages in Development of TB



Source:

World Book Encyclopaedia, (1991) Vol 19 p478 (diagram by Robert Demarest)

TB of the lung is termed PTB. On reactivation of tubercle bacilli, macrophages accumulate at these sites and this results in the formation of a caseous material which eventually liquifies, moves up the respiratory tract with the mucous layer and is coughed up in the form of sputum. Hence coughing and sputum production are one of the most common symptoms of PTB.

2.3 THE PRESENT & FUTURE OF *Mycobacterium tuberculosis*

Perhaps the pursuit of the conquest of TB and *Mycobacterium tuberculosis* has best been summarized in 1982 at a TB conference at UCT Medical School, by Lipchitz, one of the convenors of the conference committee.

He stated that:[68] "Students at this university have often been criticized for bringing 'politics into medicine.' If this conference is accused of doing this, then we must reply that in order to look at tuberculosis we must examine all factors - geographical, social, historical, and medical - which cause so many people in South Africa, a rich, food-exporting country, to contract TB. And if these factors touch on areas deemed political, we would be failing in our duty as students and as scientists if we did not expose them. Only when we

fully uncover all that lies at the root of the problem, can we begin to plan for the eventual eradication of tuberculosis in South Africa."

With the advent of the AIDS pandemic and dual TB/HIV infection, whatever small victories some of the developing countries may have had in the past in controlling the TB epidemic, will now be reversed.[62]

Even many of the developed countries are now noting an increases in TB incidence.[66,67] Furthermore the emergence of multi-drug resistant *Mycobacterium tuberculosis* (MDR-TB) has further complicated the control of this epidemic.

The chief executive of the British pharmaceutical firm, Glaxo, Richard Sykes, sums up the current TB epidemic as follows: "People thought that TB had been conquered. Tragically this leaves a gap of one generation's worth of research."[63]

CHAPTER 4
EPIDEMIOLOGY OF TUBERCULOSIS

"It is more respectable nowadays to solve problems by statistical tests rather than by logical arguments."

Editorial (1977)
British Medical Journal
2, 418 (1977)

CHAPTER 4

EPIDEMIOLOGY OF TUBERCULOSIS

SUMMARY

The first part of this chapter deals with the current trend of TB in a global context in relation to the prevalence of TB infection, prevalence of dual TB/HIV infection, worldwide TB incidence and mortality as well as the impact of HIV/AIDS.

The second part of this chapter deals with the trend of the TB epidemic in South Africa in relation to the prevalence of infection, infectiousness, risk of developing overt disease and the aspects of dual infection (TB/HIV/Mycobacterioses).

This is followed by an outline on the policy on TB infection in South Africa and the infectious pool based on the Mantoux test. And finally this section then deals with the current trends of TB in South Africa relating to policy, prevalence and mortality.

Previous studies on BCG are discussed and an estimated percentage of BCG coverage in all health regions in South Africa is reviewed in the context of TB notifications, incidence rates within the various population groups as well as the different health regions in South Africa .

CHAPTER 4

EPIDEMIOLOGY OF TUBERCULOSIS

1. CURRENT TREND IN TB

1.1 A GLOBAL OVERVIEW OF TB

1.1.1 Prevalence of TB Infection

It has been estimated that more than 1,7 billion (33%) of the world's population are infected with *Mycobacterium tuberculosis*. The highest prevalence of infection has occurred in the Western Pacific Region where it has been estimated that 44% of the population is infected. With more than a third of the world's population infected with *Mycobacterium tuberculosis* besides those individuals with active disease, TB has achieved the notorious position as one of the worlds most prevalent infections. The advent of the HIV pandemic, will further increase the incidence of TB.[1,2]

A striking difference between the TB infected individuals in Europe and Africa, can be seen in figures 8a & 8b with regards to age. In Europe 80% of the infected individuals are aged 50 year more, whereas in Africa, 77% of infected individuals are aged less than 50 years of age.

Figure 8a

Prevalence of TB infection by age in Tropical & Southern Africa (1990)

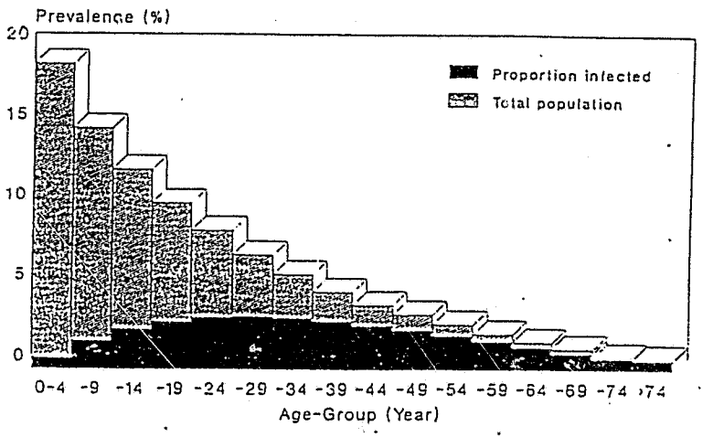
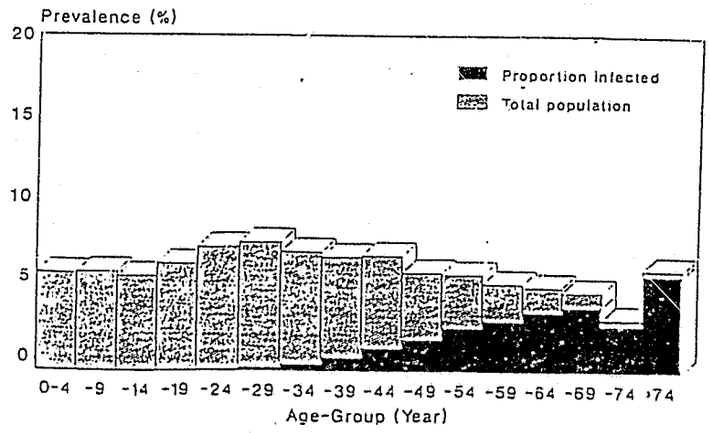


Figure 8b

Prevalence of TB infection by age in Western Europe.(1990).



Source: WHO/TUB/91.58

Estimates suggest that 48% of the adult population aged 15 to 49 years, in Africa, are infected with

the tubercle bacillus. When considering the adult population, of 15 years or older, the prevalence of infection in Africa, South East Asia and Western Pacific Regions is 54%, 52% and 62% respectively.[1]

The best single indicator for the evaluation and the observation of the trend of TB in the developed and the developing countries is the annual tuberculosis rate of infection (ARI).[2,3] This index not only measures the attacking force of *Mycobacterium tuberculosis* in the community but is also independent of TB Control Programme procedures which may have been introduced into the respective communities. Refer to table 3.

Table 3

Relationship between the annual risk of TB infection and the prevalence of smear positive cases of PTB infection and the prevalence of smear positive cases of PTB, Lesotho & Uganda

country	years in which surveys were made	annual risk of tuberculous infection at age 10 years per 10,000 (in 1960)	prevalence of smear-positive cases of pulmonary tuberculosis per 10,000 (at all ages)	ratio of infections per prevalence of smear-positive cases
Lesotho	1957, 1962-1964	410	30,0	13,7
Uganda	1958, 1971-1972	220	21,6	10,2

Source:

Sutherland, I; Fayers, PM. The association of TB infection with age. Bull Int Tuberc.1975; 50:70-81

The most important transmitters of the tubercle bacillus are those patients with active PTB who: [4a]

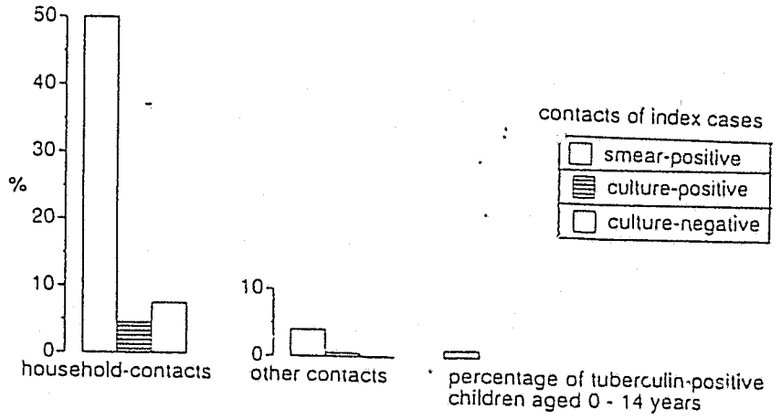
(i) cough

(ii) and produce smear-positive sputum (bacteria in sputum positive (AFB) ZN stain or by fluorescent stain)

The patient producing a smear-positive sputum plays a highly significant role in transmission of the tubercle bacilli, as compared to the patient with a culture positive sputum only or a culture negative sputum. [5] Refer to figure 9.

Figure 9

Percentage of positive reactors among contacts aged 0-14 years, Rotterdam, 1967-1969.



Source:

Geuns, HA; van Meijer, J; Styblo K.; Results of contact examination in Rotterdam, 1967-1969. Bull Int Un Tuberc.1975; 50: 107-121.

1.1.2 The Prevalence of Dual TB/HIV Infection

By 1990, the presence of dual TB/HIV infection was estimated to be greater than 3 million. And 78% (of 3 the million) of the dual TB/HIV infection was estimated to occur in Africa whereas less than 6% of dual infection had occurred in Europe and 5 industrialized countries.[1] The number of dual TB/HIV infections, by 1992, had increased dramatically. Refer to table 4.

Table 4

Tuberculin Skin Test Results In HIV(+) & HIV(-) Adults, Selected Countries, 1991.

Induration (mm)	Uganda (%)		Zambia (%)		Haiti (%)	
	HIV+	HIV-	HIV+	HIV-	HIV+	HIV-
0	50	20	66	34	30	9
1-4	13	9			1	1
5-9	15	5	2	6	15	19
>10	14	59	30	60	54	71
N	106	44	1014		145	1051

Source: WHO/TUB/92.164

Approximately 4 million TB/HIV cases were reported worldwide[6] in 1992, with about 3,12 million (which constitutes 77.8%) living in Sub-Saharan Africa (Appendix D1). A study by Standaert et al,[7] in Burundi in 1989 showed that 62% of the TB patients were HIV positive in the age group

20-40 years had TB. However, in Zimbabwe, in the same year according to Legg et al,[8] 43% of the HIV positive individuals in the age group 20-40 years had TB. This disparity possibly indicates that the full impact of dual infection has not yet been felt in Southern Africa.

It was noted that individuals with dual infection did not respond adequately to purified protein derivative (PPD) reaction (table 4). In the Ugandan study 50% of individuals were missed when a positive PPD reaction of 5 mm induration or more was used as a criterion.[9,10].

1.1.3 Secondary TB

Secondary TB may develop from endogenous exacerbation of an old infection or as a result of reinfection (inhalation of tubercle bacilli) in a previously infected individual. At present there is great difficulty, especially in adults, in determining whether the secondary TB was a result of exogenous infection or endogenous reactivation. Hence, the development of the Exogenous and Endogenous Theory to explain the development of secondary TB.

Controversy between these two theories is not only interesting from an academic point of view, but

also in the future planning in the TB control programme. If the Endogenous Theory is correct then efforts need to be directed towards the prevention of exposure to sources of infection by case finding and treatment. BCG although effective in children it however does not prevent infection. Thus its effect on the spread of TB is therefore very limited.

On the other hand, if the Exogenous Theory is correct the efforts need to be directed to the prevention of the development of the primary disease by laying emphasis on factors (such as prophylaxis) which could prevent dissemination of organisms following infection.

1.1.3.1 The Endogenous Theory

The proponents of this theory state that the tubercle bacillus which caused an earlier primary infection may remain alive for the lifetime of the human host. At anytime the tubercle bacilli in the host may multiply and produce lung pathology (for reasons as yet not clearly understood), resulting in the discharge of the tubercle bacilli via the respiratory tract.

A study, initiated in 1941 by Ferguson in Saskatchewan, Canada, suggests that the majority

of secondary TB cases were mainly due to endogenous exacerbation.[11] The data was collected from mass chest X-rays of the population which continued for 30 years and in addition a tuberculin positive registry for the population of Saskatchewan has been maintained since 1951. From this database the conclusion was drawn for the period of 1960-1969. However, this study should be noted with caution since in this population there is a relatively low incidence of TB. Thus, reinfection would in any event be relatively limited.

Further study by Horwitz,[12] endorsed this point of view. The study suggested that the rates of risk of the disease among the age groups of 15-29 years was 24/100 000; in 30-39 years was 15/100 000; and 50-59 years it was 9/100 000.

However, risk of disease in the age group of 60 years and above was 19/100 000. It was argued that the falling rates of disease were probably due to prolonged interval since infection.

1.1.3.2 The Exogenous Theory

According to Canetti,[13] tubercle bacilli do not survive indefinitely in tuberculous foci. In the pre-chemotherapy era it was shown that the

calcified primary complexes did not contain live bacilli in about 85% of the cases. However, it should be noted that regional lymph nodes often do contain viable organisms.

Further studies by Straub,[14] in 1932-1935, noted that 17% of individuals aged 15 to 20 years had post-primary foci; 50% of individuals aged 30 to 40 years had post-primary foci; and in 67% of those individuals of 50 years had post primary foci. It was thus concluded from this study that number of post-primary foci increased in proportion to the chances of primary foci being sterile.

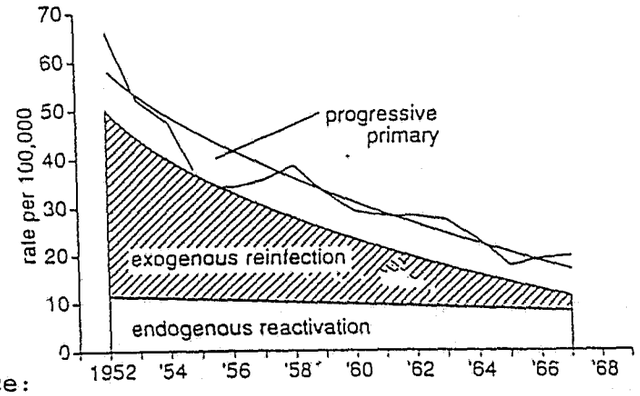
Thus this study favoured Canetti's view that the greater part of the post-primary foci were as a result of an exogenous strain which was in strong support of the Exogenous Theory.

1.1.3.3 Current Belief on Endogenous & Exogenous Theory

Figure 10, illustrates the tendency towards the current belief of these theories to the total morbidity of PTB. It would appear that both theories are correct depending whether the area is a high TB prevalence area or not. It should be noted that Netherlands is a country with a low incidence of TB with an ever declining ARI.

Figure 10

Estimated contribution of progressive primary endogenous reactivation and exogenous reinfection disease to the total morbidity from PTB in the Netherlands at ages 45 to 49 (1952-1967)



Source:

Sutherland, I; Svandova, E. Endogenous reactivation and exogenous reinfection. Their relative importance with regard to the development of non-primary tuberculosis. Bull Int Un Tuberc. 1972; 47:123-4.

A study by Sutherland, [15] in 1972, has provided some statistical evidence on the relative importance of endogenous reactivation and exogenous reinfection for secondary TB. The present belief is that both endogenous exacerbation and exogenous reinfection can give rise to secondary PTB. However, it is believed that almost all secondary PTB in the developing countries is at present due to exogenous reinfection. Whereas in developed countries it is believed that it is due mainly to endogenous reactivation. This is because the risk of TB

infection has significantly dropped in developed countries and increased in developing countries.

1.1.4 Worldwide Incidence of TB

The annual number of TB cases worldwide has been estimated to be between 8 and 10 million with approximately 3 million deaths annually.[16] This trend in the increase in TB is attributed to the HIV pandemic. It has been determined that the annual risk of the dual infected TB/HIV individuals, to develop active TB, varies between 5%-8% with a cumulative lifetime risk of more than 30%.[6] In a study by Sutherland (1968), the following points were concluded:[17a,17b]

(i) Of the 243 cases 54% developed the disease within a year and nearly 80% within two years following the infection.

(ii) Of all the PTB following primary infection, 21% was acquired during adolescence (caviting TB was present). A further 5% of the patients had extensive lesions without cavitation.

(iii) Interval from infection to onset of the disease for different forms of TB. Table 5 illustrates conversion to active form of TB.

Table 5

One-year interval from conversion to onset by form of TB

form of disease	total cases	cases in the 1st year	
		N	%
Miliary tuberculosis, meningitis	10	7	70
Pleural effusion	51	28	55
Pulmonary tuberculosis, no cavitation	111	59	53
Pulmonary tuberculosis, with cavitation	52	30	58
Non-pulmonary	19	8	42
All forms	243	132	54

Source:

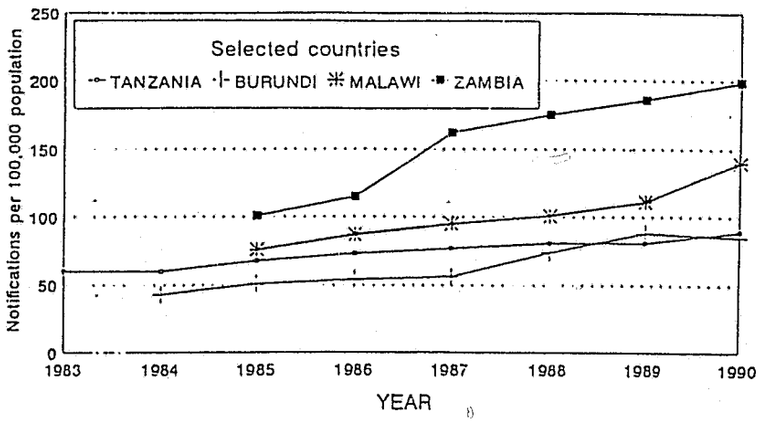
Sutherland, I. The ten-year incidence of clinical TB following "conversion" in 2550 individuals aged 14 to 19 years. TSRU Progress Report 1968. (KNCV, The Hague, The Netherlands.)

1.1.4.1 Worldwide Increase in the Incidence of TB

Over the last decade a worldwide increase in the incidence in TB, has been noted, in developing countries as well as developed countries. In the past 5 years, the increase in incidence of TB has been even more dramatic.

Figure 11

Annual TB Notification Rates, In Selected African Countries (all cases): 1983-1990



Source: WHO/TUB/92.164

In 1990, Tanzania reported an increase of 86% in notified TB cases, over cases reported compared to 1984 for the same period. An even greater increase, of 140%, 180% and 154%, was noted for the same period in countries such as Burundi, Malawi and Zambia respectively.[6]

Similar trends in TB incidence have been observed in the developed countries especially in the urban areas in HIV infected individuals. By 1984, the USA, reported an Increase in TB incidence for the first time in 30 years. Increased incidence in TB has been mainly attributed to HIV infected individuals.[18]

1.1.5 TB Mortality

The worldwide TB mortality rate in 1990, was estimated to be 50 per 100 000 population.[1] This rate included the additional deaths due to TB/HIV deaths which was estimated to be in the range of 120 000 to 150 000. It was calculated that 83% of TB/HIV deaths occurred in Africa indicating a 20% increase over the initial prediction. According to Styblo, the death rate can be estimated to be one-quarter of the prevalence of smear positive PTB.[4b]

TB/HIV infected individuals may have a short-term mortality. The case fatality ratio among HIV-seropositive individuals was 31,3% (47 of 150), at one year after diagnosis as compared to 4,4% (22 of 501), for HIV-seronegative individuals.[19] A cohort study in New York City, demonstrated that HIV infected TB patients had a poor prognosis for survival.

From the time of diagnosis of TB among seropositive patients, the median survival time was estimated to be 21 months which was similar to the median survival time of 20 months in 1 452 TB patients who already had AIDS.[20]

1.1.6 AIDS and TB

TB has now, especially in developing countries, become the most prominent opportunistic disease in those individuals infected with HIV. In Africa about 20-40% patients with AIDS developed TB as compared with 4% of AIDS patients in USA.[21,22] In 1993, the Center for Disease Control and Prevention (CDC) expanded its definition of AIDS to include those infected with HIV who also had a severely suppressed immune system, TB, recurrent pneumonia or invasive cervical cancer.[78] In brief, the HIV/AIDS pandemic will exacerbate the TB epidemic in developing countries and in all likelihood will initiate a resurgence of a TB epidemic in the developed countries. Worldwide the TB problem will worsen taking into cognizance the following factors:[23]

(i) reactivation of latent tubercle bacilli in individuals infected with HIV.

(ii) new infection of tubercle bacilli with rapid progression into active disease state in individuals infected with HIV.

(iii) HIV/TB infected individuals developing TB by either reactivation or recent infection will cause an increase in the number of tubercle bacilli that will be transmitted to contacts,

thus resulting in a further increase in the incidence of TB.

2 TREND OF TB EPIDEMIC IN SOUTH AFRICA

2.1 PREVALENCE OF INFECTION (Appendix D2)

2.1.1 Infectiousness

Studies elsewhere in the world indicate that the infectiousness of PTB can be directly linked to the:[24,25]

(i) load of the tubercle bacilli in the sputum
(ii) cough frequency
(iii) environmental factors (overcrowding)
(iv) closeness of contact
(v) duration of exposure

2.1.2 Risk of Developing Overt Disease (Appendix D3)

Data of recent studies have shown that the tubercle bacillus has infected approximately 6-10 million individuals in South Africa. It has been estimated that a substantial portion of this pool of infected people have a risk of developing active TB in their lifetime.[26] This risk has been estimated to be in the range of 5-15% in the privileged communities and it is believed that it may rise to 40-50%[27,28] in some deprived communities. Some of the contributing factors that

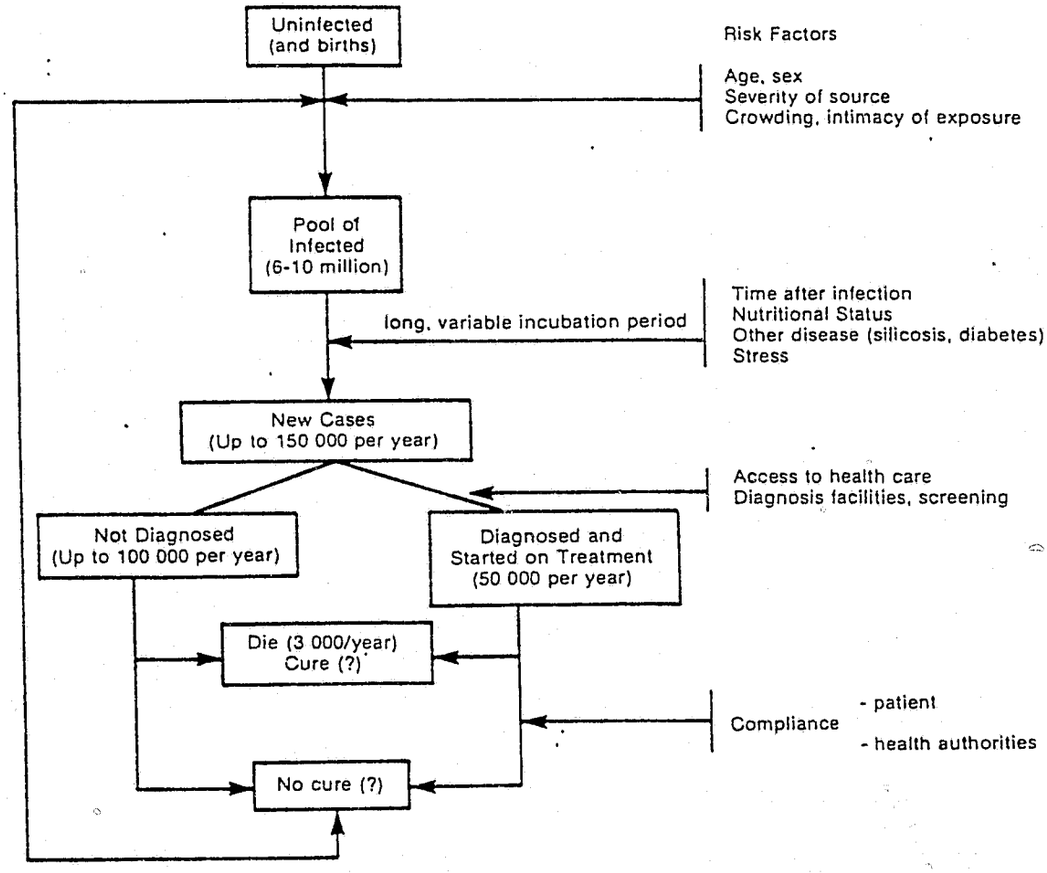
may result in TB from the time of infection are time period of infection, age, ethnic group, gender, rare medical conditions and HIV. Refer to figure 12 for factors that may contribute to infection and disease.

The risk of developing disease is the highest in the first year after infection and declines over a period of 7 years or more.[29,39] Studies with regard to age as a contributing factor for disease has shown that the risk of disease (especially disseminated forms such as TB meningitis or miliary TB), is highest in the infected children and that it decreases sharply up to the age of 10 years.[30] Although in South Africa the TB notification rates do appear to be different in the various ethnic groups, its been suggested that this is due mainly to the higher risk of infection rather than ethnicity as a factor (Appendix D3).

At present there are no available data to suggest that certain ethnic groups could have an increased genetic susceptibility to activation of latent TB infection.[37b] A simplified model of TB in figure 12, illustrates infants at birth are not infected. Over a period of time one becomes exposed to infection.

Figure 12

Simplified model of Tuberculosis



Source:

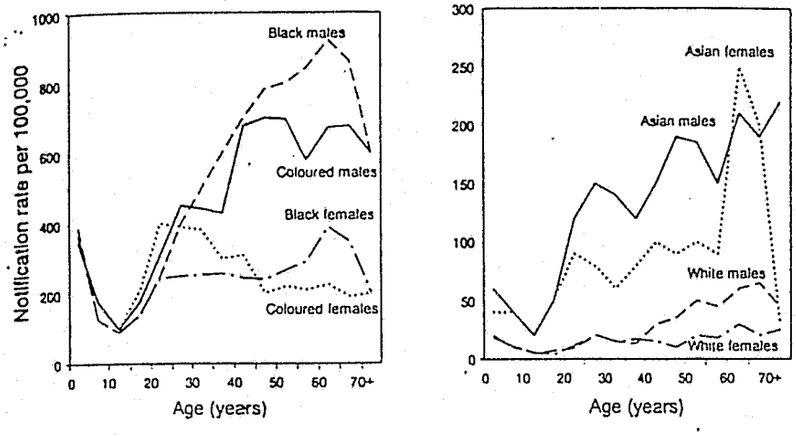
Yach, D Review of South African Mortality, MRC, Technical Report, 1984; No 1 (p48).

The 95% confidence interval for relative risk of an infected individual developing TB who is on

immunosuppressive treatment is (9;11) whereas the individual on haemodialysis is (10;15) and those individuals that are diabetic is (2,0;3,6).[31] However, there is a tendency to see a higher TB notification rates in males as compared to females for all ethnic groups in South Africa, except during adolescence as can be seen in figure 13.

Figure 13

Age-ethnic-group-gender-specific tuberculosis notification rates, South Africa in 1975.



Source:

Kustner, HGV. Trends in four major communicable diseases. S Afr Med J. 1979; 55, 460-73.

2.1.2.1 TB/HIV/Mycobacterioses

Judged by the accumulated evidence dual infection (TB/HIV) exacerbates the TB incidence. The rise is believed to be due to the interaction in the risk

of infection in developing and developed countries together with the spread of HIV. South Africa already burdened with a high prevalence rate of TB and an increasing rise in HIV sero-prevalence, will have to focus urgent attention on the TB and HIV epidemic if it is to avoid a holocaust of an unprecedented magnitude. There is an urgent need to prioritize TB for positive action. The other side of this dual infection in humans are mycobacterioses, caused by *Mycobacteria* other than TB (MOTTS). This is as a result of 'atypical' (termed environmental) mycobacterial infection in humans. These organisms (MOTTS) are potential pathogens for causing human disease which is indistinguishable from TB.[32]

Dual infection due to environmental mycobacteria is expected to be more commonly manifested in AIDS patients, especially the species *Mycobacterium avium-intracellulare* complex which is termed MAI and *Mycobacterium kansasii* which are the most common species prevalent in Southern Africa.

MAI is responsible for more 90% of mycobacterioses in AIDS patients in developed countries,[33] whereas *Mycobacterium kansasii* which is rarely associated with AIDS cases, is responsible for more than 50% of all mycobacterioses in HIV

negative individuals. According to Kleeberg,[34] these environmental ('atypical') *Mycobacteria* have a twofold effect:

(i) they are a possible source of human infection

(ii) they may have a protective effect on human susceptibility to other mycobacterial infections.

It is believed that these *Mycobacteria* sometimes confer as much protection as BCG; that is the protection afforded by:[35]

* *Mycobacteria kansassi* was 85%
* *Mycobacteria avium-intracellulare complex* (MAI) was 50%
* *Mycobacteria fortuitum* was 15%

Of the six hypotheses resulting from the Indian BCG Trial, one was that environmental *Mycobacteria* had a protective effect that was as great as BCG vaccination. Ironically, with the prevailing HIV pandemic this protective effect from environmental *Mycobacteria* against TB will no longer be maintained and a rise in the incidence of mycobacterioses will be inevitable. In the early stages of HIV/AID epidemic, of the 212 AIDS patients suffering from TB or mycobacterioses in South Africa, there were:[34]

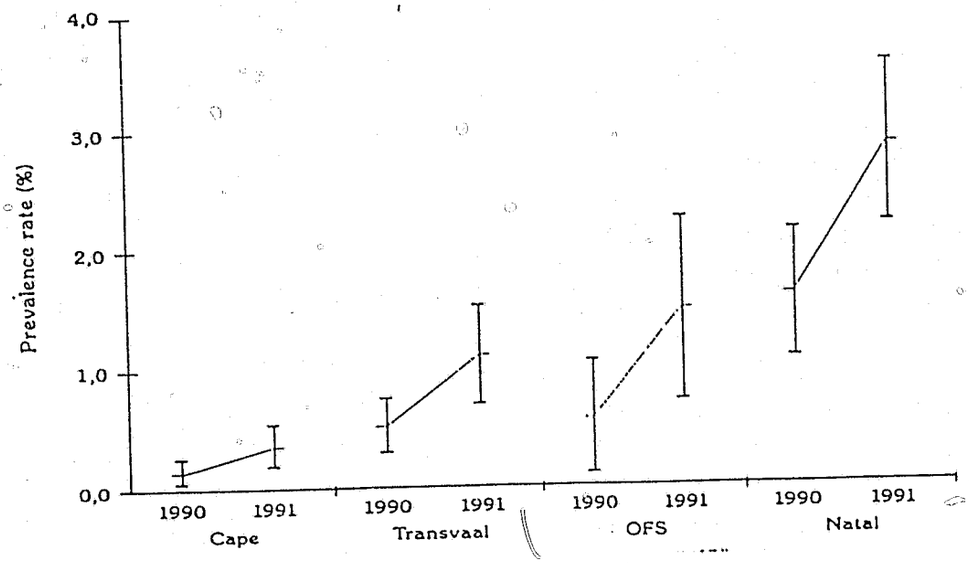
(i) 80% infected with MAI

(ii) and 9% with *Mycobacterium tuberculosis*.

A dual infection is still in the early stage of the epidemic in South Africa. In the future, as this dual infection progresses, susceptible individuals in the high TB incidence areas will develop TB rather than mycobacterioses. Figure 14 displays HIV surveys for 1990 & 1991.

Figure 14

The first & second estimate national HIV surveys 1990 & 1991. Estimated prevalence of HIV infection in women attending antenatal clinics by province including self-governing national states (estimate shown with 95% confidence intervals)



Source:

Epidemiological Comments, Second national HIV survey of women attending antenatal clinics, South Africa, October/November 1991.

Two recent national surveys undertaken in South Africa (1990 & 1991), estimated the prevalence of HIV infection in women attending antenatal clinic in all 4 province as well as the self governing national states. The surveys revealed that HIV infection was increasing in all 4 provinces with lowest percentage prevalence in the Cape and the highest in Natal. Refer to figure 14.

2.2 SOUTH AFRICAN POLICY ON TB INFECTION

The Tuberculosis Programme in South Africa (TBCP) in 1979, had recommended in its national TB policy a goal of reducing the risk of infection in the country.[36] The following is an extract of those objectives: "To reduce the risk of contracting tuberculosis infection to 0,3% and below for all population groups in the Republic of South Africa; and to ensure effective treatment of all tuberculosis disease that occurs and is diagnosed." This goal is in line with the international recommendations on TB control policy. This goal was to be achieved, by a two phase strategy aimed at the reduction of:

Phase I: Infectious pool

(i) active and passive case finding

(ii) supervised short course therapy

(iii) BCG vaccination

Phase II: Infected pool & endogenous reactivation

(i) secondary chemoprophylaxis for positive tuberculin reactors

(ii) BCG vaccination

(iii) continuation of methods used in Phase I

These idealistic goals have as yet not been achieved, and today in South Africa approximately 1 in every 4 individuals is infected with tubercle bacilli. The factors that have undermined TBCP's goals are numerous. Some of the factors that may have resulted in failure to reduce the level of infection in South Africa are, the result of the fragmented health service which has caused: [37a]

(i) weak Primary Health Care (PHC)

(ii) poor central coordination of TB control

(iii) insufficient welfare system

(iv) lack of community involvement

(v) inadequate support and referral systems

(vi) inappropriate health education

(vii) poor integration of health worker

(viii) insufficient evaluation

2.2.1 Tuberculin Skin Test - Mantoux Test

The presence of tuberculous infection can be detected by means of a tuberculin skin test. Although the Heaf and Tine tests are suitable as

screening tests in a clinical setting, the Mantoux test is more accurate and quantifiable and is not only used in a clinic setting but is best suited for epidemiological studies. The tuberculin test serves towards:

(i) determining the prevalence of TB infection in a community

(ii) evaluating the effectiveness in the anti-TB campaign

(iii) determining the prevalence of MOTT in the community (only if species specific tuberculin is used)

(iv) the differential diagnosis of TB

(vi) identifying those individuals in need of vaccination

(vii) identifying those individuals in need of chemoprophylaxis

(viii) providing information on conditions effecting reactivity

Because, infection with 'atypical' bacteria cause smaller reactions and seldom develop into disease, it is maintained that the risk of developing TB is directly related to size of the tuberculin reaction.[38,39] However, the results of this test can be dubious if viewed on its own in a clinical setting. The reason being that it becomes

difficult to distinguish the infected individuals, from the non-infected individuals because of various factors. These are:[37b]

(i) the reaction is non-specific to mycobacteria other than TB (MOTT). But seldom >15 mm.

(ii) previous BCG vaccination causing false positive reaction. But seldom >15 mm.

(iii) reaction could be false negative in malnourished individuals

(iv) reaction could be false negative in immunocompromised individuals (HIV/AIDS individuals)

Even with false positives due to localized infection of injection site as limitations (as well as the factors stated above), the test produces reactions which on an average fall between the normal positive and the negative reactions (table 6).[40,41] According to Seager et al,[42] 59% (sensitivity) of infected children were correctly identified by the Mantoux tests with only 2% (specificity) false positives.

Hence, one is able to quantify the prevalence of infection by observing the reaction sizes of induration as the result of the tuberculin skin tests. Refer to Appendix D4(a) and D4(b).

Table 6

Potential effects of misclassification in allocating 10 000, 5-15 year old children for prophylactic treatment on the basis of Mantoux Results.

	INFECTED		
	Yes	No	
MANTOUX ≥15 mm	1 350	150	1 500
MANTOUX <15 mm	935	7 565	8 500
	2 285	7 715	10 000

Source:

Seager, JR; Fourie, PB; Kleeberg, HH & Felten, MK. Is preventive treatment of schoolchildren worthwhile? SANTA News. 1985;24(4), 4-5.

In South Africa, consistent results were shown with the addition of reactors >=10mm and >=15mm and dividing this total by two, to establish a cut-off point for the positive Mantoux test. [40] It has been estimated that the incidence of TB in tuberculin positive individuals is 4 times greater than in those individuals who are tuberculin negative. [43]

The recommendations by the Tuberculosis Control Programme (TBCP) suggest that a positive reading of induration following a Mantoux test is regarded as the following: [44]

(i) >10mm (in individuals with no previous BCG)

(ii) >14mm (in individuals with previous BCG)

(iii) >4mm (HIV positives)

Currently the Department of Health regards a child under 5 years of age with a significant tuberculin reaction as notifiable (Appendix D5).

2.3 CURRENT TRENDS OF TB IN SOUTH AFRICA

2.3.1 South African TB Policy

One of the overall objectives of the TBCP clearly states:[44] "A second and more pragmatic measure, albeit considerably less accurate, is the notified annual TB incidence rate, and the reported and notified mortality rate from TB. It is the objective of the TBCP to ensure that by 1997 no community in the country shows a rising trend in any of these variables. Thereafter, the objective will be to strive for a reduction in notified TB disease and death rates of no less than 3% per five-year period.

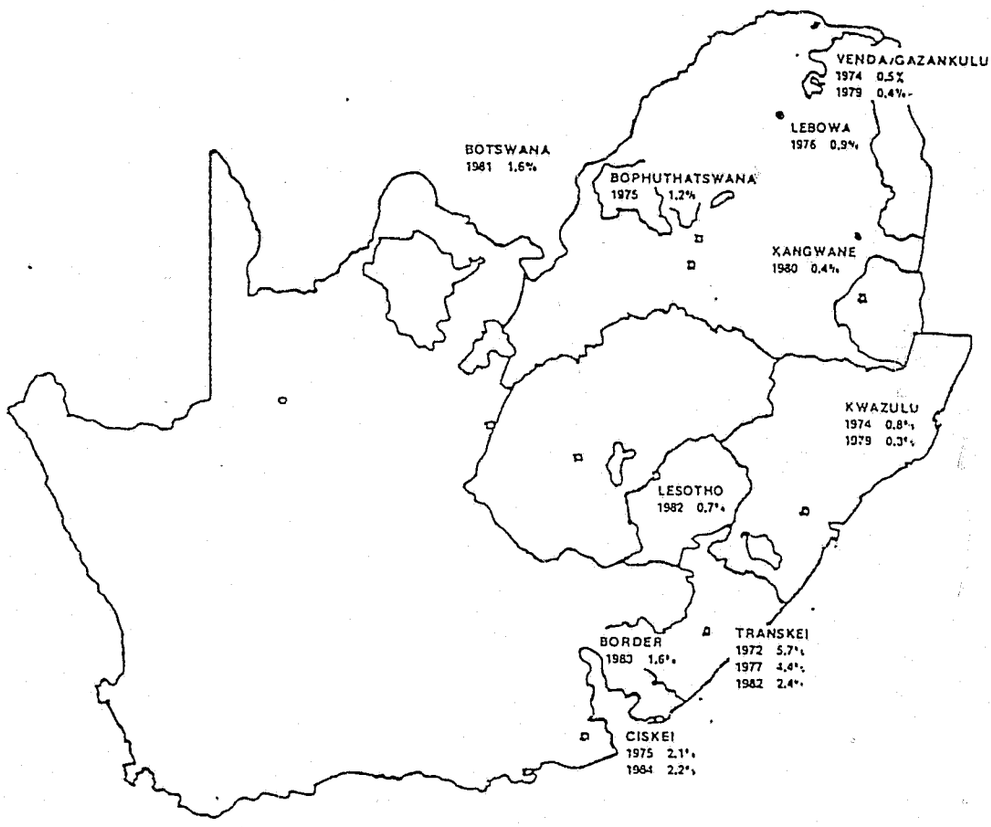
2.3.2 Prevalence of Disease (TB) in South Africa

By 1983, both the prevalence of TB in adults and the risk of infection in children were on the decline with the exception of the Coloured

population in the Western Cape.[40] Figure 15 illustrates the prevalence of culture positive TB in rural South Africa

Figure 15

Prevalence of Culture positive TB In Rural Southern Africa



Source:

Knoetze, K (1987). Review of TB research (1987-1986) and perspectives on future research at the Tuberculosis Research Institute, MRC.

In 1980, Prof Glatthaar made the following statement:[45] "The eradication of TB in our lifetime is an unattainable dream, and planning must be in terms of decades, not years. Our inability to eradicate TB can be blamed entirely on the unpredictable behaviour of the reservoir of infected persons in the Republic. We are burdened with this huge reservoir of infected persons and we can only decrease the inflow and arrest further expansion of the pool but we cannot control the outflow of the infectious cases."

According to Kleeberg,[46] although it was not possible to reduce the number of persons infected at this stage it would however be possible to find most of the infectious people in the pool and thus possibly control the outflow from the pool and hence reduce the risk of infection.

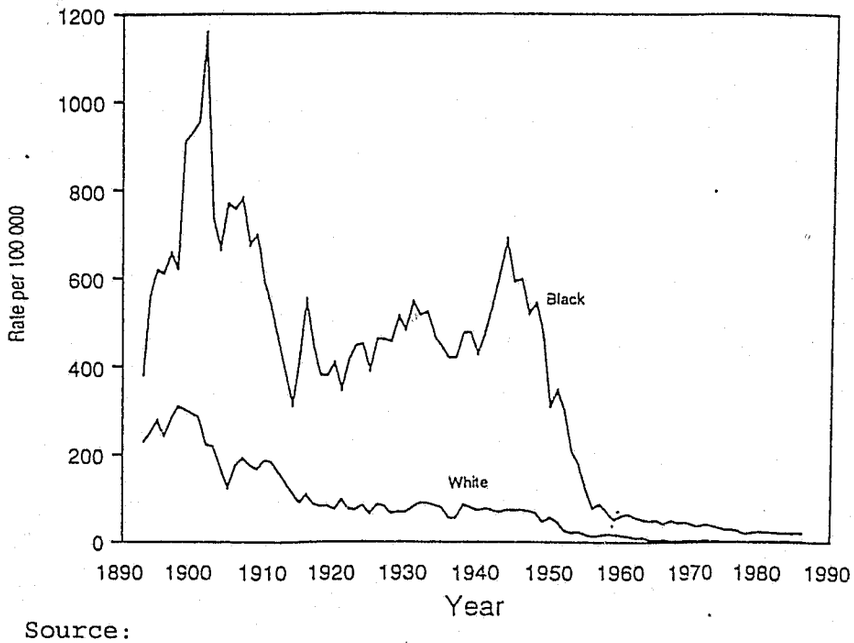
This rationale makes good sense especially if one views this in the light of the fact that one sputum smear positive individual infects an average of 10 people per year and can remain infectious for two years or more.[47] It is estimated that between 33% and 70% of the close contacts of active PTB patients are infected and in South Africa it is estimated that 8% of the contacts are suffering from TB.[29,48]

2.3.2.1 TB Mortality in South Africa

Mortality figures are probably as high as 20 or more per day in South Africa judging from the death certificates received by the Central Statistical Service in Pretoria. [49,50]

Figure 16

TB Mortality Rates (all forms of TB) by ethnic group for the city of Cape Town 1893-1986



Source:

Strebel PM, Seager, JR. 1990; A Century of Tuberculosis in South Africa: South African Perspectives. Chapter 4, Oxford University Press. Cape Town.

The TB mortality rate among whites in Cape Town during 1903 was on the decline with death rates

similar to those of Europe where the major TB epidemics had occurred in the 18th and 19th centuries.[51] According to Lee & Buch,[37a] TB mortality figures are a reflection of failure of case finding and/or case-holding. TB mortality figures are generally regarded as a good indicator of the anti TB campaign programme worldwide.

Table 7

Notified TB Cases & Fatality Ratios (CFR) by Type of TB in South Africa (1987). (excluding TBVC)

TYPE OF TUBERCULOSIS (ICD 9)	NUMBER OF CASES	(%)	NOTIFICATION RATE PER 100 000	CASE-FATALITY RATIO
Pulmonary (011)	50 243	(93,7)	173,6	4,3
Primary (010)	2 079	(3,9)	7,2	0,1
Intestinal (014)	403	(0,8)	1,4	3,2
Central nervous system (013)	242	(0,5)	0,8	27,7
Bones and joints (015)	177	(0,3)	0,6	4,5
Miliary (018)	150	(0,3)	0,5	19,3
Other respiratory (012)	71	(0,1)	0,2	8,5
Genito-urinary (016)	58	(0,1)	0,2	0,0
Other organs	204	(0,4)	0,7	2,9
TOTAL	53 627	(100,1)	185,3	4,3

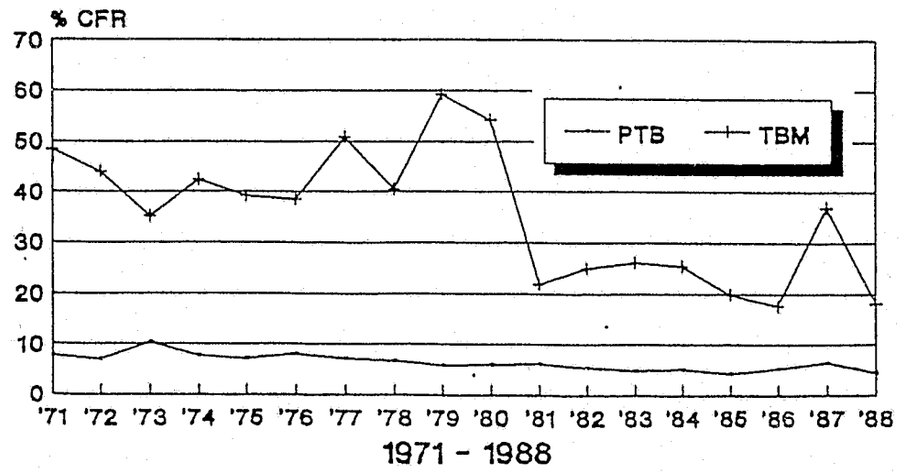
Source:

Weyer, K & Fourie, PB. Die Epidemiologie van Tuberkulose in Suid Afrika. CME. 1989; 7:239-47.

The highest case fatality ratio (CFR) of TB is as a result of TB of the central nervous system (TB meningitis which occurs mainly in children). The overall CFR in South Africa, was 4% in 1988. It was estimated that the overall CFR (based on available data), could be in the range of 4-8%. TB mortality was the highest among the Coloureds and Blacks aged 16-64 years where it was responsible for 6-8% of all deaths in South Africa.[52] Recent data suggests that TB mortality rates may be increasing. Refer to figure 17 illustrating CFR among Coloureds in South Africa (1971-1988).

Figure 17

TB CFR Among Coloured Population in South Africa



Source: Appendix D6

2.3.2.2 Bacille-Calmette-Guerin (BCG) Vaccination

This vaccine is named in honour of Calmette and Guerin who first initiated the production of this vaccine. In 1921 this vaccine was administered for the first time to a human subject.[53] In Britain BCG was introduced on a wide scale in the 1950's.[54] In South Africa the BCG campaign commenced in the 1960's and was made compulsory for all newborn children between 1973 and 1989.

There are 130 countries where BCG is widely used and 69 countries where BCG is mandatory.[55] Compulsory BCG in South Africa, was removed from the statute books in spite of the Expanded Programme on Immunization (EPI) of WHO which specified that TB is one of the six target diseases. The reasons given by South African health authority was because it had become a difficult exercise to make BCG compulsory by enforced legislation. Despite the removal of compulsory BCG from the statute books, health facilities continued to administer BCG routinely and freely to new born children in South Africa.

The efficacy of BCG ranges from 0-80%.[56] In the largest BCG trial ever conducted, (in Chingleput South India) with 260 000 people as subjects for

the trial,[57] BCG did not confer protection against PTB during the first seven and a half years after vaccination.[56,58] However, researchers involved in this study have acknowledged that non-specificity may have been the sole factor responsible for the lack of protective effect of BCG, while Grzybowski believes that presence of atypical *Mycobacteria* may have been the cause for the ineffectiveness of BCG in the prevention of development of TB.[59,60]

Studies in South Africa, have indicated that a 65% protective effect against active disease, can be attributed to BCG in children under 5 years, in an urban setting and 58% in rural population.[61,62] Although there is evidence to suggest that BCG does not reduce the risk of becoming infected, it is believed that BCG can result in less frequent and severe manifestations of TB (TB meningitis and miliary TB).[63,64] A study of children living in households in Pretoria with known adult TB cases, demonstrated that BCG provided considerable protection to children under 4 years but little protection for children above 5 to 15 years.[65] However, in Cape Town, a study of TB meningitis cases in children (1979-81), revealed that 45% of the diagnosed cases had had BCG vaccination.[66]

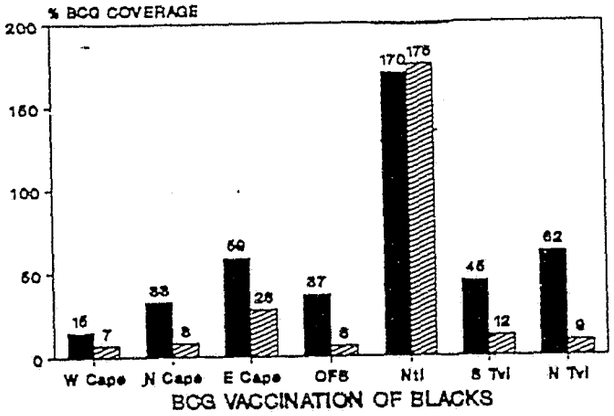
The true protective effect and the effectiveness of BCG, in South Africa may have been masked due to high levels of malnutrition, overcrowding, in an environment where TB is endemic and where heavy and repetitive infection may have overwhelmed the protection afforded by BCG.[67] BCG is recommended to the following persons:[68]

(i) Individuals living in high prevalence areas and high risk groups

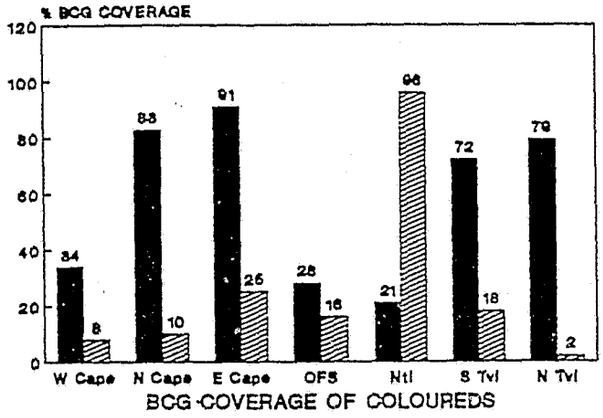
(ii) Tuberculin negative contacts of diagnosed tuberculotics

(iii) Newborns (it has been recommended that BCG be given as early in life as possible especially in countries with high risk of infection).[53]

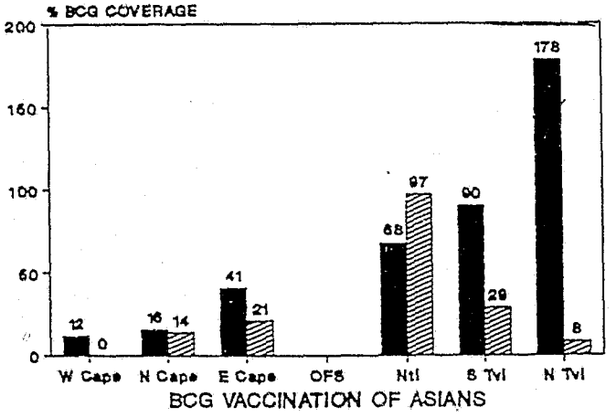
The BCG coverage in South Africa in 1988 for all health regions, was 58% and 26% for the first and second dose respectively.[69] Figure 18a-d however, reflects only clinic records and the BCG administered in hospitals was not notified to the local authorities. The actual BCG coverage is probably higher than 90%. In figure 18a-d the BCG coverage was determined from local authority clinic records only. It was the worst for all population groups in the Western Cape.



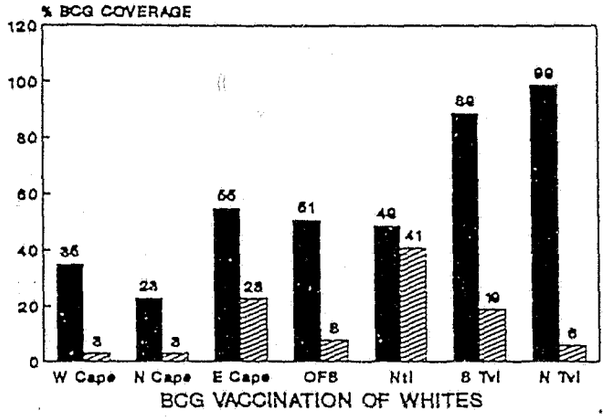
(a)



(b)



(c)



(d)

ESTIMATED BCG % COVERAGE HEALTH REGIONS (RSA - 1988)

EXCLUDING SELF-GOVERNING NATIONAL AND INDEPENDENT STATES

BCG DOSE

DOSE 1 DOSE

Source: Appendix D7

Figures 18a, 18b, 18c & 18d thus summarizes:

- Blacks; first dose 16% and second dose 7%
- Coloureds; first dose 34% and second dose 8%
- Asians; first dose 12% and second dose 0%
- Whites; first dose 36% and second dose 3%

2.3.2.3 TB Notification in South Africa

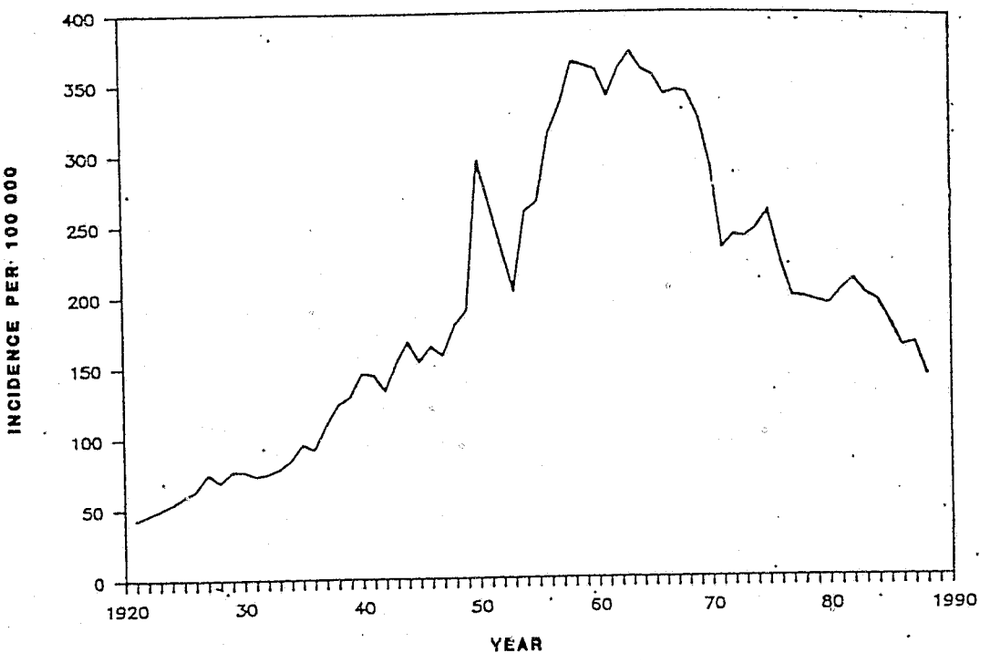
TB became a notifiable disease under Section 18 of the Public Health Act 36 of 1919. Since 1921 the TB incidence rates steadily rose from 43/100 000 population to 372/100 000 population. The peak of the TB epidemic was noted to be in 1963.[70] At present, TB accounts for more than 61% of all notifiable diseases in South Africa.[71]

Primary TB was made notifiable medical condition in South Africa, in 1980 and in 1987, 57% of all cases of primary TB was from the Western Cape.[72] It was believed (and possibly still by some) that this peak of the TB epidemic was reached by the late 1950's.

Many have attributed this TB decline to drug therapy and BCG vaccination and have led some to 'falsely' believe that the TB epidemic was on the decline. Figure 19 depicts the TB incidence in South Africa (1920-1985).

Figure 19

The Incidence of TB in South Africa, (1920 - 1985.)



Source: CME., 1989; 7:3, p241

Packard[73] holds a contrary view on the TB decline in the late 1960's and terms it as an "optical illusion". He claims that it reflects an apparent decrease rather than a true decrease of the TB rate. Packard attributes this decline to the establishment of the 'homelands' and the large scale reallocation of Blacks.

According to Packard, these factors resulted in the displacement of the disease to areas where TB cases were less likely to be detected and notified. Furthermore, he supports his theory by showing that the TB notification figures in this period were either absent or low thus indicating that under-reporting of TB notification had taken place.[74] The reasons for this could be attributed to the 'displacement affects' which affected the denominator and the numerator. And this was further compounded by under reporting. And most importantly the population that this was taking place was not included in the calculation.

By May 1992, the total number of notified PTB[75] cases in South Africa (excluding TBVC) was 79 130 in a population of 32 134 000 (based on 1985 census in the Epidemiological Comments 14(1)

2.3.2.4 TB Incidence in South Africa

Between 1977-1988, the TB incidence rates in South Africa were more or less stable. The rates varied between 180 (1986) and 205 (1983) per 100 000 population and by 1989 and 1990 a further rise in TB incidence rate was noted to the effect of 211 and 229 per 100 000 population respectively.[76] The figure 20 depicts the steady increase in TB

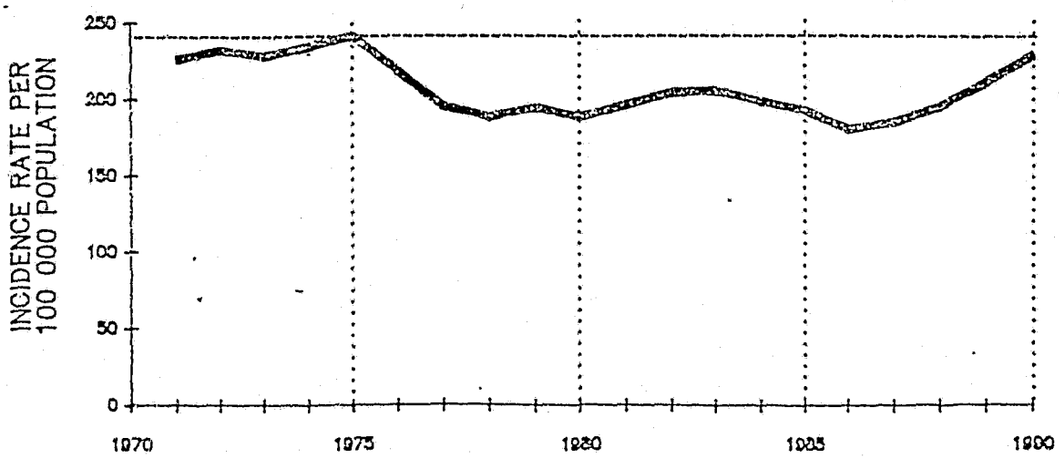
incidence of the population in South Africa.

This increase could possibly be attributed to one or more of the following reasons:

* A real increase in TB case (reasons as yet still not clear)
* Infectious individuals not on treatment resulting in the spread of infection
* Unrest in many parts of the country may have resulted in - stress
  - disruption of routine services
* increasing prevalence of HIV infection in RSA
* large portion incomplete treatment (21% in 1988)
* higher incidence in TB could be artificial

Figure 20

TB Incidence in RSA, 1971 to 1990 (24 June 1991)



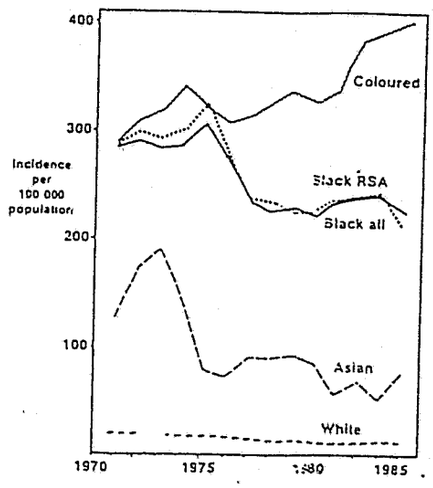
Source:

Epidemiological Comments. Tuberculosis on the Rise. 1991 18(6):147.

On review of the graph in figure 21 for the TB notification rate of the different population groups in South Africa, the current notification rate, is clearly the highest for the Coloureds.

Figure 21

TB Notification Rate by Population Group



Source:

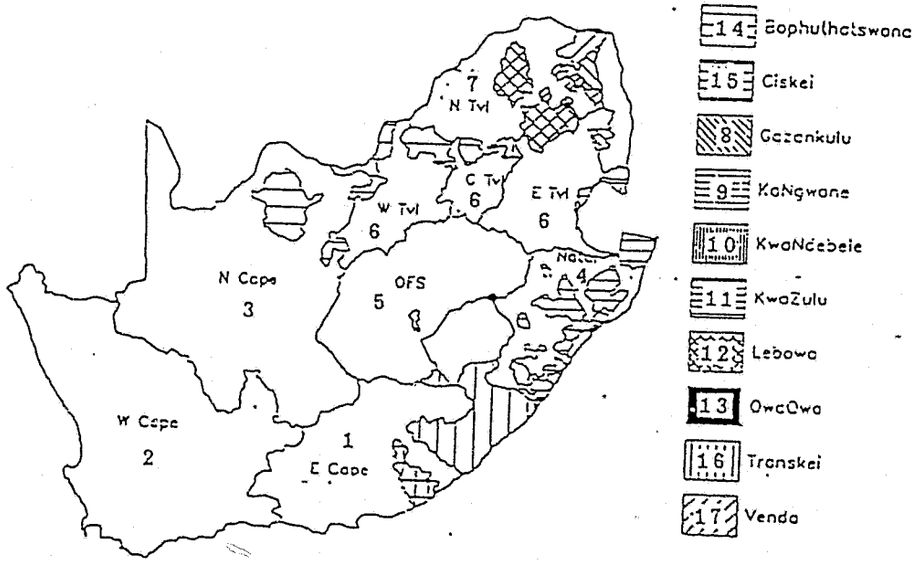
Epidemiological Comments. Epidemiology of TB. 1985; 12,9:2-19

Figure 22a is a map of South Africa which depicts all the health regions (including all the self-governing and the independent National States prior to the new constitution of 1993). Refer to Appendix D8 for additional information for each of the provinces. And the figure 22b illustrates the

notification rates of PTB (per 100 000 population), where Western Cape in 1991 has had the highest PTB as compared to all other health regions in South Africa. The Eastern Cape and the Orange Free State health regions fall into second and third place with regards to PTB notification rates.

Figure 22a

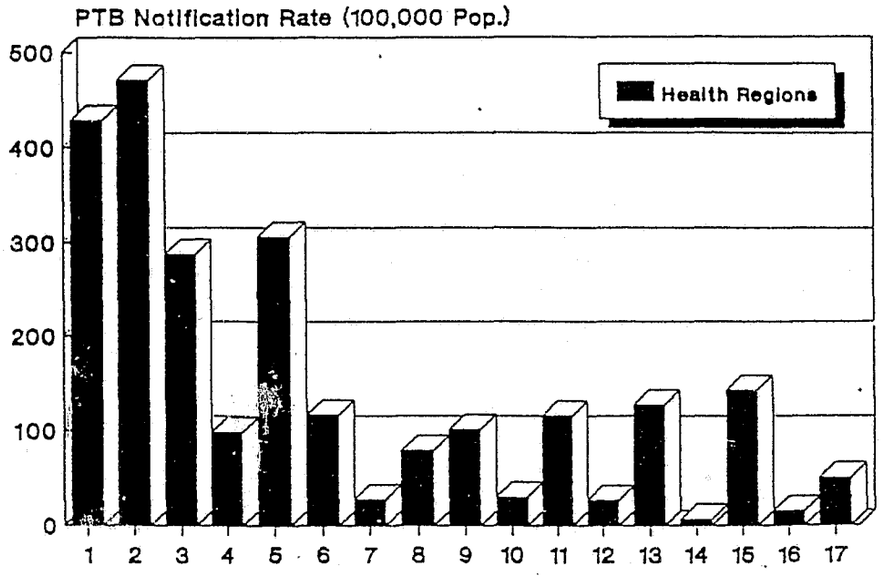
Health Regions of South Africa Self-governing & Independent National States



Source: Appendix D8

Figure 22b

PTB - Notification Rates (1991) All Health Regions (South Africa)

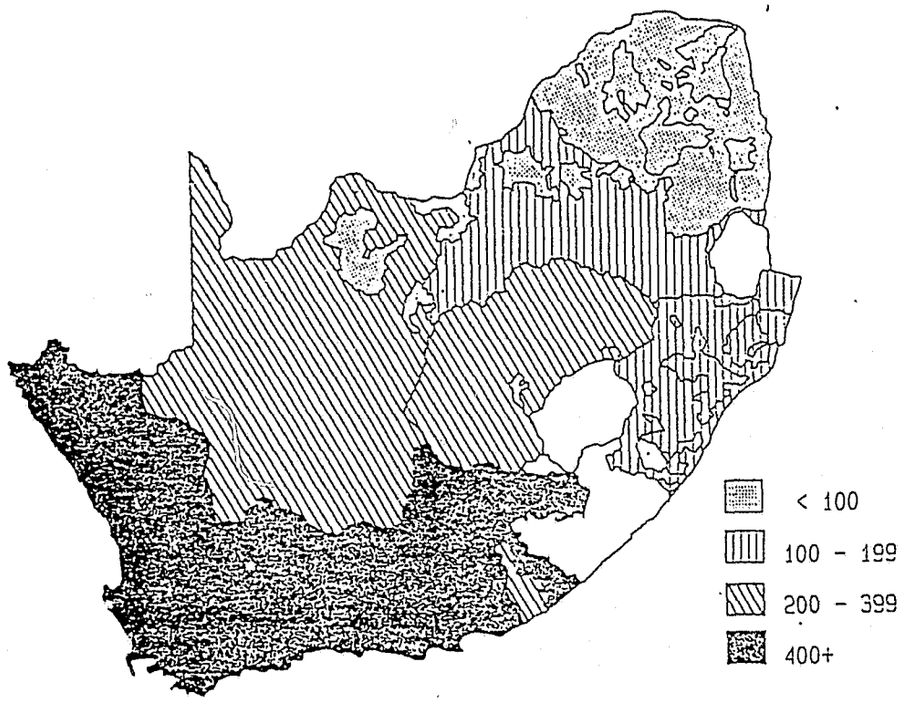


Source: Appendix D8 & Figure 22a

By 1989 the incidence of all forms of TB based on the geographical distribution in South Africa showed that the Western Cape together with the Eastern Cape had the highest TB incidence rates (>400 per 100 000 population), followed by Northern Cape together with Orange Free State with an incidence rate ranging between 200 - 399 per 100 000 population (figure 23).

Figure 23

Incidence Rate of all forms of TB Geographical Distribution (1989) South Africa



Source: Epidemiological Comments, (1991).19(1):6

The Western Cape can thus be dubbed as the TB capital of the world. Attempts to halt the increase in TB incidence, have as yet not made any significant impact on the epidemic. Now with advent of dual infection of TB/HIV, the epidemic will become even more difficult to combat.

Ironically our knowledge of the past record of *Mycobacterium tuberculosis* and the TB epidemic which has plagued mankind from time immemorial will be ignored. This facet of human nature has been succinctly summed up by Wilson Carswell:[77] "Since 1984, I have been involved in several aspects of the HIV pandemic, in Africa and elsewhere. Much of this work has been to encourage governments to implement preventive programmes. And in 1991/92, I was a Medical Adviser to the South African Government's AIDS Unit. A depressing feature of this pandemic is the certainty that when HIV arrives in a country the same cycle of responses has to be played out once again. Countries find it difficult to learn from other's mistakes, and South Africa is no exception."

CHAPTER 5

EPIDEMIOLOGY OF TUBERCULOSIS IN THE WESTERN CAPE AND MACASSAR

"All experience hath shown that mankind are more disposed to suffer, while evils are sufferable, than to right themselves by abolishing the forms to which they are accustomed."

Declaration of Independence
July 4, 1776

CHAPTER 5

EPIDEMIOLOGY OF TUBERCULOSIS IN THE WESTERN CAPE AND MACASSAR

SUMMARY

This chapter commences by describing the Western Cape Health regions and the distribution of various population groups as well as the local authority jurisdictions in the Western Cape Regional Services Council. It then proceeds to review and describe the development of the TB epidemic in the Western Cape by substantiating data of TB incidence in the Western Cape in comparison to the rest of South Africa in 1989 as well as the TB incidence rates and the trend in the Coloured population.

TB epidemic in the Western Cape is presented based on available data. It is implied that the TB epidemic in the Western Cape could be attributed to various factors such as reduction of hospital beds, poor case finding and case holding, poor quality of treatment, compliance and contact tracing. BCG coverage, nutritional status, overcrowding, occupational status as well as occupational risk are discussed at length in this chapter.

Furthermore the TB trends in the various areas of the Western Cape Health Regions are discussed and data is presented indicating the PTB incidence of the Western Region (W/RGN) which is comprised of the Cape Branch of RSC and Cape Town Municipality as well.

This chapter then proceeds to introduce the area Macassar on the False Bay coast about 40 km from Cape Town. A brief the description of the area of the study which is Macassar Camp and the demographic data pertaining to this Camp as well as the background and historical legacy of the area is presented.

The last part of this chapter deals with extent of TB in Macassar by presenting the data of TB incidence and TB mortality in Macassar. An overview of TB in the food factory, where the majority of the Camp residents were employed is also presented. Finally this chapter concludes by a describing the history of TB in Macassar Camp.

CHAPTER 5

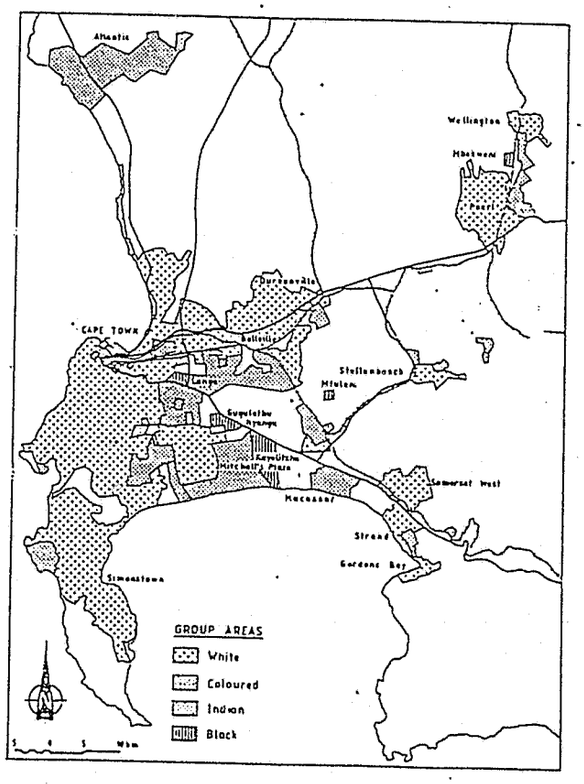
EPIDEMIOLOGY OF TUBERCULOSIS IN THE WESTERN CAPE AND MACASSAR

1 The WESTERN CAPE HEALTH REGION (WCHR)

1.1 DESCRIPTION OF WCHR

Figure 24

Ethnic Distribution of Western Cape Based on the Group Areas Act



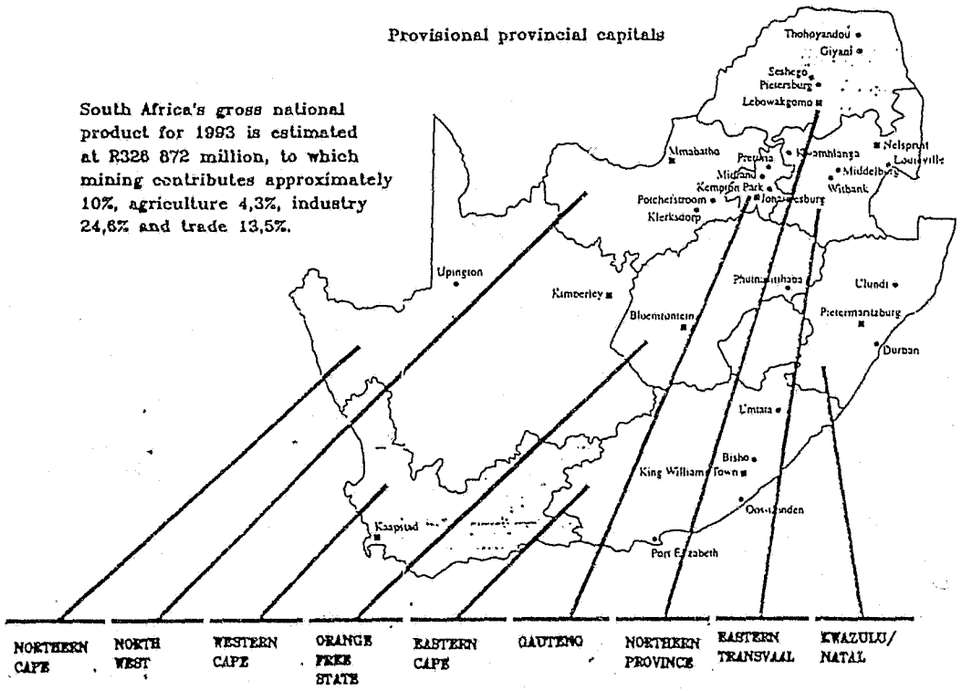
Source:

Designed by Department of Geography, UWC

Figure 24 gives the distribution and locality of the various ethnic groups based on the Group Areas Act (prior to the removal of this act off the statute book in South Africa). The Group Areas Act with all it's implications has had a tremendous impact on disease (especially TB) amongst the various population groups (especially Coloureds and Blacks).[65,66,67,68,69] The Health Regions in figure 25a are demarcated boundaries which came into effect after 27 April 1994 elections.

Figure 25a

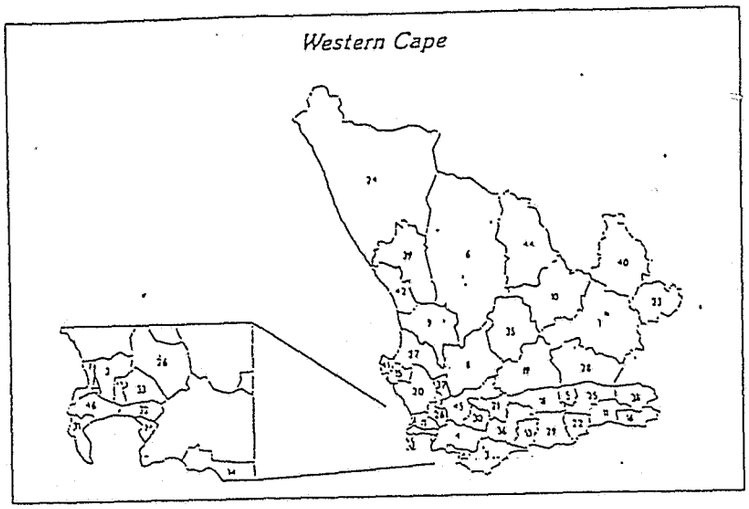
The Health Regions of South Africa



Source: Appendix E1

Figure 25b

Western Cape (Development Region A)



Source:

Epidemiological Comments April, 1992; 19 (4)

The total estimated population for the Western Cape Region in 1992 based on the 1985 census is tabulated in table 8 (Appendix E2):

Table 8

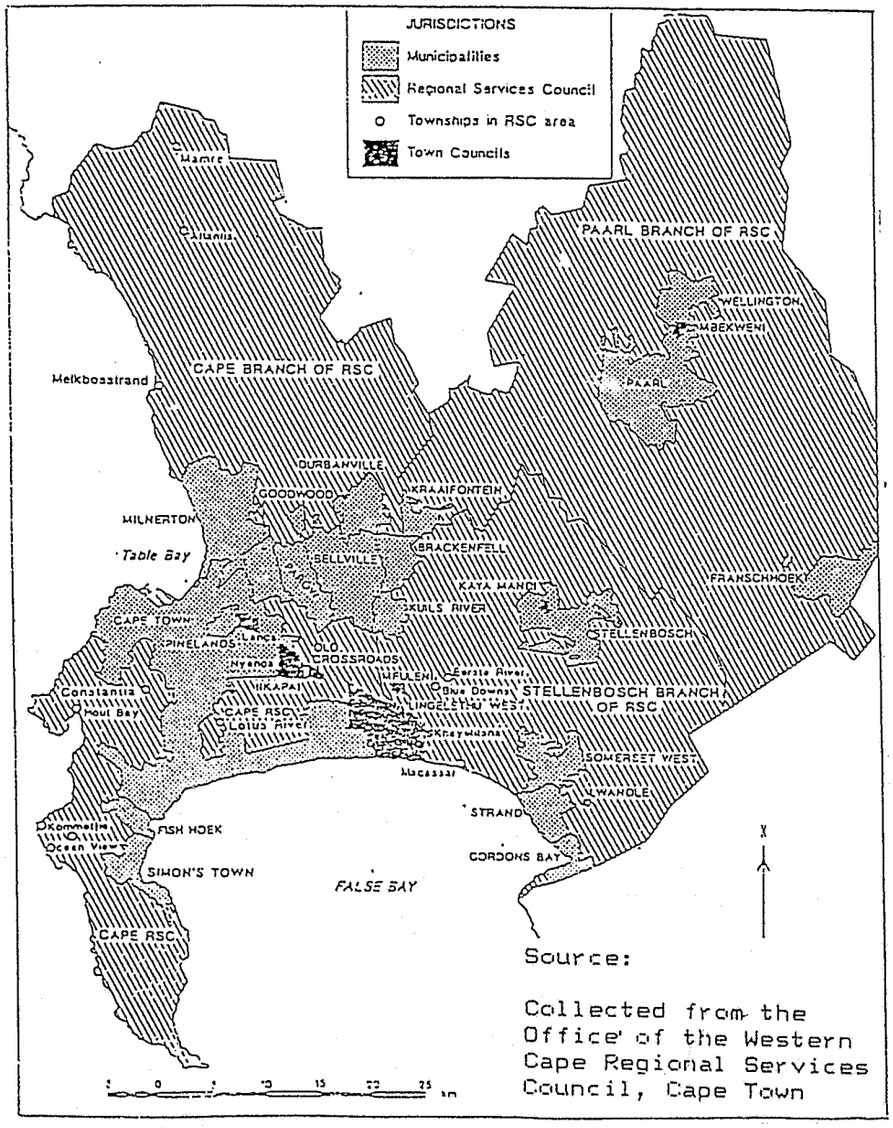
Estimated Population - 1992 in the Western Cape (Development Region A)

Ethnic Group	Estimated Population	Percentage of Total
Asian	22 660	0,6
Black	528 523	14,8
Coloured	2 124 928	59,5
White	892 241	25,0
Total	3 568 352	100,0

It was decided to continue to maintain, for the purposes of this study, the defined health regions (refer to figure 26), prior to the recent changes in the demarcation of Health Regions.

Figure 26

Local Authority Jurisdictions in the Western Cape Regional Services Council



1.2 THE WARNING SIGNS OF AN IMPENDING TB EPIDEMIC

The Western Cape Region has since 1975, experienced a rise, in the number of new cases and an overall increase in the TB notification rates.[1,2] The increase had been noted especially among the Coloured population in this region when compared with the notable downward trend in the rest of South Africa.[3] The alarm bells of a pending TB epidemic were already rung as far back as 1983. The Orange Free State and the Western Cape Health Regions at the time had the highest TB notification rates in the country especially among the Coloured population.[4] During the period of 1973 to 1986, the percentage of all TB notifications under 15 years of age, was 26,5% in the Western Cape as compared to 20,7% for all 7 health regions in South Africa.[5]

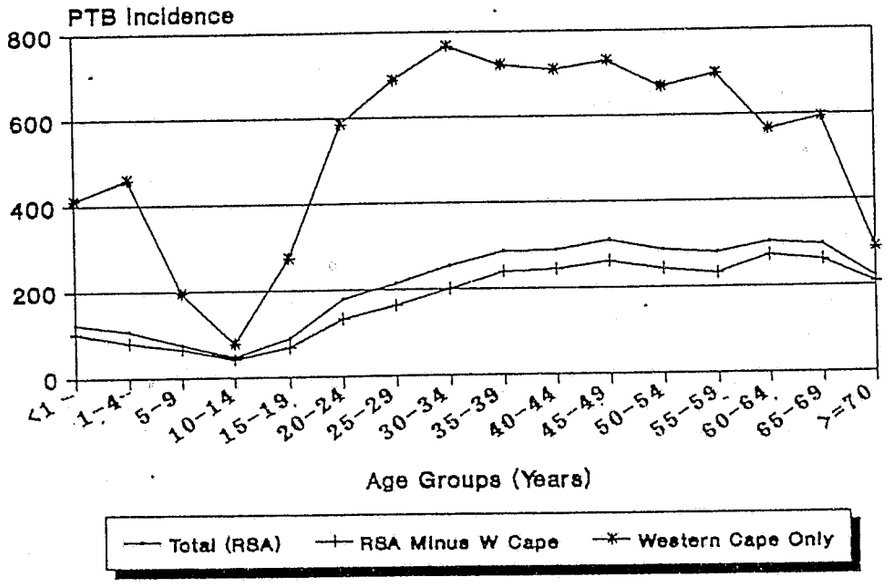
On reviewing the overall TB notification rates in 1987 with respect to the 7 Health Regions and by setting the TB rate for Northern Transvaal as 1, it was shown that the Western Cape was (highest of 7 Health Regions), 12,5 times higher than Northern Transvaal.[5]

By 1988, the Western Cape had the highest percentage of TB patients attending the clinic

with the lowest hospital percentage intake as compared to the other health regions in the country.[6]

Figure 27

Incidence of PTB in the Western Cape Versus the rest of South Africa in 1989



Source:

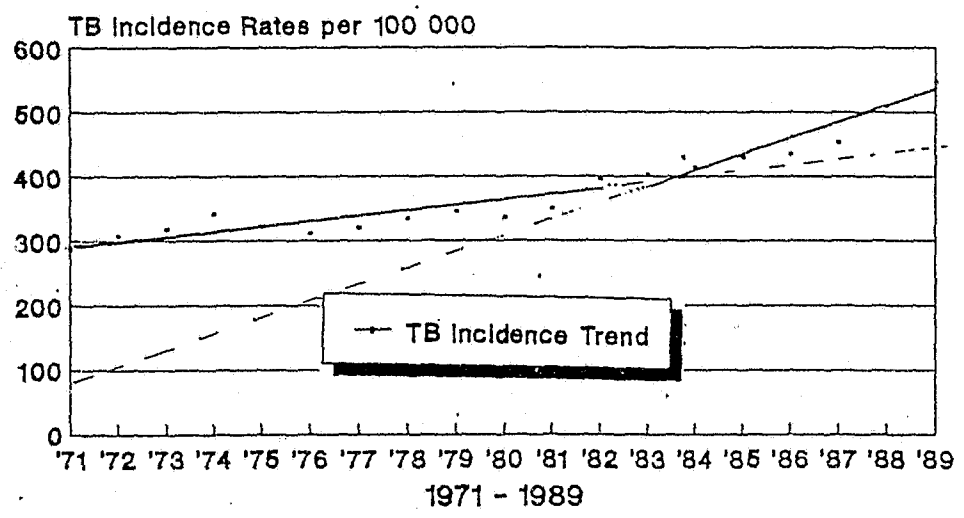
Epidemiological Comments (January, 1991. 18(1):22

By 1989, the TB incidence rates (all forms) for South Africa had reached 186,7 per 100 000 population and 547 per 100 000 for the Coloured population of South Africa.[7] Refer to figure 27. From 1971-1981, there had been a gradual increase in the incidence of TB in the Coloured Population

of South Africa. By 1983, the incidence rate was 402 and from then onwards a steep increase commenced. Figure 28 illustrates the TB incidence trend during 1971-1989, affirming this point.

Figure 28

TB Incidence Rates (All Forms) The Trend in the Coloured Population



Source

Epidemiological Comments Jan 1991. 18(1):17

1.3 POSSIBLE REASONS FOR TB EPIDEMIC IN WESTERN CAPE

1.3.1 Annual Rate of Infection (ARI)

Studies in the Western Cape in the early 1980's demonstrated that the annual rate of infection (ARI) among the Coloureds was between 0,5-1% and for Blacks 2,4% per year.[8] However, annual rate

of change over a time period showed that the ARI for the Blacks had decreased by 7% per year whereas for the Coloureds it remained static during that same time period.

1.3.2 Reduction in TB Hospital Beds

In an attempt to cut cost the Department of National Health in Pretoria, reduced the number of hospital beds during the period 1975-1980. This policy resulted in the bed ratio for TB treatment of 1/25 as compared to the national average for TB bed ratio of 1/5.[9] According to Yach,[3] this action probably contributed to the increase in the number of infectious cases in the community. Gale[10] as far back as 1943, makes mention that insufficient beds could be a problem in the control of TB. However, it should be born in mind that in 1943 hospitalization was all that could be offered to a TB patient.

It was ironic that in 1987 in the Western Cape, which had the highest incidence of TB (table 9) (510/100 000 population), had the lowest percentage (5%) of TB patients hospitalized and thus when compared to the other Health Regions, spent the least on each case (R518,00). As can be noted from the table 9, the overall (all 7 Health

Regions) TB incidence rate during the same period was 269/100 000 with an expenditure of R1 068,00 per case with 23% TB patients hospitalized. In short, the expense per TB case in the Western Cape was less than 50% of the overall TB expense per case of the 7 Health Regions. However, one has to bear in mind (when comparing expenditure per TB case in the 7 health regions), that there is a large rural population in the other areas where hospitalisation may have been required because of the long distances travelled by TB patients.

Table 9

TB Rate, Hospitalisation Rates & Expenditure per case for RSA Health Regions, 1987

Region	Incidence rate*	Expenditure per case (R)	Percent hospitalised
Western Cape	510	518	5
Northern Cape	351	1693	9
Eastern Cape	389	1168	25
Orange Free State	177	808	20
Natal	287	1692	53
Southern Tvl	193	1174	27
Northern Tvl	31	1491	45
Overall	269	1068	23

*per 100 000 population, from TB control programme data

Source

Yach, D; Metcalf, C (1989). The Epidemiology of TB in South Africa. UCT Summer School Presentation, 1 Feb.,1989.

1.3.3 Case Finding/Holding

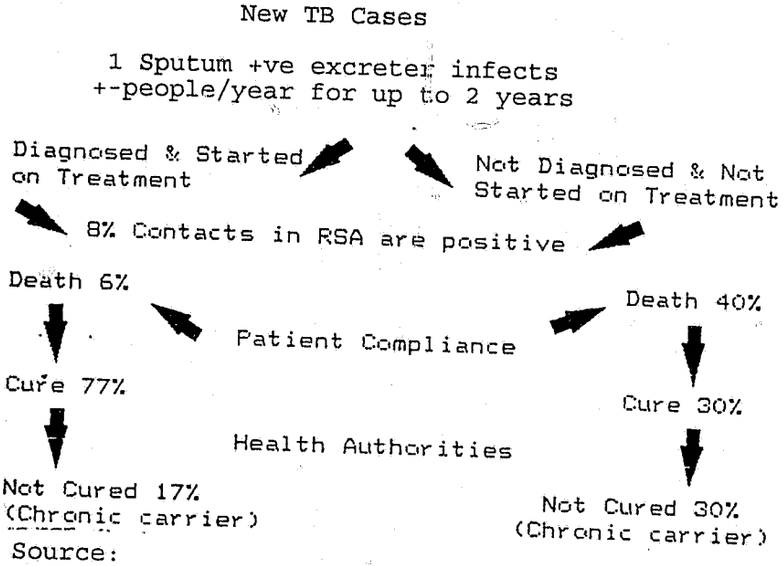
According to Gluckman,[11] the management of TB should be via health centers available at every community center which should have an integrated TB programme including facilities for rapid diagnosis and treatment. This view currently, concurs with the World Health Organizations policy. Perhaps the problems encountered in case holding have been best elucidated by Buch's case history of TB in one rural district.[12] "On the basis of the current data we estimate that there are nearly 8 000 children in Sub A and Sub B in Mhala who required TB care. As we do not have the resources to tackle the massive task, we tried the alternative of arranging supervision of care through teachers. Unfortunately it was impractical, 138 of the 338 children at one school needed care. The teachers were very willing, but spent more than two hours a day supervising care instead of teaching. The support we had to give to keep this effort going could also not be achieved again. We therefore had to decide that we will only provide TB care for about 15% of the children needing it."

Of the new TB cases that have been diagnosed and started on treatment, it has been estimated that

17% did not complete their treatment and may become chronic carriers as compared to the 30% of the chronic carriers of new TB cases that have not started on treatment.[5] Without early diagnosis and with no treatment, a TB patient who is sputum smear positive may infect approximately 20 people in a two year period and a proportion of those infected contacts will in turn infect others should they develop sputum smear positive TB. Even worse, such an untreated person could have a 40% chance of dying from the disease (figure 29a).

Figure 29a

Outcome of Treated Versus Not Treated TB Cases



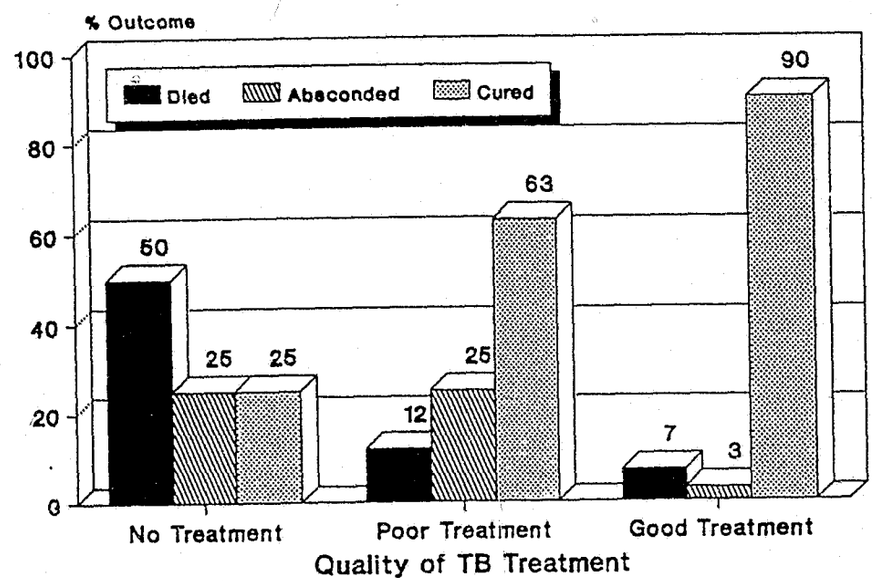
Source:

Yach, D; Metcalf, C (1989). The Epidemiology of TB in South Africa. UCT Summer School Presentation, 1 February, 1989.

As can be noted early diagnosis and treatment of TB is vital in the control of this disease. However, another vital aspect to TB treatment is the quality of treatment. Good quality TB treatment could further increase the TB cure rate to about 90% of TB patients with reduction of TB death to about 5% and a not cure/chronic rate to about 3%. Refer to figure 29b.

Figure 29b

Outcome of New TB Case, Based on the Quality of Treatment



Source:

Strebel PM & Seager JR (1991), Chapter 4 A Century of Tuberculosis, South African Perspective, (Ed) Coovadia AM & Benatar SA; Oxford University Press, Cape Town.

The indicator of health service access for passive case finding can be based on the number of curative services available to the community. [3] From 1984-1985, the number of TB sessions in the Cape Town Municipality dropped by 2,1% with a total drop of 12,5% in attendance (table 10).

Table 10

TB services in Cape Town for 1984 & 1985

Service	1984	1985	% Change
Curative			
Number of sessions	1.324	1.296	−2,1
New consultations	22 309	19 958	−10,5
Total attendances	86.619	75 785	−12,5
Screening			
Mass mini X-ray for migrants (Langa)			
persons screened	9.644	6 641	−10,4
recall for exam	599 (6,2)	180 (2.1%)	−70,0
new cases active TB	63	33 .	−47,6
Mass mini X-ray (Chapel Street)	24 884	17 088	−31,3
Preventive: BCG vaccination			
White	6.424	5.836	−9,2
Coloured	36 065	29.098	−19,3
Black	6 000	4.978	−17,0

Source:

Fourie, PB. Tuberculosis prevalence survey by random sample. Tuberculosis Research Institute. Annual Report, 1985/86.

Data from prevalence surveys have been used in determining the efficacy of contact tracing. Table 11 covers a period of 11 years in areas such as KwaZulu, Transkei, Ciskei, Lesotho and border region of Eastern Cape. The total yield

ranged from 4,8% for smear positive adults to 1,6% for contacts of culture positive adults.

Table 11

Case yields (cases found/persons screened) X100 for contact tracing from various types of index cases under optimized conditions

INDEX CASE		NO. SCREENED		CASES FOUND				CASE YIELD (PER CENT)		
TYPE	AGE	INDEX	CONTACTS	MX+ CHILD	C+ ADULT	S+ ADULT	C+S+ ADULT	TOTAL	PER FAMILY	PER CONTACT
Child										
Mx+	<15 yr	176	1 093	16	6	0	8	30	17,0	2,7
Mx+	<5 yr	129	648	16	5	0	5	26	20,2	4,0
Healthy	<15 yr	100	758	1	1	0	0	2	2,0	0,3
Adult										
C+	>15 yr	87	442	20	1	0	0	21	24,1	4,8
S+	>15 yr	26	124	2	0	0	0	2	7,7	1,6
C+S+	>15 yr	68	411	17	1	1	0	19	27,9	4,6
Healthy	>15 yr	100	722	1	0	0	1	2	2,0	0,3

Mx+ Mantoux reaction ±14 mm
C+ Culture positive
S+ Direct microscopy. Smear positive
C+S+ Culture and smear positive

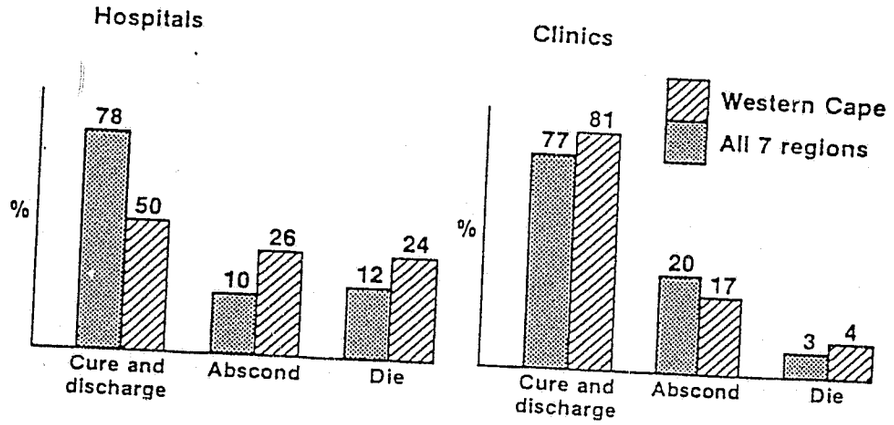
Source:

Seager, JR. Is active case-finding an effective TB control measure? S Afr J Sci. 1986; 82,389.

Contact tracing and passive case finding according Seager, were found to be the most efficient way to find cases.[13] However, in the WCHR, especially in the Cape Town area contact tracing became difficult since 1986 due to township violence. As of May 1986, there appeared to be a decrease in notifications. This decrease has been attributed to the township violence (disruption of health care services) and its long term impact has as yet not been fully determined.[14]

Figure 29c

Treatment Outcomes of TB Cases in the 7 Health Regions of RSA, 1986



Source:

Tuberculosis Control Programme - 1987. Epidemiological Comments 1988; 15:20-36

Figure 29c illustrates TB treatment in hospitals and clinics. The Western Cape has a 50% cure rate compared to 78% of the overall cure rate of all 7 Health regions. In addition there were 26% patients absconding in the TB hospitals in the Western Cape as compared to the absconding rate of 10%, the overall rate of all 7 Health regions. The implication of this poor cure rate in the Western Cape may have resulted in the increased number of tubercle excreters in the community. However, it should be noted that only the most difficult TB cases are hospitalised in the Western Cape. This

may explain the poor cure rates in hospitals in the Western Cape.

1.3.4 TB Compliance

Benatar[15] as well as Aquinas and Todd[16] have suggested that the TB Control Programme in the developing countries should be nationwide and that this can only be achieved by means of an infrastructure provided by an overall general health service which will deal effectively with other common diseases such as malnutrition, malaria and other parasitic infections.

The recommendations by the Department of National Health and Population Development with regard to improving the health service provided in order to enhance supervisory ambulatory care (or "buddy system") and TB compliance (TB adherence) are as follows:[15]

* courtesy - creating a good first impression and maintaining this through friendliness and encouragement
* communicativeness - letting the patient know what is expected and allowing two way communication
* continuity - reinforcement of clear messages through repetition

* consistency - follow up by the same staff member if at all possible
* client orientation - listening to clients (patients) feelings about the diagnosis and the problems it may cause; discussing barriers to compliance and helping the patient overcome this
* clear contractual arrangements - preferably in writing with explicitly stated expectations on both sides
* community orientation - eliciting social support for those patients with impaired access to the tuberculosis services
* convenience - short clinic waiting times and an appointment system if possible;
* contact maintenance - follow-up of non-attendance as soon as possible;
* caring relationship - develop empathy with the patient
* cleanliness in the clinic as a mark of respect for patients and for the service being offered.

Unfortunately, these recommendations have not been fully implemented. However, lack of compliance to fully implement the National Guidelines by the local authorities is believed not to have been the

reasons for the rise in the TB rate in the Western Cape.[17] This study with regards to TB compliance within the local authorities of the Western Cape regarding the use of the National Regimens, showed that uniformity existed with respect to diagnostic procedures, treatment of adults and management of contacts. However, it was noted that the overall management of children and notification criteria varied between the local authorities.

Although a study showed an overall compliance rate for the Western Cape Health Region to be 82,5% in 1984, the lowest rates were shown in children under 5 years, teenagers, Blacks and the unemployed.[18] According to Yach,[3] 35% of the TB admissions in the Brooklyn Chest hospital in Cape Town are retreatment cases and/or defaulters.

1.3.5 BCG Vaccination Coverage

Even though BCG's, role is controversial and the epidemiological impact minimal BCG does nevertheless, protect the infant and should reduce the incidence of severe forms of the disease such as TB meningitis and miliary TB.[19,20] Protection lasts for about 5 years.[21] South African studies have demonstrated that about 65% protective effect can be attributed to BCG against

active TB in children under 5 years in an urban setting.[22]

1.3.6 Nutritional Status

According to Dannenberg[23] and Hopewell,[24] poor nutrition is associated with increased incidence of TB disease. Animal studies have shown that the susceptibility to TB increases with extreme malnutrition.[25] The reason for this susceptibility is probably a decrease in the T-lymphocytes particularly the CD4+ helper cells and depressed phagocytosis thus causing the host to be susceptible to TB.

Table 12

Deprivation of Nutrients versus Immunity

Deprivation of Nutrients	Mechanism
Protein deficiency	skin anergy, decrease in the lymphoid tissue, decrease in the number & function of the T lymphocytes, and decrease in CD4+ helper cells
Iron deficiency	associated with the depressed function of phagocytosis
Vitamin C & D deficiency	associated with the depressed macrophage function
Zinc deficiency	reduced lymphocyte function

Source: Appendix E3

According to Coovadia[26] protein-energy-malnutrition and TB are social diseases par excellence which are deeply rooted in impoverishment, overcrowding and unhygienic conditions which form the basis of the social substrate of malnourishment. Coovadia summarizes the effects of malnutrition and TB as follows:[26] "In summary, the combination of early primary infection due to overcrowding and poverty and immunodeficiencies caused by malnutrition and repeated childhood infections increases exposure and decreases both protective host responses to TB and hypersensitivity reactions."

In the Western Cape Health Region there is a paucity of data on the nutritional status of children. However, a study by Whittaker,[27] showed that 41% of Coloured pre-school children were underweight. Another study in Khayelitsha showed 15% of children were below the third percentile weight for age.[28].

Mets[29] showed, that 27 (5,2%) of 520 workers that were treated at work during 1973-1979, had relapsed within 5 years:

- 8 during the first 2 years
- 12 during the 3rd and 4th years
- 7 during the 4th and 5th years

Mets attributed these relapses to alcohol abuse, concomitant malnutrition and stresses of working life.

The Report of National Health Commission of 1942-1944, the Gluckman Commission, made mention of the deficiencies in housing, nutrition and wage levels and recommended the formation of a National Health Service.[30] In 1944, a similar set of evidence was placed in before the Natal Judicial Commission by the Friends of the Sick Association (FOSA) which recommended the following:[31]

(i) Nutrition - subsidization of essential and protective foodstuffs by the government;

(ii) Housing - the community should be adequately housed with consideration being given to locality, security of tenure, construction materials, adequacy of light, size and number of rooms, sanitary and cooking arrangements.

1.3.7 Overcrowding

Marais describes an experiment performed by Trudeau[32] which demonstrated the effect of overcrowding and other environmental factors which modified the individual's susceptibility to TB. Ten rabbits were inoculated with an identical dose of the tubercle bacilli. Five of the rabbits were

set free and the remaining rabbits were confined to sunless, damp conditions and were fed on a poor diet. The rabbits under these confined conditions died whereas the unconfined rabbits when captured showed signs of healing.

Such experiments are not ethically possible in humans, but they demonstrate the possible influence of unplanned rapid urbanization leading to overcrowding and thus promoting TB infection and disease.

From 1976 to 1986 the African population in the Western Cape increased from +-200 000 to +-750 000 mainly due to influx from the homelands (Transkei and Ciskei), where it is believed that the rates of infection were higher than in urban Cape Town. [3]

This rapid increase in the population of Cape Town resulted in overcrowded Black townships, a problem further compounded by the acute housing shortage in South Africa amongst Blacks, Coloureds and Indians.

The overcrowding in townships due to the acute housing shortage is believed to be linked to a higher rate of TB. [35]

Table 13a

Housing Shortages in South Africa, 1988

RACE	GOVERNMENT ESTIMATES	URBAN FOUNDATION ESTIMATES
Coloured	100 000	—
Indian	48 747	800 000
Black:	—	—
White designated areas	702 750	—
Non-independent homelands	185 578	892 000
'Independent' homelands	125 150	125 000
Total	1 162 225	1 817 000

Source:

South African Institute for Race Relations Survey, (1989).

The household contacts of active TB cases are at the highest risk of being infected with the tubercle bacilli, since the level of infectiousness is linked to the following:[33,34]

* closeness of the contact
* duration of the exposure
* tubercle load in the sputum

The overall housing shortage in South Africa (including the 'homelands') was estimated to be in the range of 1,1 to 1,8 million units.[36] A report in 1985 from the Council for Scientific and Industrial Research stated that on average there were 13 Blacks per house in white-designated areas.[37]

Table 13b

Number of People per Habitable Room in Cape Town for Coloureds: 1970 and 1980

Suburb	1970	1980	Direction of change
Athlone	1,4	1,6	+
Bonteheuwel	1,4	2,1	+
Claremont	1,8	0,6	-
Facreton	1,5	2,2	+
Heideveld	1,5	2,0	+
Kensington	1,6	1,5	-
Manenberg	1,4	2,2	+
Retreat	1,6	1,9	+
Schotschekloof	1,6	1,9	+
Wynberg	1,2	1,3	+
Woodstock	1,4	1,0	-
Kewtown	1,2	2,0	+

Source:

Yach, D. Tuberculosis in the Western Cape Health Region of South Africa. Soc Sci Med. (1988); 27 (7): 683-689.

Thus, among the factors that have contributed to the TB epidemic in the Western Cape, especially among the Coloured and Black populations, were the rapid urbanization, poor housing and overcrowding.[38]

1.3.8 Occupational risk

History has already adequately demonstrated the impact of an economic recession on the TB epidemic. The first decade of this century saw depression in the mining industry which caused a .....

ripple effect throughout the country resulting in a generalized economic recession and causing a significant increase in the unemployment rate.

At the same time the rural areas were feeling the impact of the drought, the effects of the Anglo Boer War and the rinderpest epidemic which wiped out about 90% of African cattle.

This eventually led many of the rural population to migrate to the urban areas resulting in the development of slum areas. It was about at this time that the TB mortality rates increased in Johannesburg and several port cities such as Cape Town, Port Elizabeth and Durban.[39]

Furthermore, evidence suggests that the increased incidence of TB at the time of the drought, could be linked to the simultaneous increase in hospital admissions in Ciskei in 1919-20 and 1929-30.[40]

By mid 1988, there were 1.5 million registered unemployed people with the racial distribution set out in table 14.

The actual number of those unemployed is higher in the country since these figures reflect only registered unemployed people in South Africa.

Table 14

Percentage 'Employable' People In Age Group 15 - 65 years (1988)

Ethnic Group	% 'Employable'	% Unemployed (Registered)
Blacks	56,5	83,0
Coloureds	63,8	12,0
Indians	68,5	12,3
Whites	65,2	10,2

Sources:

(i) Department of National Health and Population Development. (1989). Epidemiological Comments 16 (11) Pretoria. Government Printer.

(ii) Central Statistical Services (1989). South African Labour Statistics 1989 and RSA Statistics in Brief 1989. Pretoria. Government Printer.

Lalloo and Mets[41] regard unemployment a serious health risk not only to the worker but also their dependents. Levi,[42] believes that if the worker is fit and his job is bad and if the worker is unable to cope and lacks social support he could succumb to potentially pathogenic reactions. The WHO,[43] makes the distinction between what are regarded as 'occupational' and 'work related diseases.' In this context, work related TB would be, TB contracted in the course of employment where the infecting agent is not inherently

present but it is rather because the worker works under stressful, poor environmental conditions and lives at a low socioeconomic level.

Lalloo and Mets aptly sum up this point:[41] "It is clear that excessive psychological job stress, malnutrition, socioeconomic gradients and destructive lifestyles, together with adverse environmental work conditions are all factors which increase the risk of TB as a work related disease for certain categories of workers in South Africa."

Stewart[44] mentions that it was E L Collis who concluded that the working conditions might affect the incidence of TB in the following ways:

* undermining the resistance to disease (poverty and alcoholism)
* by direct damage to lung tissue (silicosis)
* increasing risk of infection (overcrowding at work). In South Africa, the potentially aggravating factors (Appendix E4) which play a role in causing work related diseases are further exacerbated by Labour Policy.

Although, 'contact with TB' is listed as a prescribed disease under the Social Security Act of 1975 in the United Kingdom, no such disease

(PTB) is included in the Workmen's Compensation Act in South Africa.[40] According to Abrahams, these aggravating factors causing work related diseases are:[45]

* work conditions (environmental hazards, hours of work, excessive physical and psychological stresses)
* host susceptibility (genetic and by virtue of socioeconomic factors that influence nutritional status, educational level, cultural aspects and fatigue)
* community conditions (such as environmental pollution, housing, urbanization and industrialization)
* lifestyle behavioral factors such as smoking and alcohol abuse.

Kitnasamy and Yach[46] found that TB incidence rates varied in different categories of industry and occupations. Rates also varied between the racial categories: for Blacks it was 899; for Coloureds 537; and for Indians 438 and Whites it was 19 per 100 000. And it was noted that the rates were highest in companies which had higher proportions of labourers. Table 15 illustrates crude risk ratios for TB by occupation.

Table 15

Crude Risk Ratios for TB by Occupational/ Industrial Category for Black and Coloured Workers in South Africa.

YEAR	INDUSTRIAL CATEGORY	REGION	TB RATES/ 100 000†	RISK RATIO
1987	Gold miners	S.Tvl	500:(281)	1,8
		O.F.S	500:(205)	2,4
1986	Black foundry workers	Cape	3 700:(976)	3,8
1985	Black brick workers	Cape	1 800:(840)	2,1
1987	Coloured canning workers	Boland	706:(751)	0,94
	Black canning workers	Boland	1 288:(1 070)	1,2
	Food/canning workers	Cape	611:(681)	0,9
1981	Food industry	Cape	770:(443)	1,7
	Textile		440:(443)	0,99
1982	Engineering		400:(443)	0,9
	Paper/printing		370:(443)	0,83
1987	Textile	W.Cape	829:(718)	1,15
	Iron and steel		766:(754)	1,02
	Local authority		643:(790)	0,81
	Food/canning		611:(778)	0,79
	Building/construction		521:(695)	0,75
	Chemical		418:(754)	0,55
	Printing/paper		317:(693)	0,46
	Transport		292:(768)	0,38
	Trade and commerce		229:(717)	0,32
	Overall		472:(609)	0,78

†Rates in brackets are for, as far as possible, comparable groups of the general population in the local area

Source:

Lalloo, UG & Mets, JT, (1991). Tuberculosis, Workers and occupations in South Africa. A Century of Tuberculosis, South African Perspective, (Ed) Coovadia, AM; Benatar, SR, Oxford University Press, Cape Town (1991).

The South African Tuberculosis Association (SANTA) in 1985 made the following plea to employers not to dismiss their worker on grounds of TB alone: "The patient who is diagnosed reasonably early and who is under correct treatment will be rendered non-infectious almost at once. He is usually fit for work, and should be allowed to continue working to support his family, who are obviously at special risk because they were exposed to infection before he was diagnosed."

2.0 TB TRENDS IN AREAS OF WESTERN CAPE HEALTH REGIONS

2.1 TB INCIDENCE IN ARBITRARY DEMARCATED AREAS (WCHR)

The Western Cape Health Region (WCHR) as noted in the map in figure 26, was divided into various arbitrary areas, in order to determine area of highest incidence of TB in the Western Cape.

The TB incidence was calculated for these various areas based on the data in Appendix E5a and E5b. Figure 30 displays a graph of TB incidence for these respective arbitrary divisions. These arbitrary divisions within the areas of all health centers that were within the jurisdiction of the local authority (figure 26) of the Western Cape Regional Services Council (WCHR) were compared (PTB incidence) to the following:

(a) Western Region (W/RGN): Comprising of the Cape Branch of RSC and Cape Town Municipality

(b) Stellenbosch Area (Stelb): Comprising of the Stellenbosch Branch of RSC, Stellenbosch Municipality, Kraaifontein Municipality, Brackenfell Municipality and Kuils River Municipality

(c) Somerset West Area (SW/STR/GB): Comprising of the Somerset West Municipality, Strand Municipality and Gordons Bay Municipality

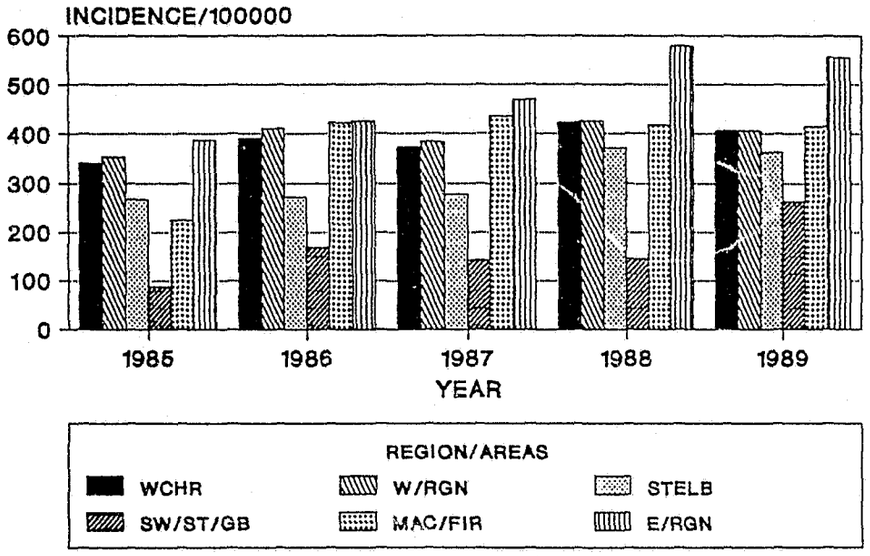
(d) Macassar/Firgrove (Mac/Fir): Comprising of the Macassar and Firgrove (Clinic)

(e) Eastern Region (E/RGN): Comprising of the Paarl Branch of RSC, Municipality, Paarl Municipality and Wellington Municipality

2.1.1 Macassar PTB Incidence Exceeds WCHR

Figure 30

Comparisons of PTB Incidence in WCHR & Its Areas



Source: Appendix E5a and E5b

Figure 30, illustrates that, although incidence of PTB in W/RGN exceeded that of the WCHR in 1985, 1986, 1987 and 1988, the highest incidence of PTB 1985, 1986, 1987, 1988 & 1989), when compared to all other arbitrary regions, was in the Eastern

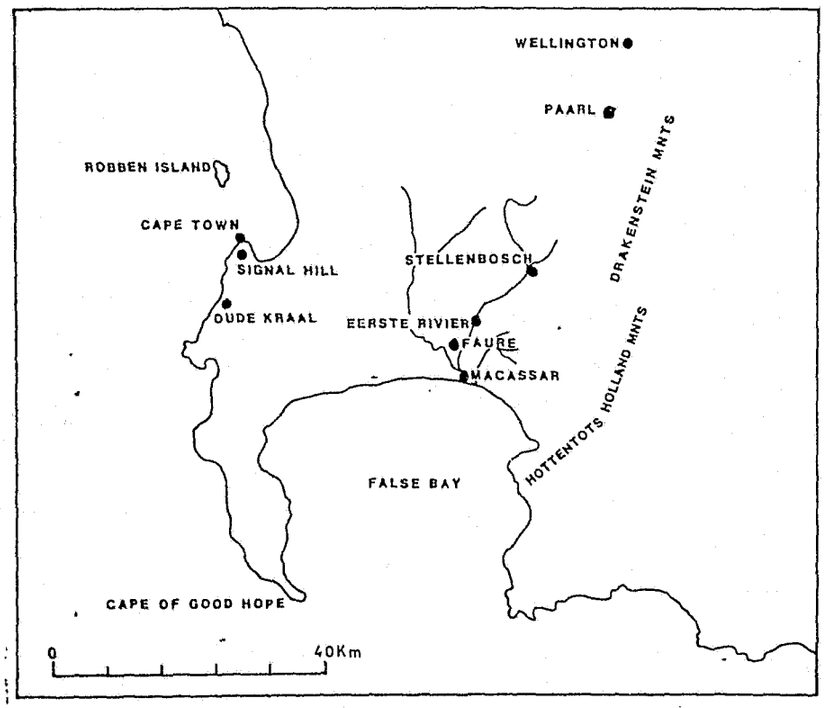
Region (E/RGN), which was comprised of the Paarl Branch of RSC, Paarl Municipality and Wellington Municipality. Macassar/Firgrove (Mac/Fir) was the area with the second highest incidence of PTB (1986, 1988, & 1989).

2.2 MACASSAR

2.2.1 Description of Area of Study

The figure 31 is a map of the Cape Peninsula illustrating the position of Macassar.

Figure 31
Map of the Cape Peninsula



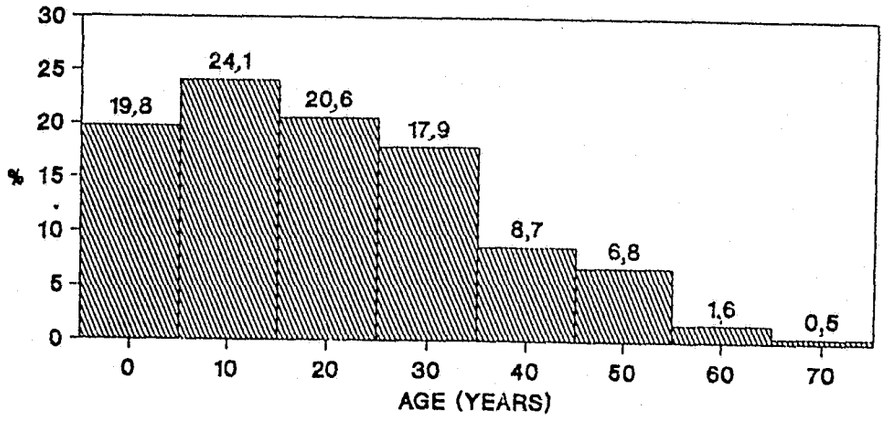
Macassar is situated on the False Bay Coast (in the Cape), about 40 km from Cape Town. Macassar and Firgrove (a suburb of Macassar), had a population of 33 917 (Appendix E5b) in 1990 comprised mainly of the Coloured community. The health facilities are a Day Hospital and a clinic. In addition a Mobile Health Clinic serves the community in Firgrove and Macassar Camp. Recently a home for the aged was built.

2.2.3 Macassar Camp

More than 50% of the Macassar Camp population is in the age group of 19 to 49 years (figure 32)

Figure 32

Population of Macassar Camp Age Distribution



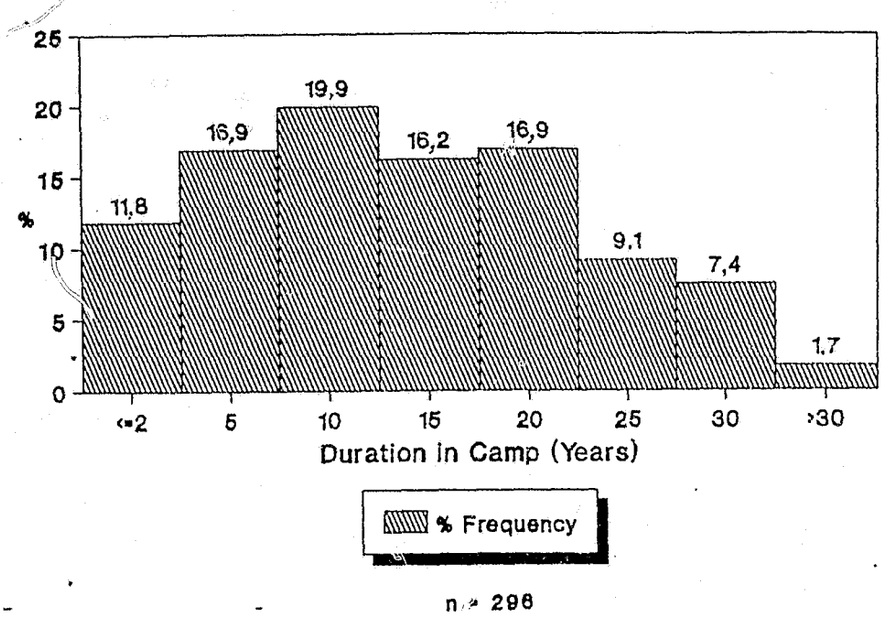
N = 369

Macassar Camp is within the residential area of Macassar/Firgrove (situated on the False Bay coast about 40 km from Cape Town). The Camp comprises of 63 dwelling units and a population of 369.

The Camp is about 1 km from the Food Company that owns this area. The Health facilities in the Camp are a private Dental practice and a weekly Mobile Health Clinic. Other facilities are a mini-hall which runs weekly film shows, a church, a sports field, a community hall and a local shop leased out by the Food Company.

Figure 33

Number of Years Residing In Macassar Camp



2.2.4 Background to Population in MacassarCamp/Macassar

Macassar is named after a Ujung Pandang in Indonesia, which was formally known as Makasar. Shaykh Yusuf, a prince whose birthplace was Goa in India, had a great reputation for learning and established himself with his family in the Court of Sultan Ageng (1651-1683) in Bantam (Indonesia). It is here that he instituted the first center of learning.[47,48,49] He became related to the Royal House of Bantam by marriage to the daughter of Sultan Ageng.[50] He later was honoured with the title of 'Shaykh Yusuf al-Taj al-Khalwati al-Maqasari' (The crown of the Khalwati Order of Makasar).[51]

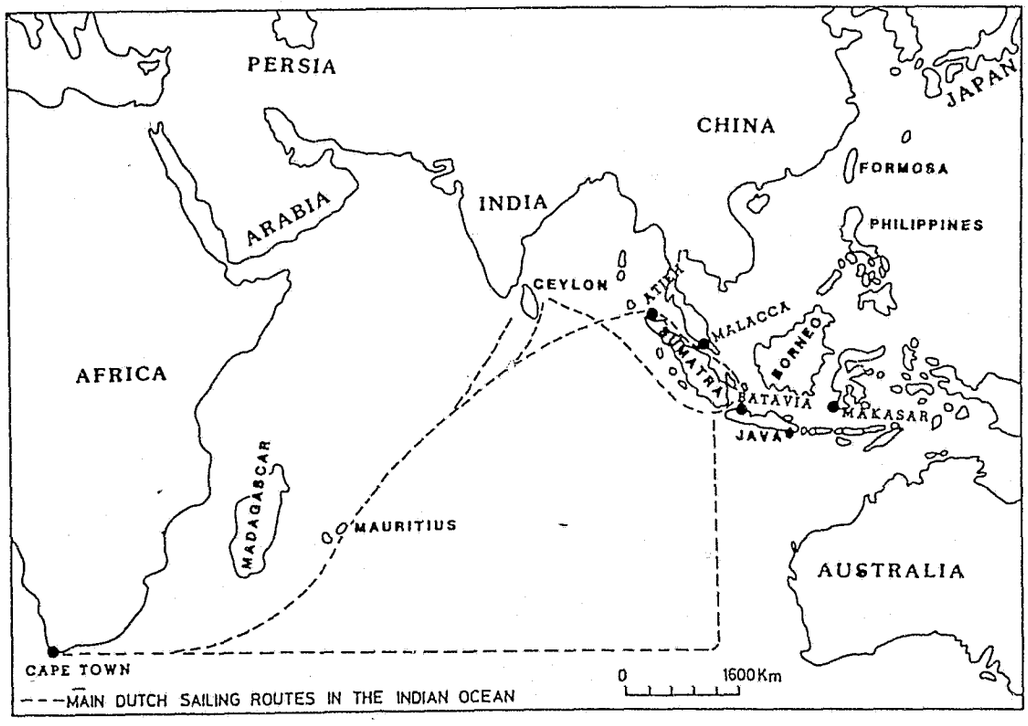
Due to his resistance and opposition to the Dutch East Company he was forced into political exile, firstly to Batavia and then Ceylon and finally the Cape.[52] He and his retinue of 49 (including his family), were brought to the shores of the Cape by the Dutch East Company, on board the ship named the 'De Voetboeg' on 2 April 1694.[47] Refer to figure 34a depicting the map of the Dutch East Company's sailing route during the 17th and 18th centuries.

They were permitted to reside on a farm named

Zandvliet which was then owned by a minister of the Dutch Reformed Church, situated on the mouth of the Eerste River.[53,54] Because of their political and religious convictions they were not allowed to make contact with other political exiles or slaves.[55]

Figure 34a

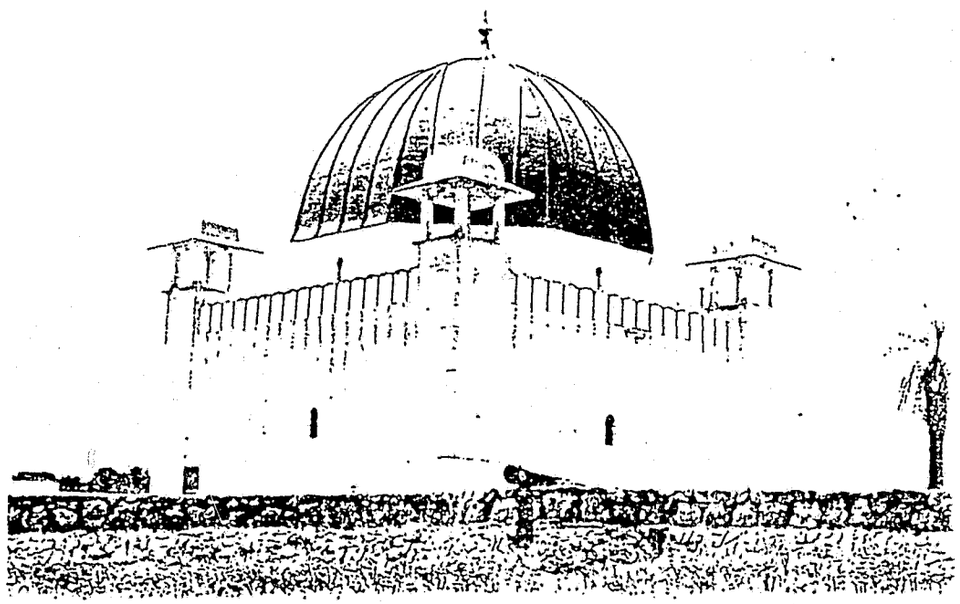
Dutch East India Company's Sailing Routes, (17th - 18th Centuries).



Cassiem[70] in his inimitable style summed up the history of these exiles: "If the struggle of the oppressed people (in South Africa) has a history then the Muslims are at the focal point of that history. They were enslaved and in exile whilst they were on board the ships of the conquerors. They arrived in chains (to the shores of South Africa) whilst the indigenous Africans were still to be chained." Figure 34b depicts the tomb of Shaykh Yusuf in Macassar as testimony to this fact.

Figure 34b

The Tomb of Shaykh Yusuf in Macassar



Today, the district surrounding Zandvliet is known as Macassar (because many of his followers hailed from Makasar). The tomb of Shaykh Yusuf in all its majestic grandeur still overlooks Macassar as a perpetual reminder of the rich historical legacy of this place. Refer to figure 34b.

Unfortunately, there are no records of prevalence of tuberculosis amongst this group or those in the surrounding area at the time. However, the first recorded TB patient in South Africa was Paul Da Gama, the brother of Vasco Da Gama.[56]

3.0 EXTENT OF TB IN MACASSAR

3.1 TB INCIDENCE IN MACASSAR

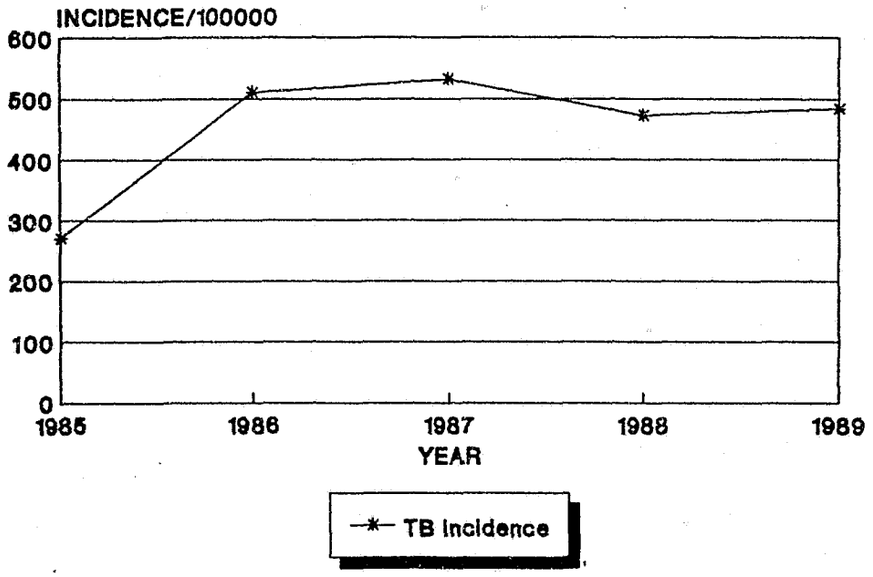
The notification rate of TB (all forms) in Macassar, 485,2 per 100 000 in 1989, was the second highest rate in a Coloured area in the WCHR. The TB notification rate in Elsies River in 1989 was 799 per 100 000 population.[57] In 1989 the PTB rate in the SW/STR/GB area increased. This could possibly be attributed to the increase in the formal and the informal settlements in Sir Lowry's Pass (near Somerset West).

According to the Medical Officer Health (MOH) of the Stellenbosch RSC Branch in 1988:[58] "Lately

the squatter problem has arisen and awaits ministerial decision for its solution. The conditions are truly horrendous and deteriorate by the day." Figure 35 illustrate the incidence of all forms of TB incidence in Macassar Camp during the period of 1985-1989.

Figure 35

TB (All Forms) Incidence In Macassar



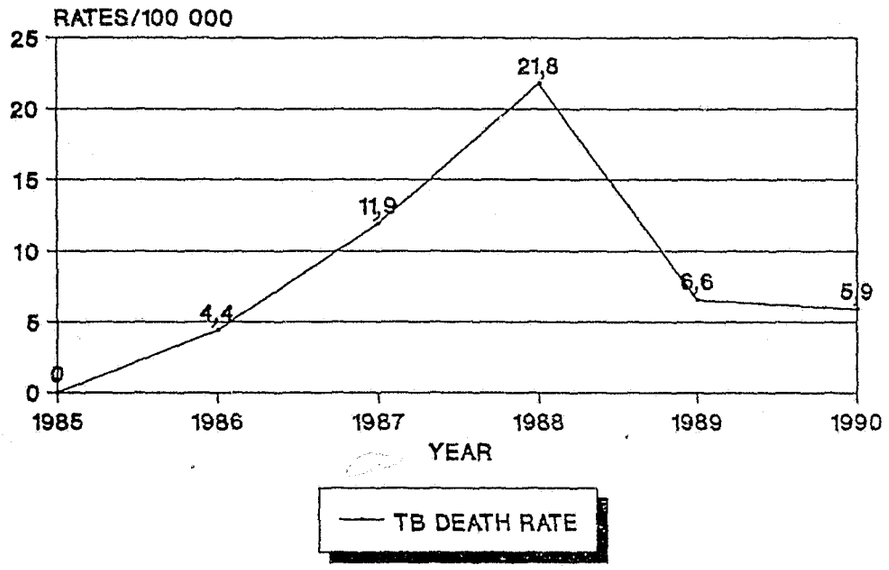
Source: Appendix E6

3.2 TB MORTALITY IN MACASSAR

The death rate due to TB in Macassar in 1988 was 21,82 per 100 000 population. Refer to figure 36 and Appendix E6.

Figure 36

TB Death Rates in Macassar



Data collected from Macassar Clinic

Source: Appendix E6

3.2.1 Overview of TB in Macassar Food Factory

A survey of the use of the First Aid Room in 1986, in the factory gave an insight into the history of TB amongst the factory workers.[59] However, not all workers were residents of Macassar Camp. Neither did this survey cover all the residents in Macassar Camp. Refer to tables 16, 17 and 18.

Table 16

Record of Past TB cases amongst the Food Factory Workers in Macassar

Patient Number	Sex	Date of Report	Employment Status
1	F	??/05/81	(Permanent Staff)
2	F	02/09/81	(Permanent Staff)
3	F	15/09/81	(Permanent Staff)
4	F	18/03/82	(Permanent Staff)
5	M	14/04/82	(Permanent Staff)
6	F	27/05/82	(Temporary Staff)
7	F	01/06/82	(Temporary Staff)
8	M	25/10/82	(Permanent Staff)
9	F	03/09/83	(Temporary Staff)
10	F	04/10/83	(Temporary Staff)
11	M	11/04/84	(Temporary Staff)
12	F	01/08/84	(Temporary Staff)
13	F	05/09/84	(Temporary Staff)
14	F	??/??/84	(Permanent Staff)
15	F	??/??/84	(Permanent Staff)
16	M	11/07/85	(Permanent Staff)
17	M	07/09/85	(Temporary Staff)
18	M	11/04/86	(Temporary Staff)
19	F	18/07/86	(Temporary Staff)
20	F	18/07/86	(Temporary Staff)
21	F	01/08/86	(Temporary Staff)
22	F	22/08/86	(Temporary Staff)

The mass chest X-ray survey of most of the permanent staff of this factory, was reviewed.[59] The results of the 189 factory workers who were X-rayed (including 8 temporary workers who had requested to be included in the survey), are listed in table 17. However, 1 female and 1 male of the permanent staff of the factory declined to be X-rayed. One worker was confirmed to have a positive chest X-ray. Further tests (smear and

culture positive) confirmed PTB. Table 17 tabulates the results of the mass chest X-rays of the Food Factory Workers prior to the commencement of this study.

Table 17

Results of the Mass Chest X-Ray of Factory Workers in Stellenbosch Clinic

	X-Ray Results		
	+VE	-VE	
MALE	0	91	91
FEMALE	1	97	98
TOTAL	1	188	189

3.2.2 History of TB in Macassar Camp

The Camp prior to the study had 9 cases of confirmed TB (since 1972 and prior to the commencement of this study in 1988), as can be seen from table 18. There was also the death of pensioner in the Camp due to TB in 1986. Records of one of the past TB cases (KCPTB) could not be located. Of the 8 TB cases, the sputum microscopy of 3 (37,5%) cases was heavy (+++) and 2 (25%) cases gave a moderate result (++), indicating the potential for a high level of transmission of *Mycobacterium tuberculosis*. Refer to table 18.

Table 18

Past Pulmonary Tuberculosis (PTB) Cases in Macassar Camp

CASE NUMBER	AGE	SEX	SPUTUM MICROSCOPY	SPUTUM CULTURE	YEAR NOTIFIED
1	42	F	+	POSITIVE	1972
2	23	M	+	POSITIVE	1982
3	32	F	++	POSITIVE	1982
4	28	F	++	POSITIVE	1984
5	50	M	+++	POSITIVE	1986
6	38	M	+++	POSITIVE	1986
7	22	F	+++	POSITIVE	1986
8	33	M	-	POSITIVE	1987

1 TB DEATH OF PENSIONER (MACASSAR CAMP -1986)

Source:

From data of survey, factory records, and data from Stellenbosch and Macassar Clinic records.

There were 9 Camp residents who had a record of TB in the past and records of only 1 PTB case could not be located. Only 1 (12,5%) case in table 18, was sputum microscopy negative, while all 8 cases were confirmed by positive culture.

CHAPTER 6
RESULTS

"The experiment serves two purposes, often independent one from the other: it allows the observation of new facts, hitherto either unsuspected, or not yet well defined; and it determines whether a working hypothesis fits the world of observable facts."

Rene' J Dubos

CHAPTER 6

RESULTS

SUMMARY

This chapter commences with details of the response rates to the various aspects of this study as well as the demographic data of Macassar Camp population. Furthermore it details the distribution of the population on the exposure to varying degrees of the Batsons Overcrowding Index value as well as the overall ventilation of the Camp residents in their respective homes.

A detailed description of the TB ELISA versus Mantoux results which indicate the rate of the TB infection (based on Mantoux test as well as the TB ELISA test), is presented. Results show that there is no correlation between the Mantoux and TB ELISA tests. The Kappa statistic yielded a poor agreement between the two tests.

The sensitivity and specificity of screening tests such X-rays, Mantoux test and TB ELISA test were determined and a comparison was made of the three screening tests.

The health status of the Camp residents such BCG vaccination, history of other diseases, past TB cases and other relevant information were also reviewed. In addition to this a detailed description of the Camp residents smoking habits and alcohol intake is also presented.

The outcome of Mantoux test based on gender and age, PTB household contact, as well as the prevalence of TB infection in the various age groups is presented. In addition the association of risk factors such as poor ventilation, overcrowding, PTB household contact, occupational status, malnutrition, Primary Food Subsistence Level and other relevant data, associated with TB infection as indicated by the results of Mantoux testing was reviewed.

Finally the diagnosis of TB cases using the various screening and diagnostic methods is described to derive a prevalence of PTB in Macassar Camp. This chapter concludes by tabulating the association of TB infection with socioeconomic risk factors established in this study and relating these risk factors to confirmed PTB cases amongst Camp residents.

CHAPTER 6

RESULTS

1 RESPONSE RATE

1.1 RESPONSE TO HOUSEHOLD QUESTIONNAIRE (Appendix F4)

Of the 63 dwelling units (Appendix B1) in Macassar Camp, 60 (95,2%) heads of households responded to the household questionnaire. Heads of households who were not present on the first visit were revisited on two more occasions. If the head of the household was not present on the third visit then this was regarded as a non-response.

1.2 RESPONSE TO PERSONAL QUESTIONNAIRE (Appendix F4)

From the 369 residents in the Macassar Camp detailed in the 60 household questionnaires, 296 (80,2%) camp residents completed the personal questionnaires (including proxies for children). Two hundred and seventy eight (75,3%) of the residents, were 13 years of age or older and were considered eligible to answer the personal questionnaire without a proxy. Of this sub-population (>=13 years), 228 (82,0%) responded.

1.3 RESPONSE TO MANTOUX TESTING AND BLOOD SAMPLING

Mantoux test was performed and blood for the TB ELISA was collected from the 209 (56,6%)

consenting residents (from the 369 residents compiled from the 60 households).

1.4 RESPONSE TO HEIGHT AND WEIGHT MEASUREMENTS

Of the 296 camp residents, 253 (85,5%) were weighed and height was measured.

1.5 RESPONSE TO MASS MINIATURE CHEST RADIOGRAPHS

Of the 369 residents in the Camp, radiographs were taken of 275 (74,5%) residents. The mass miniature chest radiographs were performed in a mobile X-ray unit which was taken to the Camp, work sites and schools. The response rate for mass miniature radiographs would have been higher but for:

- four women who were pregnant
- 21 children who were too short for X-ray unit
- 1 female who was an amputee
- 68 residents who were non-responders

2.0 **DEMOGRAPHY OF MACASSAR CAMP** (Appendix F4)

2.1 DWELLING UNITS OF MACASSAR CAMP

2.1.1 Type Dwelling Unit

The dwelling units in Camp, comprised 56 (93,3%) houses and 3 (5,0%) hostels. Only 1 (1,7%) dwelling unit was termed as 'other'. The room occupancy rate was 2,5 people per habitable room

for the residents in Camp in 1988. An earlier study (1986) had found an average number of 1,7 Coloureds per habitable room in Cape Town area. [1]

2.1.2 Batsons Overcrowding Index [2,3,16]

Based on Batsons Overcrowding Index, each of the dwelling units in the camp was categorized into four groups (not overcrowded, crowded, overcrowded and highly overcrowded). Table 19, illustrates the frequency distribution of the varying degrees of overcrowding in relation to the dwelling units in Macassar Camp.

Table 19

Extent of Overcrowding in Camp Dwelling Units

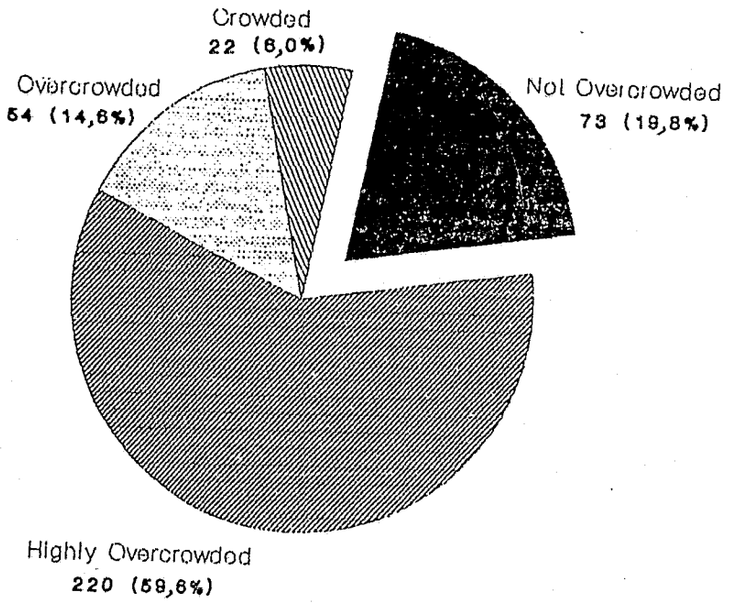
Batson Index	Degree of Crowding	Number of Dwelling Units	%
1	Not Overcrowded	17	28,3
2	Crowded	5	8,3
3	Overcrowded	11	18,3
4	Highly Overcrowded	27	45,0
	TOTAL	60	100

Figure 37 summarizes the extent of exposure to overcrowding (using the Batsons Crowding Index). Figure 37, shows that 220 (60%) of the Camp residents were in the category highly overcrowded

and only 73 (20%) residents were in the not overcrowded category.

Figure 37

Effect of Distribution of Population On Exposure Of Varying Degrees of Overcrowding



2.1.3 Type of Toilets in Dwelling Units

Of the 60 dwelling units surveyed in the Camp, there were 34 (56,7%) toilets inside the dwelling

units, 22 (36,7%) were outside the dwelling units and only 4 (6,7%) dwelling units used a communal toilet situated in the Camp.

2.1.4 Ventilation of Dwelling Units [4]

The overall household ventilation was expressed as the percentage of the total openable window area to that of the total floor area (excluding kitchen and toilet/bathroom) of the dwelling unit).
Table 20 documents ventilation of dwelling units, indicating that little more than 1 in every 4 of the dwelling units (26,7%) in Macassar Camp had an overall household ventilation of less than 5%.

Table 20

Extent Of Overall Ventilation For The Individual Dwelling Units In Macassar Camp

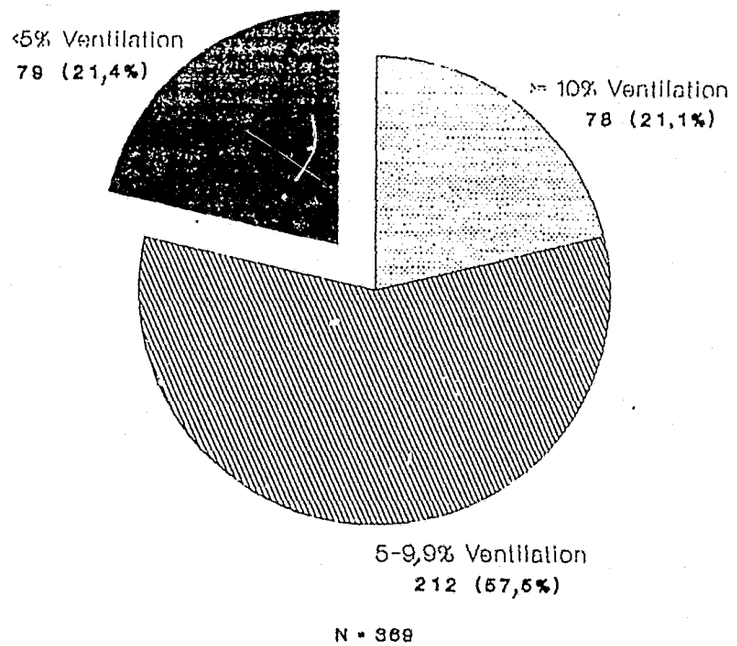
Overall Household Ventilation	Category Of Overall Household Ventilation	Number of Dwelling Units	%
<5%	Below Required Regulation	16	26,7
5 - 9,9%	Above Required Regulation	31	51,7
>=10%	Well Above Regulation	13	21,7
	TOTAL	60	100

Figure 38, illustrates the extent of the overall household ventilation exposure of the Camp

residents, in which 79 (21,4%) residents were exposed to the overall household ventilation of less than 5% in their respective homes. Thus 1 in 5 of the Camp residents was subjected to an overall household ventilation of less than 5% (below the required ventilation) of the total openable window to floor area of the respective dwelling unit.

Figure 38

Population Exposure of Overall Ventilation of Macassar Camp Residents' Respective Homes



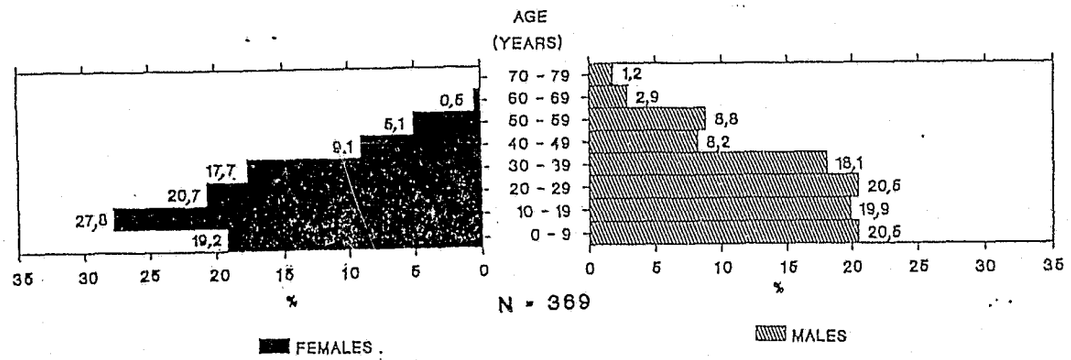
2.2 MACASSAR CAMP POPULATION

2.2.1 Age Sex Distribution

The age and sex distribution of Macassar Camp residents is illustrated in figure 39.

Figure 39

Age and Sex Distribution of the Macassar Camp Population



2.2.2 Ethnic Distribution

The proportional distribution of the ethnic groups in table 21 is not an accurate reflection of the South African population. Since the study was undertaken on the entire population of 369 Macassar Camp residents, the frequency inside some ethnic groups was too small and hence stratified

analysis was not possible. Thus the data was analyzed on the entire group without differentiating the ethnicity of the Camp residents.

Table 21

Ethnic Distribution Of Macassar Camp Residents

Ethnic Groups	Frequency	%
Blacks	58	15,7
Coloureds	305	82,4
Indians	5	1,4
Whites	1	0,3
TOTAL	369	100

2.2.3 Marital Status

In tables 22 and 23 the marital and occupational status of the Camp residents (aged 15 years or older) are respectively displayed. Table 22, illustrates that 94 (45,9%) residents were married whereas 89 (43,4%) residents were single.

Although 21 (10,2%) residents (as can be noted from table 23) were unemployed, that 35 (19,3%) residents (excluding housewives) were unemployed among the 181 residents of the 'employable group' in the Camp (comprising of the employed, unemployed and housewives).

On exclusion of the housewives from the 'employable group', the estimated unemployment rate then becomes 12,6%.

Table 22

Frequency Distribution Of Marital Status of Macassar Camp Residents (aged 15 years or older)

Status	Number	%
Married	94	45,9
Divorced	3	1,5
Separated	4	1,9
Widow/Widower	7	3,4
Living Together	8	3,9
Single	89	43,4
TOTAL	205	100,0

2.2.4 Occupational Status

Table 23

Frequency Distribution of Occupational Status of Macassar Camp Residents (above age 15 years)

Occupational Status	Number of Residents	%
Housewife	14	6,8
Student	21	10,2
Employed	146	71,2
Unemployed	21	10,2
Retired	3	1,5
TOTAL	205	100,0

Thus, the 'true' unemployment rate in Macassar Camp is 12,6% when one considers the 167 residents (comprising of the 146 employed and 21 unemployed residents), to be the productive workforce. Of the 146 residents that were employed, 106 (72,6%) were employed by the factory that owned Macassar Camp. There were 113 (77,4%) residents that were employed on a permanent basis, whereas only 29 (19,9%), were employed on a temporary/seasonal basis. Four (2,7%) were employed on a part-time basis. Employed residents of Macassar Camp were grouped into various work categories based on their position held at the company and their job description. Ninety five (65,1%) of the employed residents were unskilled or partly skilled (semi-skilled).[5] Refer to table 24.

Table 24

Employment Status of Camp Residents

Category	Type	Frequency	%
I	Professional	2	1,4
II	Intermediate	9	6,2
IIIn	Skilled Non-Manual	32	21,9
IIIm	Skilled Manual	8	5,5
IV	Partly Skilled	32	21,9
V	Unskilled	63	43,2
	TOTAL	146	100

From table 25, compared to skilled workers a significant (p = 0,001) proportion of the unskilled workers lived below the Primary Food Subsistence Level (PFSL). The relative risk was 1,6 with 95% confidence interval of 1,16-2,33.

Table 25

Relationship of Unskilled Worker and Primary Food Subsistence Level (PFSL)

Employment Category	Primary Food Subsistence Level (PFSL) Below	Above	Total
Unskilled	69(72,6%)	26 (27,4%)	95(100%)
Employment Skilled	23(45,1%)	28(54,9%)	51(100%)
Total	92(63,0%)	54(37,0%)	146(100%)

3.0 **TB ELISA VERSUS MANTOUX RESULTS** (Appendix F2 & F4)

3.1 TB INFECTION RATE BASED ON MANTOUX VERSUS ELISA

As can be noted from table 26, the total significantly positive Mantoux tests was 67 (32,1%) compared to the 19 (9,1%) total positive TB ELISA tests. There were two cut-off points for a positive Mantoux result (one for children and one for adults as mentioned in Chapter 2), whereas the TB ELISA test had only one.

Table 26

Mantoux versus TB ELISA Test Results of Macassar Camp Residents

		MANTOUX RESULT		
		+VE	-VE	TOTAL
TB ELISA RESULT	+VE	8 (a)	12 (b)	20 (9,6%) (n1)
	-VE	59 (c)	130 (d)	189 (90,4%) (n2)
	TOTAL	67 (32,1%) (n3)	142 (67,9%) (n4)	209 (n)

3.1.1 Relationship between the Mantoux and TB ELISA Tests Results

In only 8 (11,9%) of the residents tested was, the TB ELISA also positive when the Mantoux was positive. If the Mantoux was negative, then the TB ELISA was positive in 12 (8,5%) residents. The similar proportions suggests that the TB ELISA test and Mantoux test are not related. The TB ELISA is positive in approximately 1/10 of the residents tested. When the TB ELISA was positive then the Mantoux test also was positive in only 8 (40,0%) of the residents. However, when the TB ELISA was negative the Mantoux test was positive in 59 (31,2%) of the residents tested.

3.2 AGREEMENT BETWEEN MANTOUX AND TB ELISA RESULTS

In order to determine if there is reproducibility of the results obtained with regards to the two

methods of screening tests a Kappa Statistic was employed to calculate statistically the margin of reproducibility. [17]

3.2.1 Calculation of Kappa Statistic

The following calculation is based on results in table 26, (calculation of Kappa Statistic described in Chapter 2), displaying the Mantoux versus the TB ELISA tests results of the residents. Refer to Appendix F1 for the calculation formula of the Kappa Statistic (k) based on the data displayed in table 26. From this a value is derived where k = **0,070**, indicating poor agreement. Refer to table 27 for guideline for the evaluation of Kappa Statistic (k).

Table 27

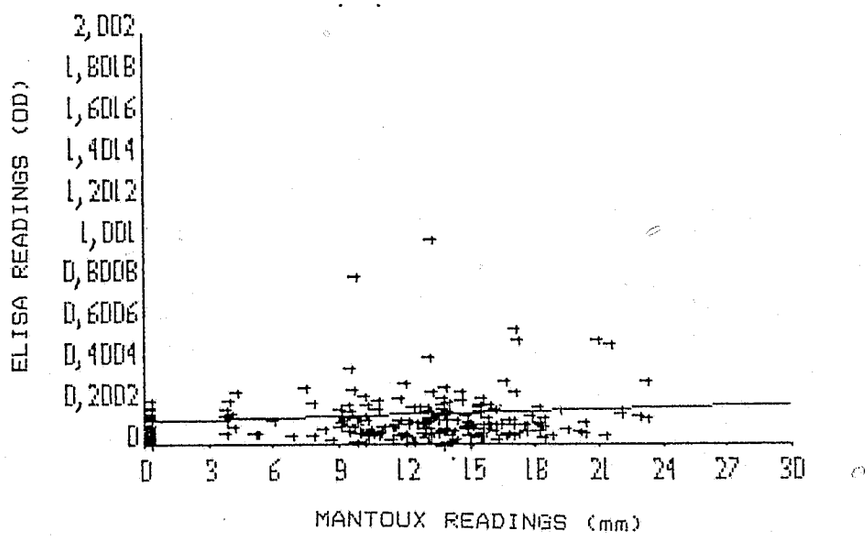
Guidelines for Evaluation of Kappa Statistic

k > 0,75	-	Excellent agreement
0,4 <= k <= 0,75	-	Good agreement
0 <= k < 0,4	-	Poor agreement

Figure 40 further illustrates that there is poor correlation between Mantoux and TB ELISA tests. The correlation coefficient is 0,14 (with 95% confidence limits of 0,1 - 0,27).

Figure 40

Correlation of All Mantoux & TB ELISA Tests of Macassar Camp Residents



)

Correlation coefficient: $r = 0,14$
$r^2 = 0,02$

95% confidence limits: $0,01 < R < 0,27$

Source	df	Sum of Squares	Mean Square	F-statistic
Regression	1	159,3973	159,3973	4,29
Residuals	207	7698,1941	37,1893	
Total	208	7857,5914		

B Coefficients

Variable	Mean	B coefficient	95% confidence Lower	95% confidence Upper	Std Error	Parti F-te
ELISA	0,1322	7,2753049	0,387576	14,163034	3,514147	4,28
Y-Intercept		9,9546657				

Figure 41a

Correlation of Positive Mantoux & TB ELISA tests of Macassar Camp Residents

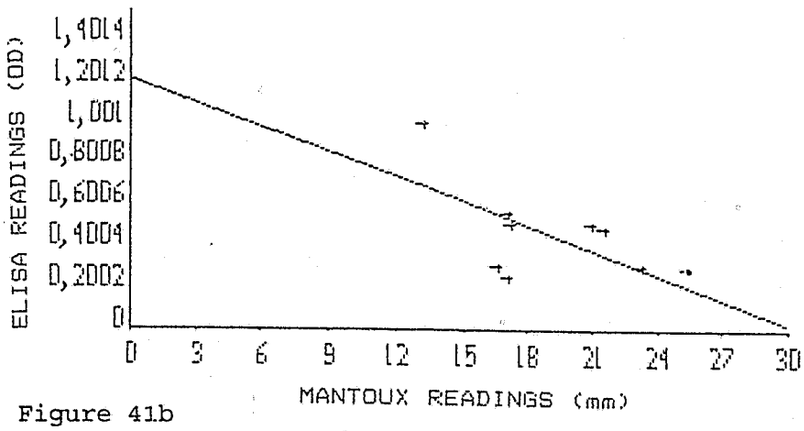
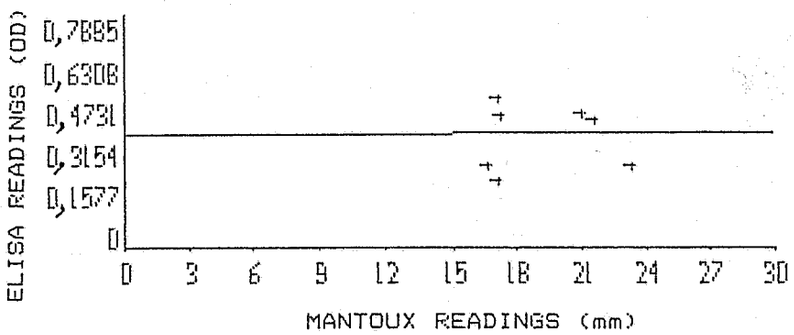


Figure 41b

Correlation of Positive Mantoux & TB ELISA tests of Macassar Camp Residents which excluded the TB ELISA Outlier Reading



The negative correlation coefficient observed in figure 41a (among the Macassar Camp residents who tested both positive for Mantoux and TB ELISA tests), is not observed when the TB ELISA reading which is an outlier (encircled in figure 41a), is exclude in figure 41b. Refer Appendix F5a and Appendix F5b.

3.3 FORMATION OF TB GROUPS/CATEGORIES

All the residents of Macassar Camp, were grouped into the following categories to evaluate the TB ELISA and Mantoux tests.

3.3.1 Newly Diagnosed Pulmonary Tuberculosis Cases

This group was based on residents who had positive sputum microscopy and culture results (n = 6).

3.3.2 Past Pulmonary Tuberculosis Cases (KCPTB) Cases

This group was based on an affirmative response by Macassar Camp residents (n = 8), in the personal questionnaire to having been previously diagnosed and treated for TB. This response was validated by the review of clinical records at the clinic for the results of the radiographs ('TB suspect'), sputum microscopy and culture.

3.3.3 Radiological 'PTB Suspects'

This group was based on those residents that were 'TB suspects', after the results of the mass miniature chest X-ray, totalling to 29 'TB suspects' which comprised of 8 radiologically active PTB (RAPTB) and 21 radiologically suspect (RSPTB), of which 11 residents were excluded (5 residents of which 2 were RAPTB and 3 RSPTB, were

not able to be traced and 6 diagnosed as new PTB case of which one was a relapse (n = 18)).

3.3.4 TB Sign & Symptoms

This group was based on those Macassar Camp residents (n = 20) who responded in an affirmative (in the personal questionnaire) as having two or more signs and symptoms of TB.

One resident who responded in the affirmative to all signs and symptoms of TB was confirmed to be newly PTB case and was excluded from this group.

3.3.5 TB ELISA Negative Control

The negative TB ELISA control population (n = 109) included those residents with a Mantoux negative result. The control population in addition to being Mantoux negative was not previously or newly diagnosed as having TB or regarded as X-ray suspect nor have responded alternatively to two or more signs or symptoms in the affirmative in the personal questionnaire.

3.3.6 Mantoux Positives Control

Of the 369 Camp residents, 209 (56,6%) were tested for TB infection by Mantoux and TB ELISA methods. The cut-off point for a significantly positive

Mantoux test was 10 mm of induration at 72 hours for children aged 0 to 14 years; and 15 mm or more of induration at 72 hours for those aged 15 years or more.[13]

Based on the criterion for cut-off point of Mantoux test, there were 67 (32,1%) residents Mantoux positive. Of these 67 residents who were Mantoux positive, 49 residents were used as positive Mantoux controls (those that did not overlap in the categories defined as: TB cases, past TB cases, TB Suspects and Sign/symptoms.

3.3.7 TB ELISA Cut-off Point

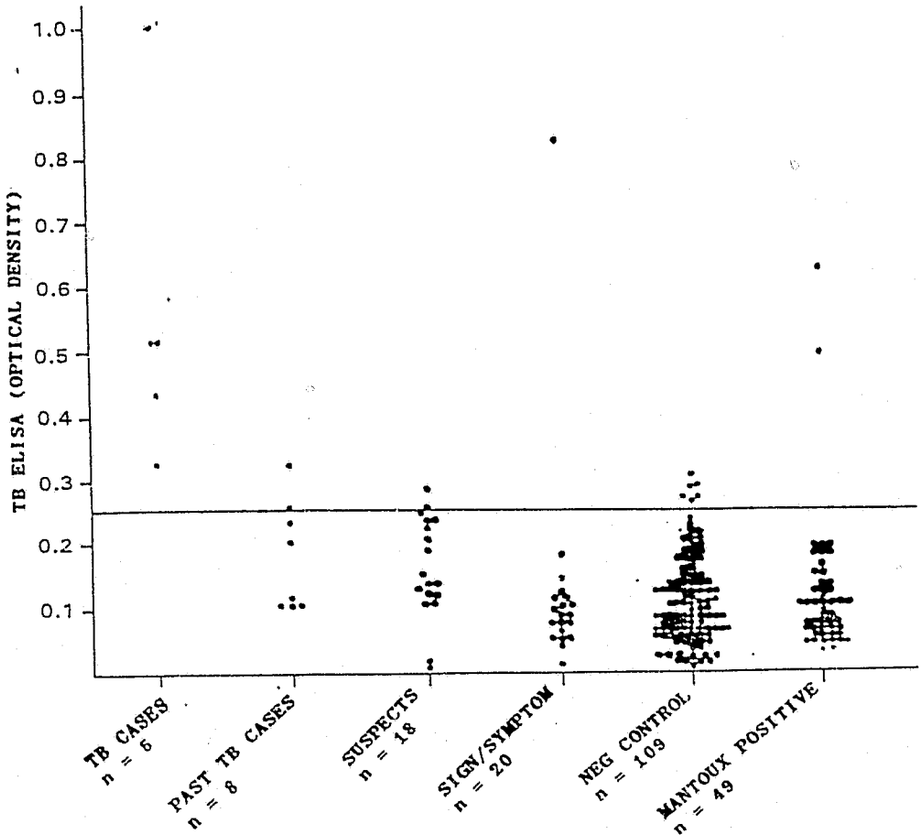
A value above the mean of the control of the TB ELISA optical density (OD) reading (results) plus 2 standard deviations: [(0,111) + (2 x 0,07)] was used to arrive at a positive TB ELISA Test cut-off point of 0,253 optical density.

Thus all readings below this value of OD were regarded as TB ELISA negative and whereas the readings which were >= 0,253 OD were regarded as a TB ELISA positive test.

This resulted in a specificity of 94,5% in the control group of TB ELISA negative controls.[18] Refer to figure 42.

Figure 42

TB ELISA versus TB Categories/Groupings
of Macassar Camp Residents



3.3.8 TB ELISA versus PTB Categories/Groupings

The respective TB ELISA OD readings (optical density) of each of the categories/groupings were plotted (a dot representing a reading for each person tested, n = 209). A horizontal line was drawn from optical density (OD) reading of 0,253 (>= 0,253 OD depicting a positive TB ELISA Test).

Figure 42 illustrates the TB ELISA readings above, on and below the TB ELISA cut-off point.

4.0 SENSITIVITY AND SPECIFICITY OF TB ELISA TESTS

4.1 CRITERION RELATED VALIDITY

The sensitivity and specificity of measurement of an instrument are statistics which assess the criterion of related validity of variables. The "gold standard" is used as a criterion to see how many values were correctly identified. [19]

4.1.1 Sensitivity

Sensitivity (true positive rate), is the proportion of test results out of all true positive tests (such as the number of confirmed PTB cases) that are also TB ELISA positive, divided by the total number of number of positive confirmed PTB cases. Refer to table 28.

4.1.2 Specificity

Specificity (the true negative rate), is the proportion of test results out of all true negative tests (such as the number of negative confirmed not to be PTB cases that are also TB ELISA negative, divided by the total number of negative confirmed not to be PTB cases (able 28).

4.2 SENSITIVITY AND SPECIFICITY OF TB ELISA/PTB CASES

Based on table 28 the sensitivity of TB ELISA tests on newly confirmed PTB cases is 100% at a specificity of 94,5%. However, this result should be noted with caution because the number of newly diagnosed PTB cases is small (n = 5), in a sample of 209 Camp residents. Refer to figure 42 which illustrates the TB ELISA negative controls.

Table 28

TB ELISA Test versus Newly Confirmed PTB Cases in Macassar Camp

		NEWLY CONFIRMED PTB CASES (CULTURE POSITIVE)		
		+VE	**-VE**	**TOTAL**
TB\ELISA	**+VE**	5	6	11
CONTROLS	**-VE**	0	103	103
TOTAL		5	109	114

4.2.1 Sensitivity and Specificity of TB ELISA (OD) versus PTB Categories/Groupings of the Macassar Camp Residents

Table 29 determines the sensitivity (at 94,5% specificity) of the various PTB categories/ groupings of Macassar Camp residents. These sensitivities of the various categories/groupings of PTB of Macassar Camp residents, are based on the results calculated from figure 42.

Table 29

Sensitivity of TB ELISA (at 94,5% specificity) versus the PTB Categories/Groupings

Group	Number	TB ELISA OD*	Sensitivity
PTB	5	0,558 (0,515)	100,0%
KCPTB	8	0,204 (0,197)	25,0%
PTB + KCPTB	13	0,340 (0,142)	53,8%
'PTB Suspects'	18	0,165 (0,146)	11,1%
Sign & Symptom	20	0,123 (0,076)	5,0%
Mantoux Result	49	0,282 (0,092)	4,1%

* TB ELISA OD value reflects: mean and the median is recorded in brackets)

The true positive test results (sensitivity) at 94,5% specificity yields the best results (100%) for newly confirmed PTB cases. The sensitivity decreases (53,8%) if known/past PTB cases (KCPTB) are included in these categories/groupings. The sensitivity for the remaining categories/groupings of PTB are between 4,1-11,1%. Hence TB ELISA cannot be used as the sole diagnostic tool. [18]

5.0 **TB SCREENING TESTS** (Appendix F2)

5.1 MANTOUX AS A SCREENING TEST FOR TB

Table 30a was based on the results of the Mantoux test (Appendix F4) used as a screening test to identify TB cases. Refer to table 30a

Prevalence of TB (table 53)	=	1,355%
Sensitivity	=	60,0%
Specificity	=	68,6%
Predictive Value (PV)	=	2,6%
Positive Predictive Value (PPV)	=	4,5%

Table 30a

Mantoux As A Screening Test To Identify PTB Cases

		PTB CASES (CULTURE POSITIVE)		
		+VE	**-VE**	**TOTAL**
MANTOUX	**+VE**	3	64	67
	-VE	2	140	142
	TOTAL	5	204	209

5.2 TB ELISA AS A SCREENING TEST FOR TB

Table 30b was based on the results of the TB ELISA test (Appendix F4) used as a screening test to identify TB cases (figure 42).

Prevalence of TB (table 53)	=	1,355%
Sensitivity	=	100%
Specificity	=	94,5%
Predictive Value (PV)	=	20,0%
Positive Predictive Value (PPV)	=	45,5%

Table 30b

TB ELISA As A Screening Test To Identify PTB Cases

		PTB CASES (CULTURE POSITIVE)		
		+VE	**-VE**	**TOTAL**
TB ELISA	**+VE**	5	6	11
	-VE	0	103	103
	TOTAL	5	109	114

5.3 X-RAY AS A SCREENING TEST FOR TB

Table 30b was based on the results of the X-ray screening (Appendix F4) used to identify TB cases.

Prevalence of TB (table 53)	=	1,355%
Sensitivity	=	100%
Specificity	=	93,0%
Predictive Value (PV)	=	16,4%
Positive Predictive Value (PPV)	=	25,0%

Table 30c

X-Rays As A Screening Test To Identify PTB Cases

		PTB CASES (CULTURE POSITIVE)		
		+VE	**-VE**	**TOTAL**
X-RAY	**+VE**	6	18	24
	-VE	0	239	239
	TOTAL	6	257	263*

* Results from the mass miniature X-ray in the table 30c excluded the 5 'TB suspects' (unable to be traced), 5 mass miniature X-ray spoilt plates (SP) and 3 diagnosed as other pathology (OP). Thus a total of 24 'TB suspects' based on the mass miniature X-ray result (categorized as: RAPTB = 6 and RSPTB = 18), were X-rayed at the clinic. Refer to table 51, 52 and 53.

5.4 COMPARISON OF THE 3 SCREENING TESTS

Of the 3 the screening tests for TB listed in table 30d, the TB ELISA gave the highest predictive value of 20,0% (identify true positives in a population), as compared to the X-ray with 16,4%, and the Mantoux test with 2,6%. However, in the TB ELISA test 45,5% would yield true positive cases (TB cases), whereas the X-rays would yield 25,0% and the Mantoux test would yield only 4,5%.

Table 30d

Screening Tests Versus Confirmed TB Cases

	TB SCREENING TESTS		
	TB ELISA	MANTOUX	X-RAY
NUMBER	114*	209	265**
PREVALENCE	1,355%	1,355%	1,355%
SENSITIVITY	100%	60,0%	100,0%
SPECIFICITY	94,5%	68,6%	93,0%
PREDICATIVE VALUE	20,0%	2,6%	16,4%
POSITIVE PREDICATIVE VALUE	45,5%	4,5%	25,0%

* Of the 209 residents who volunteered to be tested with the TB ELISA test, the results of a 114 were used for calculation. The remaining of 95 residents were excluded because they were either Mantoux positive, past TB cases, suspected TB cases based on X-rays or sign and symptoms.

** Of the 275 residents who volunteered to be X-rayed, the results for 265 of these residents was used for calculation. The reason for the exclusion of 10 residents was because there were 5 X-ray plates that were spoilt and 5 residents could not be traced for a repeat X-ray.

6.0 HEALTH STATUS OF CAMP RESIDENTS

6.1 BCG STATUS OF CAMP RESIDENTS

A total of 199 of the 296 residents had evidence of BCG vaccination (67,2%). BCG vaccination was confirmed by observation of scar and/or checking the road to health card. There were 48 (16,2%), residents who claimed they were never vaccinated as compared to 49 (16,6%) residents who could recall such an event. Of the 83 children aged 14 years or less, 74 were confirmed to have been vaccinated with BCG (89,2% BCG coverage). This

could have taken place between 1973 to 1989 when BCG vaccination was compulsory in South Africa.

6.2 HISTORY OF OTHER DISEASES IN MACASSAR CAMP

A history of other diseases (table 31) among the Camp residents, could be regarded as an added risk factor for TB.[6] An estimate of past disease and/or illness determined from the personal questionnaire is given in table 31. This table was based on a similar study done by Rieder to determine measure of association with certain risk factors.[22]

Table 31

History of Past Disease/Ilness of Macassar Camp Residents

Disease	Number	%
High Blood Pressure	21	7,1
Asthma/Allergy	19	6,4
Diabetes	3	1,0
Abdominal Surgery	15	6,0
Past Pulmonary TB	9	3,0
No History of Above Disease/s	229	77,4
TOTAL	296	100

Of the 296 Camp residents, 67 (22,6%) had a history of one or more of the diseases/illnesses tabulated in table 31. The majority of the abdominal operations involved a hysterectomy.

6.2.1 Malnutrition

The nutritional status of the Camp residents was evaluated based on the values of height and weight used to determine the Body Mass Index (BMI) for those aged 15 years or older.[7,8,60,61,62]

Whereas children, aged 14 years or less their nutritional status was evaluated with use of standard tables from the National Center for Health Statistics (NCHS)[7,9], with the use of Epi Info and Epi Nut programmes.[63,64] Refer to table 32a and 32b.

Table 32a

Nutritional Status of Macassar Camp Children Aged 0 - 14 Years

Z-Score (SD)	HAZ	WAZ	WHZ
<= -2 SD (Malnourished)	7(10,4%) F=5,M=2	5(7,5%) F=3,M=2	2(3,0%) F=1,M=1
> -2 SD TO -1.5 SD (Borderline)	12(17,9) F=7,M=5	2(3,0%) F=2,M=0	4(6,0%) F=2,M=2
> -1.5 SD (Normal Range)	48(71,6%) F=22,M=26	60(89,5%) F=29,M=31	61(91,0%) F=31,M=30
TOTAL	67(100%)	67(100%)	67(100%)

Table 32a shows, a total of 67 children, there were 7 (10,4%) children below the normal range with regards to Z-score of height for age (HAZ).

Refer to Appendix F3a and F3b for the values calculated for the median, percentile and the Z-Scores for: weight for age (WAZ), weight for weight (WHZ) and height for age (HAZ). Children below -2SD were termed malnourished. Table 32b tabulates the average ages for the respective Body Mass Index (BMI) values calculated for the 5 categories of nutritional status levels for both males and females aged 15 years or older. Refer to Appendix F3a and F3b.

Table 32b

Body Mass Index (BMI) Statistics of Macassar Camp Residents Aged >= 15 Years

MEN	OUTCOME	N	AVERAGE BMI	AVERAGE AGE
<20	Malnourished	28	18,3	31,1
20-24.9	Normal	34	22,3	32,6
25-29.9	Slightly Obese	17	27,1	42,8
30-35	Obese	5	31,3	60,0
>35	Excessively Obese	2	39,4	33,0
WOMEN	OUTCOME	N	AVERAGE BMI	AVERAGE AGE
<19	Malnourished	15	17,7	27,8
19-23.9	Normal	40	20,9	28,5
24-29.9	Slightly Obese	26	26,5	34,8
30-35	Obese	14	32,9	30,9
>35	Excessively Obese	5	41,7	30,4

When the children categorized as malnourished, based on categories of HAZ, WAZ or WHZ, (Z-score cut-off point <-2), were combined with the malnourished adults (BMI values), there was a total of 50 (19,8%) malnourished residents. In the adult group there was a significant difference (p = 0,005), between the number of males 28 (45,2%) and females 15 (27,3%) that were categorized as malnourished as compared to those that were regarded as having a normal nutritional status. Table 32b shows that the nutritional values have no bearing on PFSL values.[10]

6.2.2 Miscarriage

Women eligible to respond to this question were aged >=15 years. Of the 112 women aged 17-63 years who responded, 16 (14,3%) women reported having had one or more miscarriages (table 33).

Table 33

Frequency Distribution of Miscarriages Of Females >= 15 Years

No. Of Miscarriages	Frequency	%
0	96	85,7
1	13	11,6
2	2	1,8
4	1	0,9
TOTAL 7	112	100

6.3 INFANT DEATHS

Fourteen women aged 24-63 years reported that during their reproductive years, a total of 26 deaths had occurred among their infants aged less than one year. This gives an average of 1,9 infant deaths (aged less than one year old) for these women. One women has experienced 8 infant deaths (during her fertile years), before they reached the age of one year.

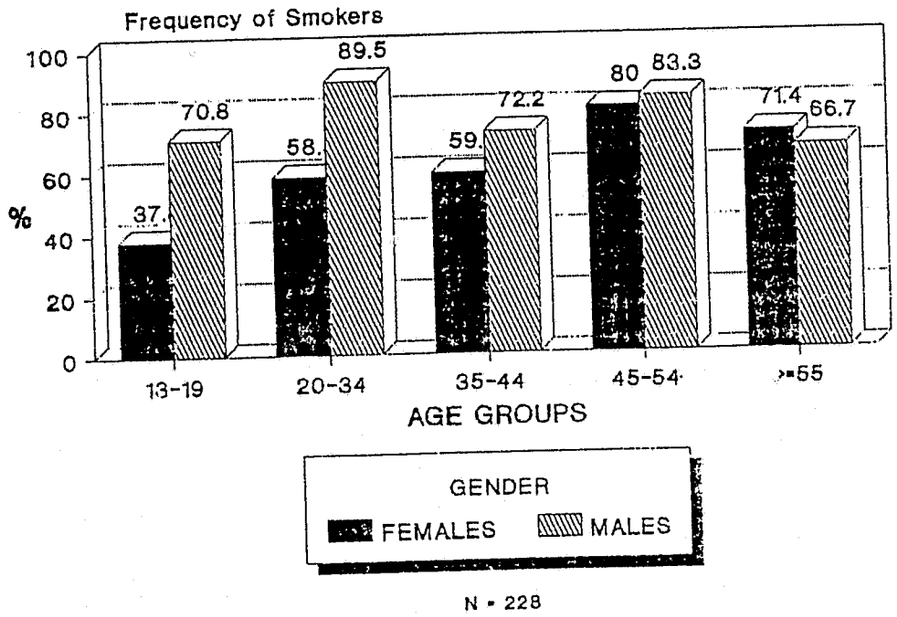
7.0 DESCRIPTION OF CAMP RESIDENTS HABITS ON SMOKING AND ALCOHOL INTAKE

7.1 SMOKING

Of the 278 eligible residents (residents >=13 years), 228 (82,0%) responded to the question regarding smoking, of which 78 (34,2%) were non-smokers.[11] Figure 43 illustrates the age and gender distribution of smokers. The prevalence of smoking amongst males (78,8%) was significantly greater (p = 0,005) than amongst the females (54,8%). Although there was a significantly greater (p = 0,005) number of male than female smokers in the age groups of 13-19 and 20-34 years, there was no significant difference observed between the male and female smokers in the age groups of 35-44, 45-54 and 65 years or older.

Figure 43

Age & Gender Frequency Distribution of Smoking Among Macassar Camp Residents,



N = 228

Not only was there a significant gender difference in the number of smokers but there was also a significant difference between the males and females with regard to the number of cigarettes smoked daily (refer to figure 44). Table 34 summarizes the difference between male and female smokers with regard to heavy and mild smokers.

Figure 44

Frequency Distribution Of Number Of Cigarettes Smoked by Men and Women

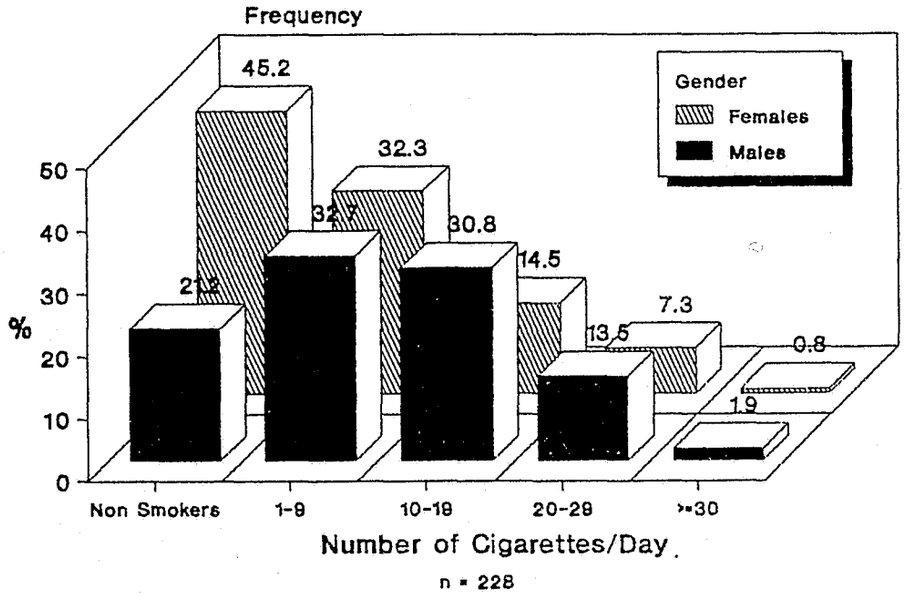


Table 34 shows that 48 (63,2%) males and 28 (36,8%) females were categorized as heavy smokers. The difference was significant (p = 0,034) with a relative risk of 0,68 (95% confidence interval of 0,47 - 0,98).

Table 34

Gender Comparison of Heavy & Mild Smokers

Number of Cigarettes Smoked Daily	Female	Male	Total
>=10	28(36,8%)	48(63,2%)	76(100%)
1-9	40(54,1%)	34(45,9%)	74(100%)
TOTAL	68(45,3%)	82(54,7%)	150(100%)

7.2 ALCOHOL INTAKE

There were 81 (57%) males, who were consumers of alcoholic beverages as compared to 61 (43%) females. This difference was significant with the value p <0,001, with a relative risk of 0,59 (95% confidence interval 0,47 - 0,74 (table 35).

Table 35

Gender Difference in Absolute Alcohol Intake in a 2 Week Period

Absolute Alcohol Intake	Females	Males	Total
Yes	61 (43%)	81 (57%)	142 (100%)
No	63 (73,7%)	23 (26,7%)	86 (100%)
TOTAL	124 (54,4%)	104 (45,6%)	228 (100%)

The heavy drinkers were those residents in the Camp that consumed more than 50 ml of absolute alcohol in a 2 week period on a regular basis.[16] Not only did males consume significantly more alcoholic beverages as compared to the females but there was a significantly greater number of males categorized as heavy drinkers (p = 0,003 with a relative risk of 1,75 and a 95% confidence interval of 1,13 - 2,72 as compared to the females (table 36).

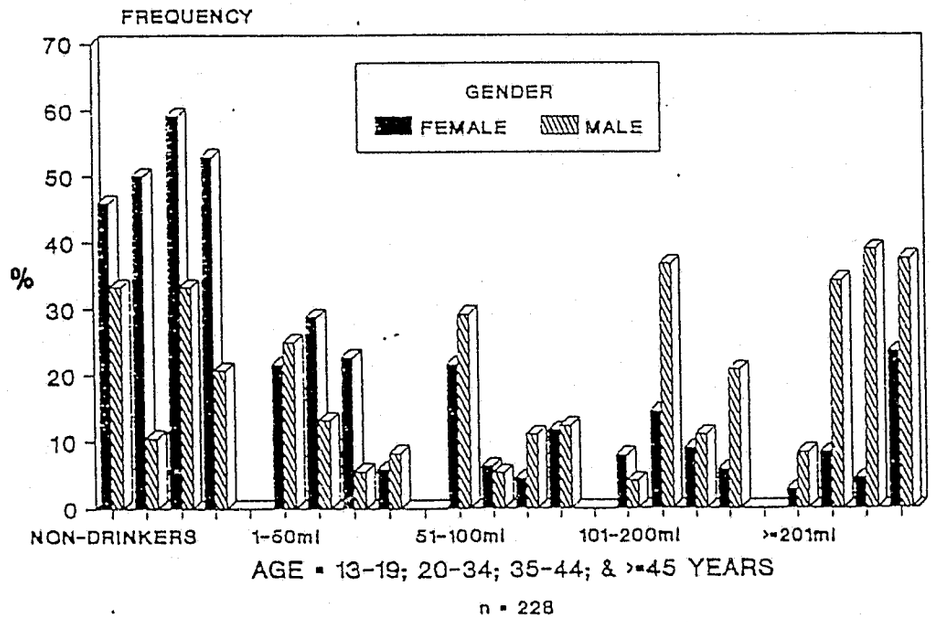
Table 36

Gender Difference of Heavy and Mild Alcoholic Consumers in 2 a Week Period between Males And Females.

Degree Of Alcoholic Beverage Intake	Male	Female	Total
Heavy Drinkers	67 (64,4%)	37 (35,6%)	104 (100%)
Mild Drinkers	14 (36,8)	24 (63,2%)	38 (100%)
TOTAL	81 (57,0)	61 (43,0)	142 (100%)

Figure 45

Age Comparison of Males & Females Intake of Absolute Alcohol In A Two Week Period



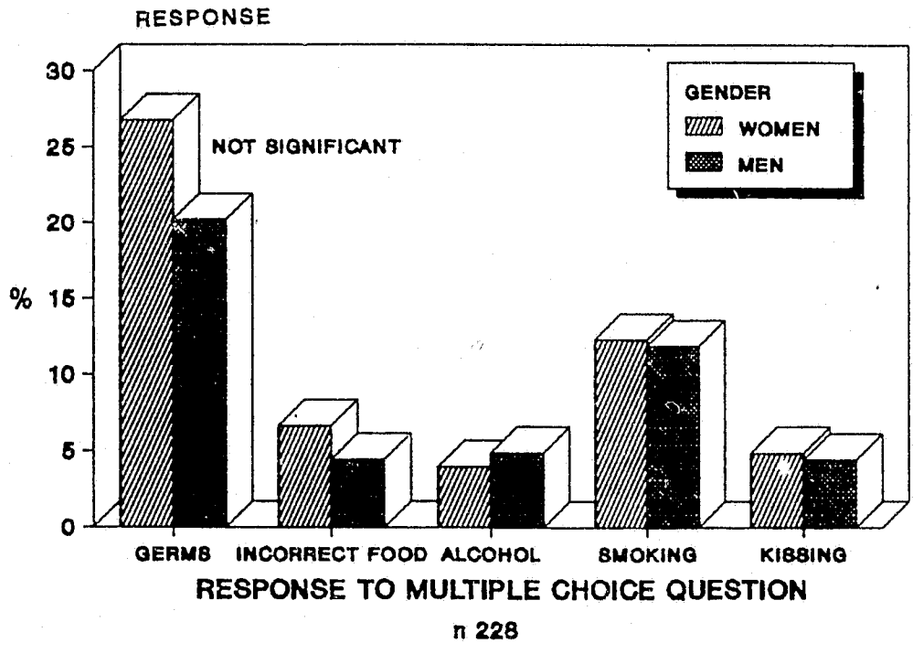
There were more heavy drinkers among the males especially in the older age group (of 45 years and older). There were also more female teetotallers (in all age groups) as compared to the male teetotallers. Refer to figure 45

7.3 RESIDENTS KNOWLEDGE OF AND ATTITUDES TO TB

Figure 46 illustrates the response to the cause of TB amongst the males and females of Macassar Camp.

Figure 46

Response of Male & Female To The Cause of TB

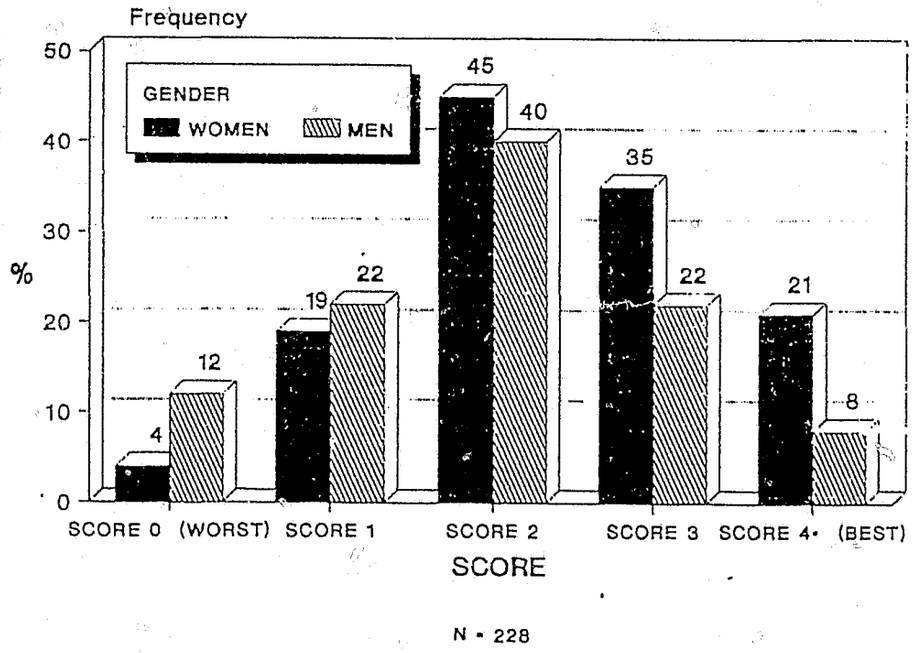


Of the 369 residents in Macassar Camp there were 278 residents 13 years or older who were eligible to respond to the section with regards to the

knowledge and attitudes on TB in the personal questionnaire. Of this sub-population, 228 (82,0%) responded to this section of the personal questionnaire. That the cause of TB was due to "germs" was responded to by females with a significantly ($p < 0,005$) greater number of correct responses as compared to the males (table 36). A majority of the Macassar Camp residents responded correctly to the questions assessing TB knowledge. There was no statistical difference between the sexes in the number of correct responses. Refer to figure 47.

Figure 47

Comparison of Male & Female TB Knowledge Scores Obtained by Macassar Camp Residents



N = 228

Based on the correct answers (refer to chapter 2), to the overall questions on TB knowledge[12], the overall TB knowledge of both sexes was good (on calculation of a score obtained by each of the residents). Figure 47, shows that more than 75% residents obtained a score of 2 or more. Females scored significantly higher (p = 0,002) than males with regard to the score 4 (a score of 4 was categorized as the best score). However, only 40% of the residents knew that it took 6 months to cure tuberculosis.

Table 37 illustrates the various responses related to the question: How long it takes to cure a TB patient? A significant (p = 0,017) majority of 92 respondents from a sub-group of 228 respondents, stated that it would take 6 months to cure TB.

Table 37

Macassar Camp Residents Response to Time Taken to Cure TB Patient

Response	%	95% Confidence Bounds
1 - 3 Months	25,9	18,4 - 33,4
4 - 5 Months	4,4	----------
6 Months	40,4	32,9 - 47,9
7 - 12 Months	10,1	2,6 - 17,6
Don't know/Other	18,4	10,9 - 25,9%

The response to the open ended question with regards to treatment of TB as summarized in table 38 was correctly answered by 108 (47,4%) residents to be tablets/medication. This response was determined to be significant (p = 0,004) in the number of correct answers amongst the residents in the Camp.

Ninety four (41,2%) of the 228 Camp residents, identified cough as the first sign/symptom of PTB whilst 55 (24,1%) residents stated the first sign/ symptom of PTB to be weight loss.

Table 38

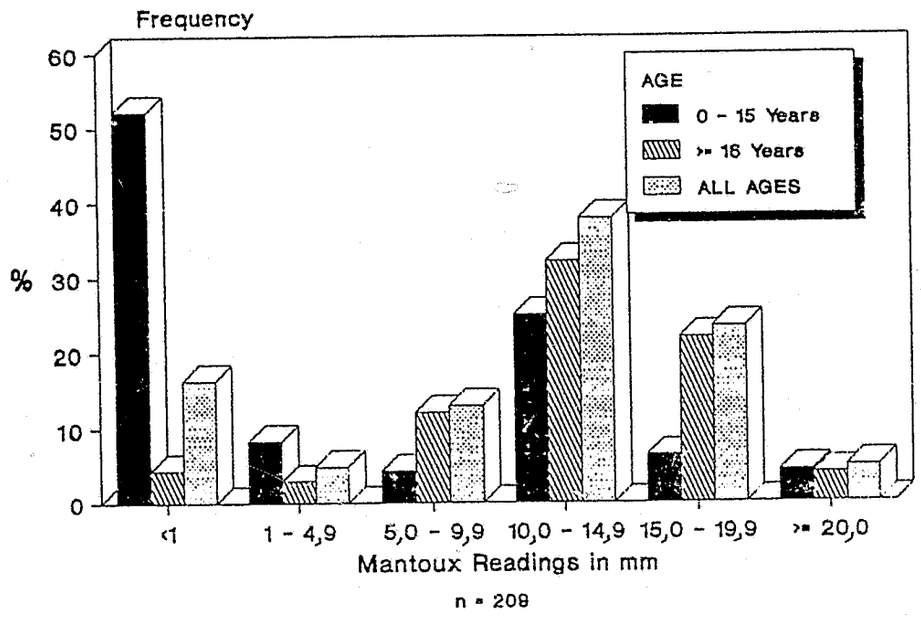
Macassar Camp Residents Response to a Question Related to the Treatment of a TB Patient

Type of of Response	% Frequency Response	95% Confidence Bounds
Tablets	47,4	39,9 - 54,9
Injections	30,3	22,9 - 37,8
Hospital	4,0	----------
X-Rays	1,8	----------
Food/Milk	0,9	----------
Don't Know	15,8	8,3 - 23,3

8.0 MANTOUX RESULTS (Appendix F4)

Figure 48

Mantoux Results Of Macassar Camp Residents.



8.1 MANTOUX (Mx) POSITIVE

Of the 369 Camp residents, 209 (56,6%) were tested for TB infection by Mantoux and TB ELISA methods. The cut-off point for a significantly positive Mantoux was 10 mm of induration at 72 hours for children aged 0 to 14 years; and 15 mm or more of induration at 72 hours for those aged 15 years or more.[13] Refer to table 39.

Based on the criterion for cut-off point of Mantoux test, there were 67 (32,1%) residents Mantoux positive. Table 39, illustrates the gender specific distribution of Mantoux positives among the two age groups.

Table 39

Outcome of Mantoux Tests Based on Age Groups and Gender

	0-14 YEARS	>=15 YEARS	TOTAL
Total Females	22	90	112
Mantoux +ve	10	33	43
% Positive	45,5%	36,7%	38,4%
Total Males	21	76	97
Mantoux +ve	2	22	24
% Positive	10,5%	28,9%	24,7%
Total for Group	43	166	209
Total +ve	12	55	67
% Positive	29,3%	33,1%	32,1%

8.1.1 Mantoux Positive Versus Gender

There were 43 (38,4%) females as compared to the 24 (24,7%) males who were Mantoux positive. This difference was significant (p = 0,035) with a relative risk of 1,55 and a 95% confidence interval of 1,02 - 2,36). Furthermore, in the less than 15 year old group, the females had 10 (45,5%) positive Mantoux results as compared to the males

who had 2 (9,5%) Mantoux positive results. This difference was significant (p = 0,009), with a relative risk of 4,77 and a 95% confidence interval of 1,18 - 19,27. This trend was noted in earlier studies.[20,21] However, for the age group 15 years or more there was no significant difference between the sexes with respect to the proportion of positive Mantoux tests.[20,21]

8.1.2 Mantoux Outcome of Household Contacts

Known PTB cases were excluded from the evaluation of Mantoux outcome amongst household contacts. Also excluded were those residents with a history of TB in the past (based on response in personal questionnaire and confirmed from the clinic records). This group comprised 196 residents of which 24 (12,2%) TB contacts were identified. These grouped as the positive TB contacts.[14]

Table 40

Contact Of PTB Cases With Household Members versus Mantoux Tests

		MANTOUX TESTS		
		+VE	-VE	TOTAL
Contact With PTB Cases In Household	**YES**	13 (54,2%)	11 (45,8%)	24 (100%)
	NO	47 (27,3%)	125 (72,7%)	172 (100%)
	TOTAL	60 (30,6%)	136 (69,4%)	196 (100%)

Table 40 illustrates that there was a significant ($p = 0,007$; relative risk of 1,98 (95% confidence interval of 1,27 - 3,08) difference in the proportion of positive Mantoux tests with regards to those residents who had been in contact with their respective household cases of PTB and those who were not in contact with household cases of PTB. On the inclusion, of past TB cases into the 'TB contact group', the difference was even more significant.

The TB household contact group was further subdivided into two groups (those residents aged less than 15 years and those residents aged 15 years or older).

Although no significant difference (with regards to household TB contacts) in the number of Mantoux positives was noted in the residents of 15 years of age or more, there was a significant difference among the residents in the less than 15 years age group.

Refer to table 41. The p value in this instance for the residents in the less than 15 years age group, was 0,027 with a relative risk of 3,25 (with a 95% confidence interval of 1,45 - 7,27).

Table 41

Contact Of PTB Cases With Household Members versus Mantoux Tests (Children <15 years)

		MANTOUX TESTS		
		+VE	-VE	TOTAL
Contact With PTB Cases In Household	**YES**	3 (75,0%)	1 (25,0%)	4 (100%)
	NO	9 (23,1%)	30 (76,9%)	39 (100%)
TOTAL		12 (27,9%)	31 (72,1%)	43 (100%)

8.1.3 Vesiculation

A total number of 29 (13,9%) vesiculations were observed among the 209 residents tested for Mantoux. Of these 29 residents with a vesiculation, 16 (55,2%) were Mantoux positive and only 13 (44,8%) had vesiculation but were not Mantoux positive (after 72 hour). This difference was significant (p = 0,004). And of the 209 residents only 129 were both Mantoux negative and had no vesiculation.

There 12 (41,4%) females that had vesiculation amongst the 29 vesiculations noted. However, there was no significant difference in the number of vesiculations observed based on gender. Also no association could be attributed to the appearance of vesiculations in 29 (13,9%) residents with regards to TB household contact, poor ventilation,

overcrowding or PFSL. However, none of the 29 residents with vesiculations were malnourished.

8.1.4 Annual Risk of Infection (ARI) of TB

Table 42 displays the Annual Risk of Infection (ARI) of TB in the respective age groups calculated on the number of Mantoux positives (Appendix B5 and F4)).

Table 42

Annual Risk of Infection (ARI) of TB in the Various Age Groups

Number Tested (n)	Age Group (Years)	Mode for Specific Age Group (mm)	No. of Readings >Mode	Annual Risk of Infection** (ARI)	No. of Mantoux* Positive
19	0-9	0	4	0,4	2 (10,5%)
24	10-14	0	14	1,2	10 (41,7%)
31	15-19	0	27	1,7	6 (19,4%)
47	20-29	0	43	1,8	16 (34,0%)
39	30-39	15,0	16	0,9	18 (46,2%)
25	40-49	11,6	3	1,4	8 (32,0%)
16	50-59	12,7	9	1,3	4 (25,0%)
8	>= 60	16,0	2	0,6	3 (37,5%)
209	ALL AGES	0	175	1,8	67 (32,1%)

* The cut-off point for Mantoux positives was 10mm and 15mm or more induration readings (at 72 hours after injection of PPD) for the 0-14 and 15 year or more age groups respectively.

** Refer to Appendix B5

The annual risk of infection (ARI) was determined by the method of by Styblo.[15] The cut-off point (positive) for Mantoux readings for the determination of prevalence in the community was 15 mm for both children and adults (figure 48).

9.0 ASSOCIATION BETWEEN MANTOUX OUTCOME AND RISK FACTORS

No significant difference in the proportion of positive of Mantoux tests among those residents who suffered from one or more diseases as compared to those who did not suffer from any one of these diseases could be detected (table 31).

9.1 EFFECT OF OVERCROWDING ON OUTCOME OF MANTOUX +VE

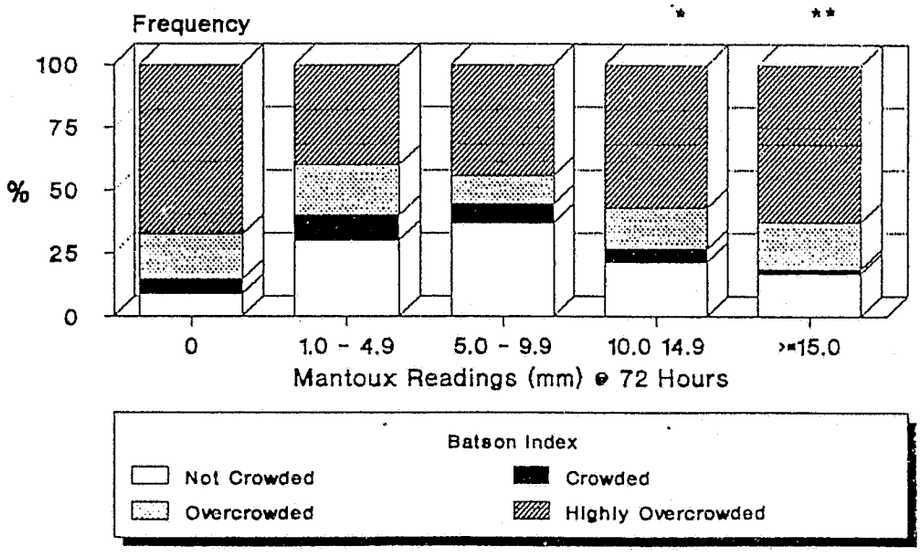
No significant difference was noted amongst those residents that were Mantoux positive and who lived in the various categories of overcrowding such as crowded, overcrowded and highly overcrowded (Batsons Index), as compared to those who did not reside in these categories of homes (Figure 49).

No difference could be confirmed even after comparing the number of Mantoux positives in those residents residing in not overcrowded homes as compared to those residents residing in highly overcrowded homes. However, the data in figure 49 demonstrates that it would appear overcrowding

does play a role in infection. Even after combining the category of 'not crowded' and 'crowded' as control and 'overcrowded' and 'highly overcrowded' as the crowded group, there was no significant difference in the number of Mantoux positives for those in the control group as compared to those in the crowded group. [16]

Figure 49

Distribution Of Mantoux Readings Based On Batsons Index Of Overcrowding



N = 369

* MANTOUX TEST WAS POSITIVE FOR SOME IN THIS CATAGORY

** MANTOUX TEST WAS POSITIVE FOR ALL IN THIS CATAGORY

No difference could be confirmed even after comparing the number of Mantoux positives in those residents residing in not overcrowded homes as compared to those residents residing in highly overcrowded homes.

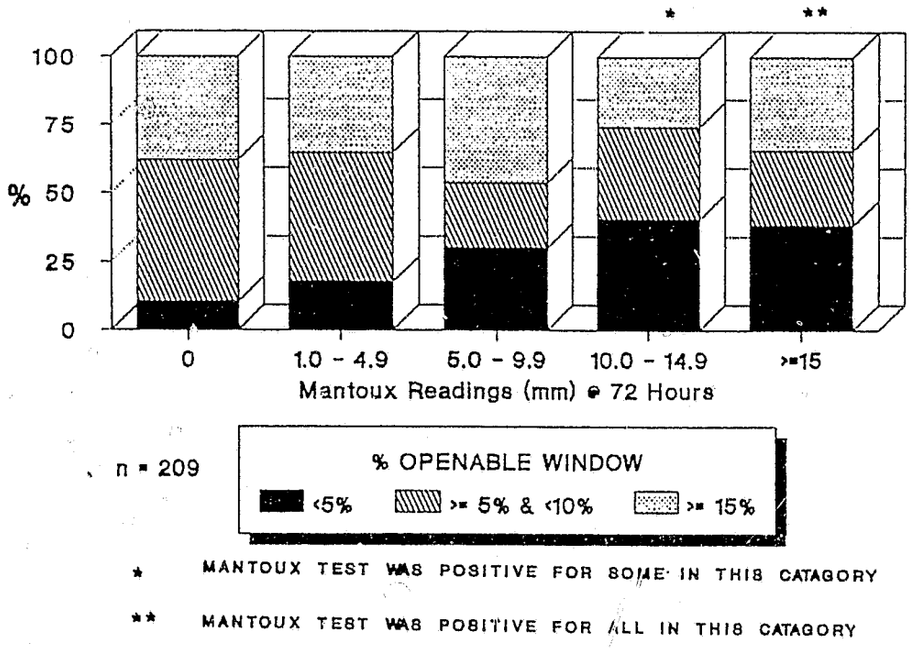
However, from the data in figure 49, it would appear that overcrowding does play a role in TB infection. Even after having combined the category of residents in the 'not crowded' and 'crowded' groups used as the 'control group' and 'overcrowded' and 'highly overcrowded' as the 'crowded group', there was no significant difference in the number of Mantoux positives for those in the 'control group' as compared to those in the 'crowded group'.

9.1.1 Effect of Ventilation on Outcome of Mantoux +VE

There was no significant difference in the number of Mantoux positives for those in the group exposed to less than 5% (based on the openable window area with respect to floor area), ventilation and those in the group that were exposed to 5% or more ventilation in their homes. Figure 50 illustrates the degree of ventilation when compared to the Mantoux readings.

Figure 50

Distribution of Mantoux Readings Based on Degree of Ventilation



* MANTOUX TEST WAS POSITIVE FOR SOME IN THIS CATAGORY

** MANTOUX TEST WAS POSITIVE FOR ALL IN THIS CATAGORY

9.1.2 Effect of Overcrowding and Ventilation on Outcome of Mantoux Positives

From the population of 209 residents in Macassar Camp, a control group was selected from those residents who were not residing in overcrowded homes (Batson Index of 1 & 2) and were not subjected to overall household ventilation of less than 5% (table 43). The test group consisted of residents that were living in overcrowded homes (Batson Index of 3 and 4) and were subjected to overall household ventilation of 5% or more. The 11 (44%) Mantoux positives in the group that was

regarded as not overcrowded and with a ventilation of <5% were significantly more (p = 0,018) than the 5 positives in the group who were in the group regarded as not overcrowded and with a ventilation of >=5%. The relative risk was 2,82 (with a 95% confidence interval of 1,12 - 7,06.

Table 43

Combined Effect Of Overcrowding & Poor Ventilation on Outcome of Mantoux +VE

	MANTOUX TEST +VE	-VE	TOTAL
Overcrowded & Ventilation of < 5%	11 (44,0%)	14 (56,6%)	25 (100%)
Not Overcrowded & Ventilation of >= 5%	5 (15,6%)	27 (84,4%)	32 (100%)
TOTAL	16 (28,1%)	41 (71,9%)	57 (100%)

9.1.3 Effect of Type of Toilet on Outcome of Mantoux Positives

The dwelling units in the Camp had three types of toilets:
- inside the dwelling unit
- outside the dwelling unit
- communal toilets

There was no significant difference in percentage of those who were Mantoux positive and who lived in dwelling units with toilets inside as compared to those who were Mantoux positive and lived in dwelling units with communal toilets or toilets situated outside their homes.

9.2 EFFECT OF A MISCARRIAGE ON THE OUTCOME OF MANTOUX POSITIVE RESULT

Table 44 shows the effect of miscarriage on the outcome of Mantoux results. Mantoux positive females were matched for age (within 5 year range) and race with 2 females who were Mantoux negative. There was a significant difference (p = 0,023) in the percentage of those who were Mantoux positive and who had one or more miscarriages in comparison to those who were Mantoux negative and did not have a miscarriages at all. The relative risk was 2,38 (95% confidence interval of 1,28 - 4,42).

Table 44

Effect Of A Miscarriage On Outcome Mantoux Results

		MANTOUX TESTS		
		+VE	-VE	TOTAL
MISCARRIAGE	YES	6 (66,7%)	3 (33,3%)	9 (100%)
	NO	16 (28,1%)	41 (71,9%)	57 (100%)
	TOTAL	22 (33,3%)	44 (66,7%)	66 (100%)

9.3 EFFECT OF DIABETES/ABDOMINAL OPERATION ON OUTCOME OF MANTOUX POSITIVES

The presence or past history of one or more of these disease/ailments in the Camp residents interviewed did not contribute to a significant increase in the number of Mantoux positives.

9.4 ASSOCIATION OF SPORT/EXERCISE ON OUTCOME OF MANTOUX POSITIVES

Table 45 illustrate the association of sport/ exercise with the outcome on Mantoux results. Among the participants aged 13 years or older there was a significant difference (p = 0,025) with regards to the large proportion of those residents who participated in sport/exercise (at least once a week), were Mantoux negative (80%) as compared to those residents who were Mantoux positive (20%) and who participated in sport/ exercise (at least once a week). The relative risk was 0,53 with 95% confidence interval of 0,29 - 0,97. Refer to table 45. Thus it would appear when one is 'exposed' to sport/exercise chances are less (since relative risk less than 1) in being Mantoux positive (infected) than when residents were 'not exposed' to regular sport/exercise. This result might suggest that suggest tnat sport/ exercise has a protective effect.

Table 45

Protective Effect of Sport In Reduction of TB Infection

		MANTOUX TESTS +VE	-VE	TOTAL
SPORT	YES	10 (20,0%)	40 (80,0%)	50 (100%)
	NO	43 (37,7%)	71 (62,3%)	114 (100%)
	TOTAL	53 (32,3%)	111 (67,7%)	164 (100%)

9.5 EFFECT OF MALNOURISHMENT ON MANTOUX OUTCOME POSITIVES

Table 46a, 46b, and 46c illustrates the Mantoux results with regards to malnutrition based on height for age (HAZ), weight for age (WAZ) and weight for height (WHZ) respectively.

Table 46a

Mantoux and Z-Scores of Height for Age (HAZ) values in Children Aged <=14 Years

		MANTOUX TESTS		
	MALNUTRITION CATEGORIES	**+VE**	**-VE**	**TOTAL**
Z-SCORES VALUES	**Z** <= -2 (Malnourished)	1	1	2
	-2 > **Z** <= -1,5 Borderline)	2	7	9
	Z > -1,5 (Normal Range)	9	23	32
	TOTAL	12	31	43

Table 46b

Mantoux and Z-Scores of Weight for Age (WAZ) values in Children Aged <=14 Years

		MANTOUX TESTS		
	MALNUTRITION CATEGORIES	**+VE**	**-VE**	**TOTAL**
Z-SCORES VALUES	**Z** <= -2 (Malnourished)	1	2	3
	-2 > **Z** <= -1,5 Borderline)	1	0	1
	Z > -1,5 (Normal Range)	10	29	39
	TOTAL	12	31	43

Table 46c

Mantoux and Z-Scores of Weight for Height (WHZ) values in Children Aged <=14 Years

		MANTOUX TESTS		
	MALNUTRITION CATEGORIES	**+VE**	**-VE**	**TOTAL**
Z-SCORES VALUES	**Z <= -2** (Malnourished)	0	2	2
	-2 > Z <= -1,5 (Borderline)	0	1	1
	Z > -1,5 (Normal Range)	12	28	40
	TOTAL	12	31	43

Table 47 illustrates Mantoux results with regards to malnutrition (for males and females) based on BMI for residents aged 15 years or older.

Table 47a

Mantoux and Body Mass Index (BMI) Categories Residents aged >=15 Years

	MANTOUX TESTS		
MALNUTRITION CATEGORIES	**+VE**	**-VE**	**TOTAL**
Malnourished	4 (F=0, M=4)	33 (F=13, M=20)	37
Normal	37 (F=24, M=13)	33 (F=14, M=19)	70
Slightly Obese	10 (F=6, M=4)	25 (F=15, M=10)	35
Obese	7 (F=7, M=0)	10 (F=6, M=4)	17
Excessive Obese	2 (F=1, M=1)	4 (F-3, M=1)	6
TOTAL	60 (F=38, M22)	105 (F=51, M=54)	165

When the children that were categorized as malnourished, based on categories of HAZ, WAZ or WHZ and were added to those adults that were evaluated as malnourished based on the BMI values there was no significant difference (p = 0,19) between those that were Mantoux positive and malnourished as compared to those that were not malnourished and Mantoux negative. Thus a total of 42 (20,2%) residents could be regarded as malnourished (table 47b).

Table 47b

Mantoux versus Malnourished Categories (Based on BMI Standard, HAZ, WAZ or WHZ)

		MANTOUX TESTS		
		+VE	-VE	TOTAL
MALNOURISHED BASED ON BMI, WAZ,WHZ OR WHZ	**YES**	10(23,8%)	32(76,2%)	42(100%)
	NO	57(34,3%)	109(65,7%)	166(100%)
TOTAL		67(32,2%)	141(67,8%)	208(100%)

9.6 EFFECT OF PRIMARY FOOD SUBSISTENCE LEVEL (PFSL) ON MANTOUX OUTCOME POSITIVES

Table 48 illustrates that there were significantly more 54 (37,8%) residents below the PFSL and Mantoux positive compared to those residents who were above PFSL and Mantoux positive 13 (19,7%). The p value = 0,009 with a relative risk of 1,92 (95% confidence interval of 1,13 - 3,26).

Table 48

Relationship Of Residents who are Below PFSL & Mantoux Positivity

		MANTOUX TESTS		
		+VE	-VE	TOTAL
PFSL	BELOW	54 (37,8%)	89 (62,2%)	143 (100%)
	ABOVE	13 (19,7%)	53 (80,3%)	66 (100%)
	TOTAL	67 (32,1%)	142(67,9%)	209 (100%)

9.7 EFFECT OF TB SYMPTOMS/SIGNS ON MANTOUX OUTCOME POSITIVES

There existed no relationship between those residents who responded in the affirmative (a response of two or more 'yes') to the sign and symptoms of tuberculosis with regard to the outcome of the Mantoux results.

However, it should be noted that one resident who had responded in the affirmative to all the questions of signs and symptoms for TB (before the results of the mass miniature chest X-ray were received), was accompanied on that very day to the clinic for chest X-ray which indicated a strong evidence of PTB. The resident was immediately placed on treatment. Results of sputum microscopy and culture of this resident further confirmed the earlier diagnosis of PTB.

9.8 EFFECT OF SMOKING ON MANTOUX OUTCOME

There was no significant difference in the number of Mantoux positives with regards to smokers and non-smokers. Even after categorizing the smokers into mild smokers (<= 9 cigarettes per day) and heavy smokers (>= 10 cigarettes per day). Further more no significant difference in the number of Mantoux positives was observed, even on matching 1 Mantoux positive with 2 Mantoux negatives, randomly selected from the study sample for age and sex (age +-5 years) and race. Thus there was no significant difference between those that were Mantoux positives and smokers as compared to those that were Mantoux positive and non-smokers even after matching. Refer to table 49.

Table 49

Relationship between Smoking & Mantoux Positivity

	MANTOUX TESTS		
	+VE	**-VE**	**TOTAL**
SMOKERS	29 (39,2%)	45 (62,8%)	74 (100%)
NON-SMOKERS	11 (23,9%)	35 (76,1%)	46 (100%)
TOTAL	40 (33,3%)	80 (66,7%)	120 (100%)

9.9 EFFECT OF ALCOHOL ON OUTCOME OF MANTOUX POSITIVES

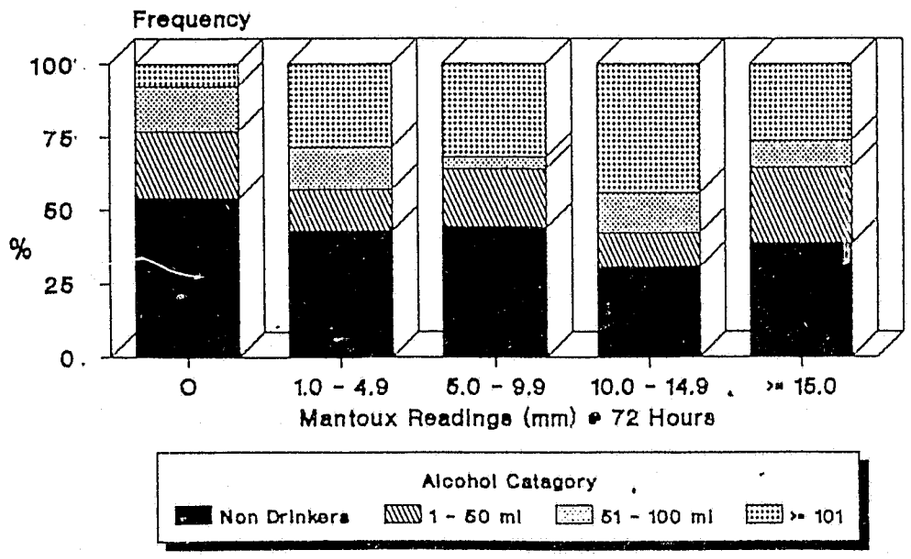
There was no significant difference between the teetotallers and those consuming alcohol with regards to Mantoux positivity. However, there

was a considerable decrease in the p value amongst those that were Mantoux positive and teetotallers as compared to those that were Mantoux positive and who consumed alcohol more than 20 ml of absolute alcohol.

Figure 51, illustrates that more than 50% of the teetotallers had Mantoux readings <1 mm. Those residents within the range of Mantoux readings of 1,0-4,9 mm and 5-9,9 mm comprised approximately of 40% of the teetotallers in both instances. However, there appeared to be a decrease among teetotallers among those residents within the Mantoux readings of 10,0-14,9 mm and >= 15 mm as compared to those residents that had consumed alcohol >= 51 ml.

Figure 51

Impact Of Alcohol Intake On Mantoux Readings



9.10 EFFECT OF OCCUPATIONAL STATUS ON MANTOUX OUTCOME

When occupational status was categorized into two main groups, namely skilled and unskilled workers, there were significantly more (p = 0,012) Mantoux positives amongst the unskilled workers 29 (37,2%) as compared to the skilled workers 5 (13,9%). Refer to table 50. The relative risk was 2,68 with 95% confidence interval of 1,13 - 6,35.

Table 50

Effect Of Work Status On Mantoux Positivity

		MANTOUX TESTS		
		+VE	**-VE**	**TOTAL**
WORK STATUS	**UNSKILLED**	29 (37,2%)	49 (62,8%)	78 (100%)
	SKILLED	5 (13,9%)	31 (86,1%)	36 (100%)
	TOTAL	34 (29,8%)	80 (70,2%)	114 (100%)

10.0 **TB DIAGNOSIS** (Appendix F4)

10.1 X-RAYS (RADIOGRAPHY)

Table 51, illustrates the results of 275 mass miniature chest X-rays that were performed in the mobile unit which was used as one as one of the TB screening test.

Table 51

Mass Miniature X-Rays Results Taken in Mobile X-Ray Unit

Miniature X-Ray Report	Number	%
Radiologically Suspect PTB (RSPTB)	21	7,6
Radiologically Active PTB (RAPTB)	8	2,9
Other Pathology (OP)	3	1,1
Spoilt Plates (SP)	5	1,8
No Abnormalities Detected (NAD)	238	86,5
TOTAL	275	100

Table 52 displays all suspicious mass miniature chest X-rays as well as residents with a history past TB (KCPTB) including those with spoilt plates (SP), were referred to the clinic for further tests. The total number of residents referred to the clinic for further diagnosis based on mass chest X-rays in the mobile X-ray unit was 32 of which 5 could not be traced. Of the 6 PTB cases confirmed radiologically in 1988 (table 52), 1 was confirmed a positive PTB case in 1989, when the resident who was initially reported to be radiologically active PTB (miniature chest X-rays performed in the mobile X-ray unit) was traced. Of the 275 residents, 238 had no abnormalities detected (NAD) on the chest X-ray and were not referred to the clinic for further diagnosis.

Table 52

Comparison of Mass Miniature Chest X-Rays and Repeat Of Suspicious X-Rays Taken At The Clinic

Mass Miniature X-Rays	Outcome Of Repeat X-Rays @ Clinic				
	PTB	KCPTB	OP	NAD	TOTAL
RSPTB	2	8	-	8	21
RAPTB	4	1	-	1	8
OP	-	-	3	-	3
SP	-	-	-	5	5
TOTAL	6*	9	3	14	32

10.2 SPUTUM MICROSCOPY AND CULTURE

Table 53 tabulates the results of the microbiological analysis of the sputa.

Table 53

Sputum Microscopy & Culture

Patient Number	Sex		Mass Miniature Result	Microscopy	Culture
8308*	51	M	RAPTB	++	+
9101	30	F	RAPTB	-	+
5504	27	F	RAPTB	++	+
1702**	58	M	RAPTB	+	+
5502	36	F	RSPTB	++	+
5101	63	M	RSPTB	++	+

* Resident with identity number 8308, was reported radiologically active on mass

miniature chest X-ray (past PTB case sometime in 1985 and had a history of being non-compliant) and was traced in early part of 1989 and PTB was confirmed with a repeat X-ray at the clinic. Further confirmation of the disease was supported by microscopy and culture of the resident's sputum. Hence due to reactivation, patient 8308 was regarded as a new PTB case.

** Resident 1702, had a history of past PTB and had completed treatment but had experienced a relapse within one year later (1988) and hence this was regarded as a relapse case and not a new PTB case.

Sputum evaluation included the 32 'TB suspect cases' (which included the following categories: 18 RSPTB, 6 RAPTB, 3 OP and 5 SP) but excluded 5 'TB suspects' who could not be traced for a sputum specimen. The sputa were examined by microscopy and culture.

10.3 PREVALENCE OF PTB DUE TO ACTIVE CASE FINDING

In 1988, 5 new cases of PTB were confirmed in Macassar Camp and the prevalence of PTB (due to active case finding) in this population was calculated to be 1 355 per 100 000 in 1988.

11.0 ASSOCIATION OF RISK OF TB INFECTION WITH SOCIOECONOMIC DETERMINANTS

11.1 RISK FACTORS FOR TB INFECTION VERSUS TB ACTIVATION

The data compiled in table 54, was based on results of this study confirming the risk factors for being infected.[23a-23n] Many of these risk factors have in the past studies shown to contribute to the development of active TB. However, in this instance the study has shown the risks of being infected rather than factors that would activate the development of TB after the initial infection with *Mycobacterium tuberculosis*.

11.2 PROTECTIVE EFFECT OF SPORT/EXERCISE AGAINST TB INFECTION

This study has shown that regular sport/exercise is associated with a protective effect against TB infection and may have contributed to at least 47% (1-0,53) reduction in (TB infection) the number of Mantoux positives.

11.3 VARIOUS RISK FACTORS RELATED TO SOCIOECONOMIC STATUS ATTRIBUTED TO TB INFECTION

Table 54, tabulates the risk factors for TB infection (Mx positive versus exposure to various risk factors established in this study). The asterisk in table 54 indicates (Appendix E7):

* AR: Attributable risk
** AR%: Attributable Risk %
*** RR: Relative Risk
**** Regular Sport/Exercise - protective effect (because RR <1)

Table 54

Risk Factors for *Mycobacteria* Infection

Risk Factors	n	RR***	p	95% CI	AR*	AR%**
Gender	209	1,55	0,035	1,02-2,36	0,136	33,3
Females <15 years)	43	4,77	0,009	1,18-19,27	0,360	79,0
Household Contacts	196	1,98	0,007	1,27-3,08	0,269	49,5
Contacts (<15 yrs)	43	3,25	0,027	1,45-7,27	0,519	69,2
Poor Ventilation & Overcrowding	57	2,82	0,018	1,12-7,06	0,284	64,5
Primary Food (PFSL) Subsistence Level	209	1,92	0,009	1,13-3,26	0,502	47,9
Unskilled & Partly Skilled	114	2,68	0,012	1,13-6,35	0,233	62,7
Miscarriage	66	2,38	0,023	1,28-4,42	0,386	57,9
Regular Sport/ Exercise****	164	0,53	0,025	0,29-0,97	-0,177	88,7

11.3.1 Risk factors associated with TB, such as gender, miscarriage and socioeconomic factors such as poor nutritional intake (PFSL), unskilled work, household contact with TB patients and poor ventilation and overcrowding were confirmed to be risk factors associated with TB infection in this study. The aspect of overcrowding as an important contributing factor for TB is illustrated by an earlier study (figure 52).

11.3.2 It was noted that all the PTB patients listed in table 53 responded in the affirmative to these risk factors in excess of 50% Refer to table 55 which tabulates the association of risk factors with confirmed PTB cases of Macassar Camp.

Table 55

Association of Risk Factors With Confirmed PTB Cases Amongst Macassar Camp Residents

	PTB PATIENTS OF MACASSAR CAMP					
RISK FACTORS*	8308	9101	5504	1702	5502	5101
GENDER	N	Y	Y	N	Y	N
HOUSEHOLD CONTACT	N	N	Y	Y	Y	N
POOR VENTILATION	Y	Y	N	N	N	Y
OVERCROWDING	N	Y	Y	Y	Y	Y
POOR VENTILATION AND OVERCROWDING	N	Y	N	N	N	Y
MISCARRIAGE	N/A	Y	N	N/A	N	N/A
SPORT/EXCERCISE	N	N	N	N	N	N
BELOW PRIMARY FOOD SUSISTANCE LEVEL (PFSL)	Y	Y	Y	Y	Y	Y
SMOKER	Y	N	Y	Y	Y	N
UNSKILLED/UNEMPLOYED	Y	Y	Y	Y	Y	Y
MALNOURISHED	**	Y	Y	Y	Y	N

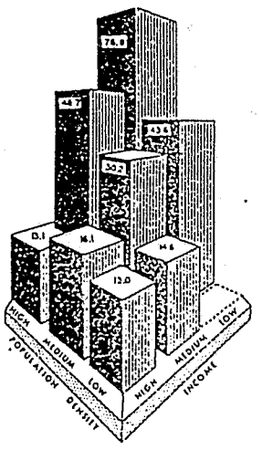
* REFER TO TABLE 54 FOR RELATIVE RISK
** DATA NOT AVAILABLE

11.4 INCREASE IN TB INCIDENCE ASSOCIATED WITH HIGH DENSITY AND LOW AND MEDIUM INCOME

The study in New York City 1970, (figure 52) which grouped income by population density showed that there was no significant change associated with population density and TB incidence rates for the residents with a high income. However there appeared to be direct association between the population density and the TB incidence rates for both medium and low income groups. This point further confirms that overcrowding is an important contributing factor for TB infection.

Figure 52

Newly Reported Active Tuberculosis Case Rates per 100 000 Population in Health Districts Grouped By Income & Population Density in New York City, 1970



Source:

Immunization in Tuberculosis, Fogarty International Proceedings No. 14, (1971), p30, DHEW Publication No. (NIH) 72-68:Chaves, AD; The Problem of Tuberculosis in Selected Populations.

11.5 ECONOMY VERSUS HEALTH AND TB

Table 56a lists the leading causes of death In Taiwan. TB being the third leading cause of death in 1952 and by 1989, TB was listed as the eleventh cause of death. The the disease profile in 1952 was very similar to that of Blacks in South Africa whereas in 1989 the disease profile of the same population (Taiwanese) is similar to the Whites of South Africa (table 56b).

Table 56a

The Leading Causes of Death In Taiwan (1952-1989)

1952					1989				
Order	Causes	No. of Deaths	Death Rate Per 100,000	%	Order	Causes	No. of Deaths	Death Rate Per 100,000	%
	All causes	76,053	950.80	100.00		All Causes	102,242	511.07	100.00
1	Gastritis, duodenitis, enteritis & colitis except for diarrhoea of the newborns	10,799	135.01	14.20	1	Malignant Neoplasms	18,878	94.36	18.46
					2	Cerebrovascular Disease	14,461	72.28	14.14
					3	Accidents	14,047	70.22	13.74
					4	Heart Disease	10,699	53.48	10.46
2	Pneumonia	10,516	131.47	13.83	5	Diabetes mellitus	3,868	19.33	3.78
3	Tuberculosis, all froms	7,324	91.56	9.63	6	Chronic liver Disease und Cirrhosis	3,550	17.75	3.47
4	Diseases of heart	3,922	49.03	5.16					
5	Vascular lesions affecting central nervous system	3,902	48.78	5.13	7	Hypertensive Disease	2,966	14.83	2.90
					8	Pneumonia	2,946	14.73	2.88
					9	Bronchitis Emphysema & Asthma	2,551	12.75	2.50
6	Causes of perinatal mortality	3,524	44.06	4.63					
					10	Nephritis, nephrotic syndrome und nephrosis	2,094	10.47	2.05
7	Nephritis & nephrosis	2,904	36.31	3.82					
8	Malignant neoplasms	2,459	30.74	3.23		Others	26,182	130.87	25.61
9	Bronchitis	2,250	28.31	2.96	11	Tuberculosis	1,900	9.50	1.86
10	Malaria	2,196	27.45	2.89	12	Suicide	1,573	7.86	1.54
					13	Septicaemia	1,449	7.24	1.42
	All other causes	26,257	328.26	34.52	14	Ulcer of stomach and duodenum	862	4.14	0.81
					15	Congenital anomalies	771	3.85	0.75

Remarks: Mid-year population: 7,998,810 Mid-year Pop.: 20,005,626

Source:

A Review Of The Tuberculosis Control Program In Taiwan (1941-1989), Taiwan Provincial Chronic Disease Control Bureau, 1991.

No one can deny the impact of socioeconomic factors on the health and disease of a population. However, no one has yet conclusively proved to what extent if any, the effect of socioeconomic conditions on the health of the population (because of ethics and problems in designing a case control study to prove this factor). However, Taiwan is one country (Cuba may be another example) that illustrates very clearly the impact, on the health of the population before and after the significant shift in the socioeconomic conditions ('The Taiwan Economic Miracle').

Table 56b

The Leading Causes of Death In South Africa (1976)

Order of importance	*White*	*Indian*	*Coloured*	*African*
1	Ischaemic heart diseases	Ischaemic heart diseases	Enteritis & other diarrhoeal diseases	Pneumonia (excluding viral pneumonia)
2	Cerebrovascular diseases	Cerebrovascular diseases	Pneumonia (excluding viral pneumonia)	Enteritis & other diarrhoeal diseases
3	Pneumonia (excluding viral pneumonia)	Pneumonia (excluding viral pneumonia)	Cerebrovascular diseases	Homicide & wilful injury by others
4	Motor vehicle accidents	Hypertension	Ischaemic heart diseases	Cerebrovascular diseases
5	Bronchitis, emphysema & asthma	Motor vehicle accidents	Homicide & wilful injury by others	Tuberculosis of the respiratory system
6	Malignant neoplasms of the trachea, bronchus & lung	Enteritis & other diarrhoeal diseases	Immaturity (not specified)	Immaturity (not specified)
7	Senility (without psychosis)	Immaturity (not specified)	Tuberculosis of the respiratory system	Motor vehicle accidents
8	Venous thrombosis & embolism	Bronchitis, emphysema & asthma	Motor vehicle accidents	Anoxic & hypoxic conditions
9	Diseases of the arterioles & capillaries	Diabetes mellitus	Bronchitis, emphysema & asthma	Malignant neoplasm of the oesophagus
10	Suicide & self-inflicted injury	Cirrhosis of the liver	Hypertension	Measles

Source:

van Rensburg HCJ & Mans A, Profile of Disease & Health Care in South Africa, Academica, Pretoria/ Cape Town/Johannesburg, 1982, p78, Table 22.

CHAPTER 7
CONCLUSION & DISCUSSION

"If someone tells me that in making these conclusions I have gone beyond the facts, I reply: 'it is true that I have freely put myself among ideas which cannot be rigorously proved. That is my way looking at things.' Only theory can bring forth and develop the spirit of invention."

Louis Pasteur

CHAPTER 7

CONCLUSIONS AND DISCUSSION

SUMMARY

The chapter commences by stating that insufficient data is available to determine the origin of *Mycobacterium tuberculosis* in South Africa. However, there exist two diametrically opposing views of the origin of TB epidemic. But the opponents of both viewpoints have nevertheless the same common goal, which is focussed on the control and the elimination of TB.

The TB epidemic as a worldwide phenomenon is summarized and the prevalence of infection in Africa, South Africa, Western Cape and Macassar is discussed.

This chapter concludes on the available literature reviewed for this study, the clinic records of TB, the survey questionnaire and the measurement of certain variables.

Furthermore it concludes on the Mantoux results and compares this with the TB ELISA results. And finally presents the prevalence of TB cases confirmed in this study which leads on into a discussion with regards to the Camp residents who were exposed to risk factors associated TB infection (confirmed in this study) and the development of active TB.

CHAPTER 7

CONCLUSION AND DISCUSSION

[A] CONCLUSIONS

[I] This study has shown from the available literature reviewed that:

1 THE CAUSAL AGENT - *Mycobacterium tuberculosis*

The causative organism of TB was isolated and identified as *Mycobacterium tuberculosis* by Robert Koch in 1882.

1.1 EVOLUTION/MUTATION OF *Mycobacterium tuberculosis*

1.1.1 *Mycobacterium tuberculosis* may have evolved or mutated from *Mycobacterium bovis*, the causal agent of TB in cattle.[1,2,30]

2 THE 'VIRGIN POPULATION' THEORY OF TB

2.1 THE ORIGIN OF *Mycobacterium tuberculosis*

2.1.1 The origin of *Mycobacterium tuberculosis* in South Africa and the causes of the current TB epidemic remain debatable. However, proponents of these two diametrically opposing theories (the 'Virgin' and 'Non Virgin' Theories) have the same common goal; that is the control, prevention and elimination of TB.[3,4,5,6,7]

3 THE TB EPIDEMIC IS A WORLDWIDE PHENOMENON

3.1 GLOBAL OVERVIEW OF TB

3.1.1 TB epidemics are thought to be cyclical epidemic and the span of the epidemiological curve without any intervention could last for centuries. The cyclical curve of an epidemic is believed to have a short ascent but a longer descent with a plateau in between.[9,8,10]

3.1.2 It has been estimated that more than 1,7 billion (33%) of the world's population are infected with *Mycobacterium tuberculosis*.[14]

3.1.3 *Mycobacterium tuberculosis* has now successfully infected more than one third of the worlds population (besides those with active TB). The advent of the HIV pandemic, will further exacerbate the spread of this TB epidemic, in the developing countries and has caused a resurgence of the TB in developed countries.[40,41,42]

3.1.4 The approximate annual incidence of TB worldwide has been estimated to be between 8 and 10 million resulting in approximately 3 million deaths annually.[34]

3.2 PREVALENCE OF TB INFECTION IN AFRICA

3.2.1 In Africa, 77% of infected individuals are less than 50 years of age whereas in Europe 80% of the

infected individuals are aged 50 years or more. Refer to figures 8a and 8b.

3.2.2 The prevalence of infection (tubercle bacillus), in Africa, has been estimated to be 54%. The presence of dual TB/HIV infection was estimated (by 1990), to be greater than 3 million and 78% of the 3 million of dual HIV/TB infections was estimated to occur in Africa whereas less than 6% of the dual infections had occurred in Europe and 5 industrialized countries.[14] Refer to table 4.

3.2.3 The number of dual TB/HIV infections, by 1992, increased dramatically. Approximately 4 million of TB/HIV cases were reported worldwide in 1992, with about 3,12 million (77,8%) living in Sub-Saharan Africa.[31,37,43] Refer to Appendix D1.

3.3 TB IN SOUTH AFRICA

3.3.1 Since 1921 the TB incidence rates steadily rose from 43/100 000 population to 372/100 000 population in 1963 and it was believed (by some) that the TB epidemic had reached its peak.[16]

3.3.2 However, between 1977-1988, the TB incidence rates in South Africa were more or less stable and by 1989 and 1990 a further rise in TB incidence rate was noted to the effect of 211 per 100 000 and 229 per 100 000 population respectively.[11,12,13,17]

3.3.3 By 1989, the TB incidence rates (all forms) in South Africa had reached 186,7 per 100 000 population and 547 per 100 000 for the Coloured population of South Africa (figure 27) [18]

3.4 **TB IN THE WESTERN CAPE**

3.4.1 Since 1975, the Western Cape Region has been experiencing a rise in the number of new cases and an overall increase in the TB notification rates. [19,20] By 1991 the Western Cape had the highest PTB notification rates as when compared to all other health regions in South Africa (Appendix D8 and figure 22a).

3.4.2 The Western Cape had the highest TB notification rates amongst all the 7 Health Regions and 12,5 greater TB notification rate than Northern Transvaal Health Region. [21] This increase has been noted especially among the Coloureds.

3.4.3 The Western Cape (in 1988), had also the highest percentage of TB patients attending the local authority clinics for TB treatment and the lowest percentage of the hospital treatment of TB patients amongst all the 7 Health Regions in the country. [22]

[II] **This study has also shown from the review of clinic records of TB that:**

3.5 TB IN MACASSAR

3.5.1 Macassar/Firgrove (Mac/Fir) was the area with the second highest incidence of PTB (1986, 1988, & 1989) when compared to all other arbitrary regions. The area with the highest incidence of PTB (in 1985, 1986, 1987, 1988 & 1989) was Eastern Region (E/RGN), which comprised of the Paarl Branch of RSC and Municipalities of Paarl and Wellington (figures 26,30 and Appendixes E5a,E5b).

3.5.2 TB notification rate (all forms) in Macassar was 485,2 per 100 000 in 1989, (the second highest TB rate in a Coloured area in the WCHR). And the deaths due to TB in Macassar in 1988, had reached 21,82 per 100,000 population (figure 35, 36 and Appendix E6).

3.5.3 Of the 146 workers employed in Macassar Camp, 106 (73,1%) were employed by the Food Factory which owned Macassar Camp. The workforce of this Food Factory fluctuated between 300-600 workers and there was history of TB amongst these workers. A total of 22 TB cases was confirmed between 1981-1986 (which included many of the Camp residents). Refer to tables 16, 17 and 18.

3.5.4 Of the 8 past TB cases in Macassar Camp (1972-86), all were recorded to be TB culture positive and only 1 was AFB sputum negative (table 18).

[III] **This study has also shown from the review of the survey questionnaire and the measurement of certain variables that:**

4.0 **DWELLING UNITS OF MACASSAR CAMP**

4.1 The room occupancy rate in Macassar Camp in 1988, was 2,5 people per habitable room.

4.2 Only 17 (28,3%) dwelling units were categorized as not overcrowded (table 19) based on the Batson Overcrowding Index.[24,25,26] As a result 73 (20%) of the residents had no exposure to overcrowding (not overcrowded category). Refer to figure 37.

4.3 Sanitation was fairly adequate in Macassar Camp, with 22 (36,7%) dwelling units having an outside toilet and (6,7%) dwelling units using a communal toilet.

4.4 Based on a housing regulation (Government Gazette No. 3805),[27] on ventilation for housing, it was determined that 16 (26,7%) of the dwelling units (26,7%) in Macassar Camp had an overall household ventilation of less than 5% of openable window to floor area (within the limits of the housing regulations). Refer to table 20.

4.5 As a result of the overcrowded dwelling units, this resulted in 79 (21,4%) residents that were

exposed to the overall household ventilation of less than 5% of openable window to floor area, in their respective homes. Refer to figure 38.

5 **EMPLOYMENT**

5.1 There were 21 (12,5%) residents in the Camp unemployed among those whom comprised the 'employable group' or the productive workforce of 167 residents and (65%) of employed residents were unskilled or partly skilled (semi-skilled). Refer to tables 22, 23 and 24.

5.2 There were 69 (72,6%) residents that were unskilled and below PFSL as compared to 23 (45,1%) residents that were skilled and below PFSL. This difference was significant (p = 0,001), with an odds ratio of 1,6 at a 95% exact confidence interval range of 1,16 - 2,23. Refer to table 25.

6.0 **HEALTH STATUS OF CAMP RESIDENTS**

6.1 **BCG STATUS OF CAMP RESIDENTS**

A total of 199 (67,2%) of the 296 residents had evidence of BCG vaccination and of the 83 children aged 14 years or less, only 74 (89,2%) of these children were confirmed (by the observation of scar/road to health card) to have been vaccinated with BCG.

6.2 HISTORY OF TB AND OTHER DISEASES IN MACASSAR CAMP

6.2.1 There were 9 Camp residents who had a records of TB in the past and records of only 1 PTB case could not be located.

6.2.2 There were 229 (77,4%) residents who claimed not to have had any diseases/illness which is believed to be a risk factor for TB (table 31). Studies by Strebel et al have mentioned certain diseases to be risk factor for TB.[28]

6.3 MALNUTRITION

6.3.1 There were 50 (19,8%) children and adults found to be malnourished amongst the Camp residents (based on categories of HAZ, WAZ or WHZ, with Z-score cut-off point <-2 and adults based on BMI. Refer to tables 32a and 32b.

6.3.2 There was a significant difference (p = 0,040) between the malnourished males and females. There were 7 (10,4%) children less than 15 years who were malnourished. Refer to Appendixes E8a, E8b and tables 32a, 32b.

6.4 MISCARRIAGES AND INFANT DEATHS

There were 16 (14,3%) women who had reported having had one or more miscarriages in their fertile years. An average of 1,9 infant (less than

1 year) deaths were reported by 26 females during their fertile years. Refer to table 33.

6.5 **SMOKING HABITS**

6.5.1 There were 150 (65,8%) residents who were smokers. Refer to table 34, figure 43 and 44.

6.5.2 The prevalence of smoking amongst males (78,8%) was significantly greater (p <0,005) amongst females (54,8%) especially in the age groups for 13-19 and 20-34 years. A significant difference (p = 0,034) between the males and females also existed with regards to heavy and mild smokers. Refer to figures 43, 44 and table 34).

6.6 **ALCOHOL CONSUMPTION**

6.6.1 Only 86 (37,7%) Camp residents regarded themselves as teetotalers. Here too, there was a significant difference (p <0,001)) between the 81 (57%) males, who were consumers of alcoholic beverages as compared to 61 (43%) females. Refer to table 35 and figure 45.

6.6.2 Not only did males consume significantly more alcoholic beverages as compared to the females but there was a significantly greater number of males categorized as heavy drinkers (p = 0,003). Refer to figure 45, table 35 and 36.

6.7 RESIDENTS KNOWLEDGE OF AND ATTITUDES TO TB

6.7.1 Significantly ($p < 0,005$) more females than males identified the cause of TB as due to "germs." Refer to table 46.

6.7.2 The overall TB knowledge of both sexes was good, (based on a scoring system described in the chapter 2). More than 75% residents obtained a score of 2 or more (table 47).

6.7.3 Females scored significantly higher ($p = 0,002$) than the males with regard to the score 4 (which was categorized as the best score). Refer to figure 47.

6.7.4 The survey also showed that 94 (41,2%) Camp residents identified cough as the first symptom/ sign of PTB and only 55 (24,1%) residents stated the first sign/symptom of PTB to be weight loss.

6.7.5 A significant ($p = 0,017$) majority of 92 (40,4%) respondents stated the correct time duration that it would take to cure a TB patient (table 37).

6.7.6 There were 108 (47,4%) residents who responded that tablets/medication were used for the treatment TB. Correct response to this question was significant ($p = 0,004$). Refer to table 38.

[IV] This study has also shown from the Mantoux test results that:

7 PREVALENCE OF TB INFECTION

7.1 A total of 67 (32,1%) Camp residents were Mantoux positive. Refer to figure 48, table 39 and 42.

7.2 A total number of 29 (13,9%) vesiculations were observed on reading the Mantoux. On observation of the 29 vesiculations it was noted that 12 (41,4%) were females. Difference based on gender was not significant.

7.3 There were 13 (6,2%), vesiculations observed on residents who were not significantly Mantoux positive.

7.4 The group with the highest annual risk of infection (ARI) were those residents in the age group 20-29 years with a 1,8% ARI. And the residents in the age groups of 0-9 years and 60 years and older had an ARI of 0,4% and 0,6% respectively (table 42).

7.5 RISK FACTORS FOR TB INFECTION

7.5.1 No association could be attributed to the appearance of vesiculations in 29 (13,9%) residents with regards to TB household contacts below PFSL and all 29 residents with vesiculations were not malnourished.

7.5.2 Also no association could be found between those who were Mantoux positive and who consumed alcohol (figure 51).

7.5.3 No association was noted among those who were Mantoux positives and who had one or more illness/ disease as compared to those residents who had no record of illness/disease as listed in table 31.

7.5.4 This study has shown the various risk factors attributed to TB infection.[29a-29n] Table 54, tabulates these risk factors for TB infection ie Mantoux positive versus exposure to various risk factors (Appendix E7).

7.5.4.1 Residents partaking in regular sport/exercise was associated with protection against TB infection and may have contributed to at least 47% (1-0,53) reduction in TB infection (Mantoux positives).

7.5.4.2 The risk factors were: gender; females were greater at being infected than the males (RR = 1,55, 95% CI 1,02-2,36, p = 0,035, AR = 0,136 AR% = 33,3 and n = 209). This observations were even more pronounced in females aged less than 15 years (RR = 4,77, 95% CI 1,18-19,27, p = 0,009, AR = 0,360, AR% = 79,0 and n = 43).

7.5.4.3 Household contacts with TB patients was confirmed as a risk factor (RR = 1,98, 95% CI 1,27-3,08, p = 0,007, AR = 0,269, AR% = 49,5 and n = 196). Again the observation were more pronounced in the

household contacts with TB patients aged less than 15 years (RR = 3,25, 95% CI 1,45-7,27, p = 0,027, AR = 0,519, AR% = 69,2 and n = 43).

7.5.4.4 The combination of poor ventilation and overcrowding was established as another risk factor for infection (RR = 2,82, 95% CI 1,12-7,06, p = 0,018, AR = 0,284, AR% = 64,5 and n = 57).

7.5.4.5 Poor nutritional intake based on the PFSL was confirmed as a risk factor for TB infection (RR = 1,92, 95% CI 1,13-3,26, p = 0,009, AR = 0,502, AR% = 47,9 and n = 209).

7.5.4.6 Occupational status (unskilled worker versus the partly skilled worker) was yet another risk factor identified for TB infection. (RR = 2,68, 95% CI 1,13-6,35, p = 0,012, AR = 0,233, AR% = 62,7 and n = 114).

7.5.4.7 Not surprisingly females who had experienced 1 or miscarriages were at risk of being infected (RR = 2,38, 95% CI 1,28-4,42, p = 0,023, AR = 0,386, AR% = 88,7 and n = 66).

[V] This study has also shown by comparing the Mantoux to the TB ELISA test results that:

8 INFECTION RATE OF TB BASED ON MANTOUX VERSUS ELISA

8.1 No relationship could be found between the results of Mantoux test and the TB ELISA test. Thus the

positive test results were not in agreement. A total of 67 (32,1%) positive results were found by the Mantoux test as compared to the total of 19 (9,1%) positive results by the TB ELISA tests. Refer to table 26.

8.2 The of Mantoux and the TB ELISA results (with regards to infection rate) were found to have poor agreement. The Kappa Statistic was calculated to be k = 0,070. Refer to tables 26, 27 and Appendix F1.

8.3 There was a poor correlation between the results of Mantoux test and the TB ELISA test. Refer to figure 40.

8.4 The negative correlation coefficient among the residents who tested both positive for Mantoux and TB ELISA tests, was is not observed when the TB ELISA reading (an outlier) was excluded. Refer to figures 41a and 41b and Appendixes F5a & F5b.

8.5 The sensitivity of TB ELISA test at 94,5% specificity, when applied to the various groups was the newly confirmed PTB cases 100%; the group known KCPTB (past PTB cases) 25%; and those that were regarded as PTB 'suspect' based on X-rays (RSPTB or RAPTB) 11,1%. Refer to table 29, 30a, 30b and 30c.

8.6 Of the 3 screening tests, the TB ELISA test could be concluded to be the superior test as compared to the X-ray screening and Mantoux tests, based on the predictive value of 20,0% (highest among the 3 screening tests); positive predictive value of 45,5%, (also highest among the 3 screening tests); sensitivity of 100% and a specificity of 94,5%.

[VI] **The data collected and the results of the various tests which were confirmed by the doctor at the clinic, of the newly confirmed PTB cases have shown:**

9 **TB DIAGNOSIS**

9.1 **X-RAYS (RADIOGRAPHY)**

9.1.1 Of the 275 mass miniature chest X-rays taken in the mobile unit only 5 were confirmed to be new PTB cases, yielding a 1,8% rate of 'picking up' positive cases with the use of X-ray (table 51).

9.1.2 Of the 21 mass miniature chest X-rays reported as RSPTB, 2 (9,5%) were confirmed to be PTB cases and 8 of the RAPTB reported mass miniature chest X-rays, 4 (50%) were confirmed to be PTB cases (1 of these 4 TB cases was a relapse PTB case). And of the 29 mass miniature chest X-rays (comprising of 21 RSPTB and 8 RAPTB) 6 (20,7%) were confirmed PTB cases (table 52).

9.2 SPUTUM MICROSCOPY AND CULTURE

9.2.1 There were 5 (15,6%) Camp residents who were confirmed to be new PTB cases on microscopy. Sputum of the 32 suspect cases, comprising of mass miniature X-rays reported as 18 RSPTB, 6 RAPTB, 3 OP and 5 SP, (2 residents reported as RAPTB and 3 as RSPTB could not be traced and were excluded from further tests), were examined microscopically (table 53).

9.2.2 If one excludes the 3 residents reported as OP and 5 SP (from the suspect group of 32), then the percentage of the 5 PTB cases confirmed is 20,8%. based on the mass miniature X-ray reports.

9.2.3 Of the 5 microscopy confirmed PTB cases, 4 (80%) had a microscopy of ++, indicating a high degree of infectiousness. The results of microbiological analysis of the sputum can be seen in table 53.

9.2.4 Of the 32 sputa (mini chest X-ray suspects), cultured, 6 (18,8%) yielded a positive culture for *Mycobacterium tuberculosis* confirming active PTB. Refer to table 53.

9.2.5 If one excludes the 3 residents reported as OP and 5 SP (from the suspect group of 32), then from the 24 sputa cultured, 6 (25%%) sputa yielded a positive culture for *Mycobacterium tuberculosis* thus confirming active PTB (table 53).

9.3 **PREVALENCE OF PTB DUE TO ACTIVE CASE FINDING**

Hence in 1988, 5 new cases of PTB were confirmed in Macassar Camp (excluding the 1 relapse PTB case). The incidence of PTB (due to active case finding) in this population was calculated to be 1 355 per 100 000 in 1988.

[B] DISCUSSION

1 **THE ORIGIN AND DEVELOPMENT OF TB**

(a) **THE EVOLUTIONARY/MUTATION THEORY**

This theory is based on the fact the two organisms bear close genetic similarities and that *Mycobacterium bovis* is pathogenic to cattle and Man whereas *Mycobacterium tuberculosis* is pathogenic to humans and non-pathogenic to cattle.

Finally it has been suggested that humans may have been infected by *Mycobacterium tuberculosis* in the Neolithic period which correlates with the time when cattle were domesticated.[1,2]

The origin of *Mycobacterium tuberculosis*, is of more than pure academic interest. Valuable lessons relating to the development of pathogenicity and its relation to the immune system may be learnt. Errors in the past efforts to control TB should

also be noted as successes. A review of TB in the historical perspective, further emphasizes the need to maintain valuable intellectual traditions of the past.[44]

(b) **THE TWO PROPONENTS OF THE ORIGIN OF TB ARE:**

--- and the proponents of the 'Virgin Soil' Population theory.

--- the proponents of the 'Non-Virgin Soil' Population theory

The 'Virgin Soil' Population theory is based on the theory that a population that has had very little or no exposure to TB and the industrial way of life has had no time to acquire resistance. Thus this population is termed the 'Virgin Population' and the belief is that this population is more susceptible to TB. Then there are others that believe that the history of TB in South Africa is due to the same set of political and economic factors that had shaped the history of TB in the West or developed countries. The proponents of the 'Non-Virgin' Population theory also believe that the uniqueness of the South African TB experience is focussed in the manner in which the alignment of the changing political and economic policy evolved into legislative law.[3,4.4,5,6,7]

Both theories seem to have valid arguments in support of their respective theories.

What is now significant is not where the disease originated or who is right, but where is the disease going to and what is being done to halt the spread of this disease.

2 TB EPIDEMIC A WORLDWIDE PHENOMENON

(a) THE CYCLIC NATURE OF TB EPIDEMIC

The cyclic nature of TB can be followed from the 16th century in England when TB reached epidemic proportions and peaked in the mid-18th century (with the advent of the Industrial Revolution). By the early part of the 19th century this epidemic reached its peak in Western Europe and the East Coast of North America.

It eventually reached Eastern Europe and South America by late 19th century and is currently raging unabated in Asia and Africa and many of the developing countries.

TB has been with Man since antiquity and the TB epidemic has been a worldwide phenomena which is currently raging in the developing countries especially in Africa and Asia. The full impact of dual infection (HIV/TB) has as yet not been felt

in Southern Africa.[14,15] With the advent of the HIV epidemic the TB epidemic stands poised to re-emerge in developed countries as well.

(b) **TB EPIDEMIC IN SOUTH AFRICA**

The increase of TB notifications in South Africa since 1988, could possibly be attributed to one or more of the following reasons:

* A real increase in TB case (reasons as yet still not clear)
* Infectious individuals not on treatment resulting in the spread of infection
* Unrest in many parts of the country may have resulted in: - stress
  - disruption of routine services
* increasing prevalence of HIV infection in the country
* large portion (21% in 1988) whose treatment was incomplete
* higher incidence in TB could be artificial

One of the most important contributing factors for the spread of TB epidemic has been stated to be: overcrowding, squalor and rapid urbanization and industrialization amongst the most susceptible and oppressed people.[45,46]

(c) TB IN THE WESTERN CAPE

The increase in TB in the Western Cape has been noted especially among the Coloured population in contrast to the notable downward trend in the rest of South Africa.[23] Alarm bells of a pending TB epidemic in the Western Cape, had already rung at least as far back as 1983. From figure 28, it will be noted that there has been a gradual increase in the incidence of TB amongst the Coloureds of South Africa (1971-1981). By 1983 the incidence rate was 402 per 100 000 (Coloureds) and since then there has been a steep increase.

Exactly why this epidemic rages on unabated in the Western Cape and why there are high TB notification rates amongst Coloureds remains to this day a mystery. Many theories have been put forward but none with any conclusive evidence. One of the theories put forward as the reason for the TB epidemic has been attributed to the rapid urbanization, poor housing and overcrowding.[28]

However, one theory that may need further investigation is a common environmental factor such as environmental pollutants since the TB notification rates are the highest for all the race groups in the Western Cape when compared to

the TB notification rates with the respective race groups of all other health regions in South Africa.

(d) **TB IN MACASSAR**

It can be concluded TB in Macassar is a serious problem. Refer to figures 30, 35 and 36 and Appendix E6). The incidence of PTB in 1986 and 1987 exceeded that of W/RGN and the WCHR and the TB notification rate of all forms in Macassar in 1989 was the second highest rate in a Coloured area in the WCHR. Thirdly, deaths due to TB in Macassar had reached a peak in 1988, with a rate of 21,82 per 100 000 population.

3 **TB IN MACASSAR CAMP**

(a) **HISTORY OF TB IN MACASSAR CAMP**

TB data of residents in Macassar Camp (1972-1987), prior to this study being undertaken, suggested that there was cause for concern. Firstly records as indicated in tables 16, 17 and 18, showed that some of the workers employed at the Food Factory in Macassar, had a history of TB. Secondly all of the 8 past TB cases in Macassar Camp, were recorded to be TB culture positive and only one was AFB sputum negative.

(b) **RISK FACTORS FOR TB INFECTION IN MACASSAR CAMP:**

Based on the results of this study, risk factors for TB infection (Mantoux positive), were gender (females <15 years), contact with a TB patient within a household, poor ventilation miscarriage and overcrowding, Primary Food Subsistence Level (rating <1,5) and unskilled work. The results of a study in figure 52 gives further credence to the result established in this study (table 54).

The relative risk of TB infection of female residents was 1,55 (with p = 0,035, n = 209 and a 95% CI of 1,02 - 2,36) as compared to the male residents. The risk attributed to TB infection in this study based on gender (female) was calculated to be 136 per 100 000. Being a female resident increased the chances of TB infection by 33,3%%.

The risk of TB infection increased amongst female residents aged 14 years or less. The relative risk of TB infection of female residents aged 14 years or less was 4,77 (with p = 0,009, n = 43 and a 95% CI of 1,18 - 19,27) as compared to the male residents aged 14 year or less. The risk attributed to TB infection in female residents aged 14 years or less was calculated to be 360 per 100 000. Being a female resident aged <=14 years increased the chances of TB infection by 79,3%.

(i) **CLOSE TB CONTACTS**

Of the 8 sputa of confirmed PTB index cases examined microscopically, there were 7 microscopy positive smears. The level of organisms in the microscopic field was recorded as: 2 sputum as +, 2 sputum as ++, and the remaining 3 sputum as +++ (table 18). Of the 5 microscopy confirmed PTB cases, 4 (80%) had a microscopy of ++, indicating a high degree of infectiousness and that the patient may have had active TB for quite some time thus increasing the vulnerability of the contacts.

These results indicate a high level of infectiousness in these patients and the late diagnosis (case finding) increases the level of transmission of the tubercle bacillus (infection). Thus this could have contributed to the high prevalence of infection in this community. Refer to table 18.

Smear positive individuals infect approximately 10 persons or more per year and remain infectious for 2 years or more. Earlier studies have estimated that 33-70% infected subjects are infected by the close contacts of active PTB sufferers and that 8% of these contacts have active TB.[32,47] This study has shown that 54,2% of the close contacts

were infected and that 75% of the children aged 14 or less years who were in close contact were infected (tables 40 and 41).

Already TB accounts for more than 61% of all notifiable diseases in South Africa,[23] and the fact that the findings in this study, (with regards to infection of close contacts), concurs with earlier studies further stresses the vital need to improve active and passive case finding with rapid diagnostic methods. The TB mortality figures are a good reflection of the failure of case finding and/or case holding.[48] TB is responsible for 6-8 % of all deaths. The indications are that TB mortality figures in South Africa far exceed the estimated 20 deaths per day.[38,39] The prevalence of PTB due to active case finding in Macassar Camp (1988) was found to be 1 355 per 100 000 (table 53).

The relative risk of TB infection of a resident residing in a household with TB patient was 1,98 (with p = 0,007, n = 196 and a 95% CI of 1,27 - 3,08) as compared to a resident not residing with TB patient in the household. The risk attributed to TB infection in this study due TB household contacts was calculated to be 269 per 100 000.

Contact of a resident with a TB patient within the respective household increased the chances of TB infection by 49,5%.

The risk of TB infection increased amongst the residents aged 14 years or less residing in a household with TB patient. The relative risk of TB infection of a resident aged 14 year or less and residing in a household with TB patient was 3,25 (with p = 0,027, n = 43 and a 95% CI of 1,45 - 7,27) as compared to a resident aged 14 year or less and not residing with TB patient in the household. The risk attributed to TB infection in this study due TB household contacts aged 14 years or less was calculated to be 519 per 100 000. Contact of a resident aged 14 years or less with a TB patient within the respective household increased the chances of TB infection by 69,2%.

(ii) **OVERCROWDING AND VENTILATION**

As in earlier studies,[55] overcrowding was confirmed to be a risk factor like many of the risk factors listed in table 54 and illustrated in figure 52. Thus it can be stated unequivocally that the Camp has risk factors for TB such as poor housing and overcrowding (high density). An earlier study in 1986, had found an average number

of 1,7 Coloureds per habitable room in the Cape Town area.[23] In this study the average number of persons per habitable room was calculated to be 2,5 in Macassar.

Furthermore, a combination of variables such as overcrowding and poor ventilation was proved in this study, to render residents highly vulnerable to being infected. This added to the fact that only 28,3% of the dwelling units in the Camp were not crowded and in addition that 26,7% of the dwelling units were confirmed to have poor ventilation adds a serious risk of infection and developing active TB. Refer to table 19, 20, 30d, 43, 54, 56, and figure 37.

The relative risk of TB infection of a resident residing in a household and subjected to overcrowding and poor ventilation was 2,82 (with p = 0,018, n = 57 and a 95% CI of 1,12 - 7,06) as compared to a resident not being subjected to overcrowding and poor ventilation in the respective household. The risk of TB infection in this study attributed to overcrowding and poor ventilation was calculated to be 284 per 100 000. The resident exposed to overcrowding and poor ventilation within the respective household increased the chances of TB infection by 64,5%

The study of TB amongst a population grouped by income and population density in New York in 1970 in figure 52 further confirms the point that overcrowding is a important contributing factor for TB infection.

(iii) **TB KNOWLEDGE**

The overall TB knowledge score can be rated as good since 76% of the Camp residents obtained a TB knowledge score between 2-4 points. The females scored significantly higher ($p = 0,02$) than their male counterparts. Refer to figure 47. The reason for this phenomena could possibly attributed to the fact that females play a role as care givers and have a higher frequency of clinic visits (accompanying children to the clinic), resulting probably in greater exposure to TB education. Although the overall TB knowledge in Macassar Camp was good, a constant review of the overall effectiveness of TB education and awareness programme would be necessary. However, TB knowledge alone is insufficient. TB knowledge should be accompanied by motivation for the person concerned to seek medical help at the earliest suspicion of TB. This motivation can be enhanced by community participation and cooperation.

To sum up the vital role that an 'aggressive' education and awareness programme in the management and control of TB could play in the community, by the community, for the community, is succinctly elucidated by Bullock:[56] "The greatest resources available to any organization, are the human ingenuity, experience and loyalty it can draw on. Any investment put into tapping these by education, by securing active participation and with it the commitment of those working in any enterprise to its success, will produce greater returns than piling up investments in sites, buildings and equipment."

With a growing level of stigma attached to TB due to ignorance and attitudes (which is likely to be exacerbated by its association with HIV/AIDS), sufficient knowledge would be a vital protective shield against unreasoning prejudices. This point is aptly summed up by Franklin D Rooseveldt when he stated:[57] "Knowledge, that is, education in its true sense, is our best protection against unreasoning, prejudiced and panic-making fear whether engendered by special interest groups, illiberal minorities or panic stricken leaders."

(iv) **MALNUTRITION AND PFSL**

When the 37 adults that were malnourished (based

on BMI values) were added to the 2 malnourished children (based on the HAZ Z-Scores), 18,8% residents in the Camp were malnourished. Refer to table 46a and 47a. This means approximately 1 in 5 residents in the Camp are at risk of being infected due to malnutrition. Although previous studies have shown risk of infection due to malnutrition [50,51,52,53,54], this study was not able to show a relationship between Mantoux positive residents and those that were determined to be malnourished (table 12 and Appendix E3). The risk from malnutrition will probably not contribute to infection but rather to the disease after infection.

On categorizing all the children that were malnourished, based on the HAZ, WAZ or WHZ values as well as those adults that were malnourished (BMI values), there was no significant difference ($p = 0,19$) between those that were Mantoux positive and malnourished as compared to those that were not malnourished and Mantoux negative. Thus a total of 42 (20,2%) residents could be regarded as malnourished (table 47b).

The reason for this could possibly be that a malnourished status impairs the immune system to the extent that it would give a false negative

result to the Mantoux test. It is for this reason that possibly the PFSL values could be a more accurate means of determining the susceptibility of the resident to infection based on the poor nutritional intake just prior to the impairment of the immune system due to malnutrition.

The relative risk of TB infection of a resident with a PFSL value less than 1,5 was 1,92 (with p = 0,009, n = 209 and a 95% CI of 1,13 - 3,26) as compared to a resident with a PFSL of 1,5 or more. The risk attributed to TB infection in this study due to values below PFSL was calculated to be 502 per 100 000. The resident a PFSL below 1,5 increased the chances of TB infection by 47,9%

(v) **ALCOHOL AND SMOKING**

Smoking and alcohol have in previous studies been associated with TB.[62] Although no difference could be shown between the teetotallers and those consuming alcohol with regards to risk of infection, it would appear that heavy drinkers are more susceptible to infection.

Firstly there was a considerable decrease in the p value (not significant) amongst those that were teetotallers as compared to those that were consumers of alcohol when those that were Mantoux

positive were matched with those that were Mantoux negative. Secondly, more than 50% of the teetotallers had Mantoux readings in the range of 0-9,9 mm. Thirdly the trend of those consuming alcohol in the range of 1-50 ml and 51-100 ml (in a 2 week period) was more or less the same with regards to the Mantoux readings, but those residents consuming 101 ml or more alcohol had a marked increase in the 10,0-14,9 mm Mantoux readings (figure 51).

The reason why no association could be shown with regards to infection amongst the Camp residents, based on the Mantoux results and alcohol consumption could be due to the masking effect of alcohol which is believed to depress the immune system. The association of alcohol consumption and risk of infection requires further in depth research, with a larger sample size.

The prevalence of smoking amongst males was significantly greater (p=0,005) than the females but there was also a significant difference between gender with regards to the number of cigarettes smoked daily. Furthermore, this significant difference in prevalence of smoking was noted amongst the males and females in the age groups of 13-19 and 20-34 years but not in the age

groups of 35-44, 45-54 and 65 years or older. Refer to figure 44 and table 34. There was no significant difference in the number of Mantoux positives with regards to smokers and non-smokers Refer to table 49.

(vi) **MISCARRIAGES**

A disturbingly high number of 16 (14,3%) females aged 17 to 63 years reported one or more miscarriage in their fertile years. Rumours in the Camp indicated that women were inducing miscarriages due to unwanted pregnancies. However, whether the miscarriages were 'natural' or induced, this adds a tremendous amount of stress on the women who has miscarried making her extremely vulnerable to infection due to her lowered immune status.

This study has shown that amongst those women who had 1 or more miscarriages, the outcome of Mantoux results was significantly affected. When Mantoux positive females were matched with females who were Mantoux negative there was a significant difference ($p = 0,023$) in the percentage (tables 33 and 44) of those who were Mantoux positive and who had 1 or more miscarriages in comparison to those who were Mantoux negative and did not have a miscarriage.

The relative risk of TB infection of a female resident with a miscarriage was 2,38 (with p = 0,023, n = 66 and a 95% CI of 1,28 - 4,42) as compared to a female resident with a history of no miscarriage. The risk for TB infection in this study attributed to miscarriage was calculated to be 386 per 100 000. The female resident with a history of a miscarriage increased the chances of TB infection by 57,9%.

(vii) **SPORT/EXERCISE**

An interesting phenomena was noted among the residents aged 13 years or older where there was a significant difference (p = 0,025) between the 10 (20%) Mantoux positive residents participated in sport/exercise at least once a week as compared to 40 (80,3%) Mantoux negative residents who participated in sport/exercise at least once a week (table 45). The relative risk was 0,53 with 95% confidence interval of 0,29 - 0,97. This result suggests that when one is 'exposed' to sport/exercise, chances are less (RR <1) of being Mantoux positive (infected) than those that were 'not exposed' to regular sport/exercise. Thus regular sport/exercise would appear to have some sort of protective effect in reducing the level of TB infection by at least 47% (1-0,53).

Could this be because sport/exercise improves circulation thus enhancing the immune status of the residents exposed to regular sport/exercise?

(viii) **OCCUPATIONAL STATUS AND EMPLOYMENT**

Dubos has very aptly expressed the major determinant of the TB epidemic:[61] "TB was perhaps the first penalty that the capitalistic society had to pay for the ruthless exploitation of labour."

There were 12,6% residents unemployed in the Camp (table 23). Unemployment poses a serious health risk not only to the potential worker but also their dependents and if the worker is employed and the worker is unable to cope and lacks social support she/he could succumb to potentially pathogenic infections.[63,64]

Furthermore WHO[65] has made distinction between what is regarded as 'occupational' and 'work related diseases.' In this context, work related TB would be, TB contracted in the course of employment where the infecting agent is not inherently present but it is rather because the worker works under stressful, poor environmental conditions and lives at a low socioeconomic level.

The TB incidence rates varied in different categories of industry and occupations (table 15) and studies have shown that the TB rates were highest in companies which had higher proportions of labourers.[66] The majority 95 (65,%) of the workers were partly skilled or unskilled labourers It came as no surprise that 37,2% of the unskilled workers were infected as compared to the 13,9% skilled workers. This difference was significant. Refer to table 50.

The relative risk of TB infection of the unskilled worker was 2,68 (with p = 0,012, n = 114 and a 95% CI of 1,13 - 6,35) as compared to the partly skilled worker. The risk attributed to TB infection in this study due to occupational status (unskilled worker) was calculated to be 233 per 100 000. The worker who was unskilled increased the chances of TB infection by 62,7%.

When the risk factors listed in table 54, were cross tabulated with the responses (the same risk factors) of the newly confirmed PTB in Macassar Camp, it was noted that all the PTB patients listed in table 53 responded in the affirmative to these risk factors in excess of 50% Refer to table 55 which tabulates the association of risk factors with confirmed PTB cases of Macassar Camp.

(ix) ANNUAL RISK OF INFECTION (ARI)

The overall annual risk of infection (ARI) in Macassar Camp population was estimated to be 1,8 (all ages), with 1,8 the highest in the age group of 20-29 years. In the children aged 0-9 years and 10-14 years the annual risk of infection was 0,4 and 1,2 respectively. These figures are far higher than the goals set out by Tuberculosis Programme in South Africa (TBCP) in 1979. The goal of TBCP was to reduce the risk of TB infection to 0,3% and below for all population groups in the Republic of South Africa, which is in line the international recommendations on TB control policy.[67]

The risk of developing disease is higher during the first year of infection and declines over a period of 7 years or more.[32,33] The lifetime risk of developing active TB on being infected has been estimated to be 5-15% in the privileged communities and 40-50% in the deprived communities.[35,36] It has been estimated that the annual risk of dual infected TB/HIV individuals, to develop active TB, varies between 5-8%, with a cumulative risk of more than 30%.[31]

The vesiculations observed on reading the Mantoux test were noted in those that were significantly

Mantoux positive as well as those that were negative. Presence of vesiculation is believed to be as result of the continual exposure to the tubercule bacillus at various times. However, no association could be detected between the presence of vesiculations with regards to poor ventilation TB contacts, overcrowding or PFSL . It was also unclear to why 13 (44,8%) residents displayed vesiculations but were negative to Mantoux test, since all of these 29 residents with vesiculations were determined not to be malnourished.

(x) **DIAGNOSTICS (TB ELISA TEST)**

There was no correlation between the results of Mantoux and TB ELISA tests (figure 40), regardless whether a person was Mantoux positive or negative. This could be attributed to the fact that the TB ELISA test is specific with regards to the antigen and antibody reaction, whereas the Mantoux test consists of PPD (a mixture of proteins). However, the reason/s why there was a negative correlation (figure 41) for all the those tested positive for both Mantoux and TB ELISA tests is due to the fact TB ELISA reading which is an outlier. Refer to figures 41a and 41b and Appendixes F5a and F5b. However, the Mantoux test when compared to the TB ELISA Tests, based on the Kappa Statistic which

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is: k = 0,070, with regards to the results indicating infection rate has shown to have a poor agreement as can be seen in table 27.

The TB ELISA test was shown to be 100% sensitive at 94,5% specificity for the 5 newly confirmed PTB cases (figure 42), but this should be noted with caution because the number of newly diagnosed PTB cases is small (n = 5) and these case were found not in a clinic/hospital setting but due to active case finding during this study. It should also be noted that the sensitivity decreases with regards to other groupings (figure 42 and table 29).

From the screening tests for TB (table 55) the TB ELISA gave the highest predictive value of 20,0% (identify true positives in a population), as compared to the X-rays screening with 16,4%, and the Mantoux test with 2,6%. However, in the TB ELISA test, 45,5% yielded true positive cases (TB cases), whereas the X-rays yielded 25,0% and the Mantoux test yielded only 4,5%. Thus, of the 3 screening tests, the TB ELISA test could be concluded to be the superior test (as compared to the X-ray screening and Mantoux tests), based on the predictive value of 20,0%, positive predictive value of 45,5%, sensitivity of 100% and the specificity of 94,5% (tables 30a,b,c and 56).

Hence, the TB ELISA alone cannot be used as a sole diagnostic tool for the diagnosis and confirmation of TB (tables 28, 29, 30a,b,c). The fact that TB ELISA cannot be used as a sole diagnostic tool for TB has been also confirmed by Hussey et al.[68] Thus a positive TB ELISA would indeed indicate a high degree of suspicion of the patient having developed TB, but this would have to require further confirmation.

The 'ideal' screening test for TB, besides having a high degree of sensitivity, specificity, predictive value and positive predictive value and positive, needs to be fast easy to form (user friendly) and inexpensive. In addition to these criteria for a TB screening test, it would be an added plus for the test if it would be able to:

- differentiate past illness to active disease
- differentiate TB to MOTTS
- detect pre-illness infection
- useful in patient monitoring (compliance)
- useful for population screening

4 ECONOMY, HEALTH AND TB

The 1914 TB Commission made a glaring admission that the reason for the overall decline in the TB prevalence and mortality rates amongst the Whites

(while on the increase amongst the other ethnic groups), was an improved standard of living.[45b] An obvious modern day example of how the economy impacts on TB and health can be seen in the case of Taiwan and Cuba

The Taiwan 'Economic Miracle' shifted TB, (the third leading cause of death in the country in 1952) to the eleventh leading cause of death in 1989. Refer to table 56a. Taiwan has also one of the lowest unemployment rates (less that 1%) in the world and has shortage of unskilled labour and thus is dependent on 'imported unskilled labour'. A similar trend in the elimination of TB can be noted in Cuba. Ironically, the political ideology of Taiwan is capitalist, whereas in Cuba it is socialist, yet both countries have introduced a health system that has now become the envy of the world. What made the difference?

Perhaps the answer to this question has been very aptly contextualised by Dr Hiroshi Nakajima (The Director General of WHO),[69] "The conditions under which people live have a vital influence on the state of health. In fact health cannot be achieved or sustained without a supportive environment. An enlightened public is an essential

prerequisite for health supportive action. All societal forces must be mobilized to create conditions that will enable people to live healthy lives."

TB was the third leading cause of death in Taiwan in 1952 and by 1989, TB was listed as the eleventh cause of death (table 56b). What has caused this change? The 'Taiwan Economic Miracle'? The disease profile (1952) was very similar to that of Blacks in South Africa whereas in 1989 the disease profile of the same population (Taiwanese) is similar to the Whites of South Africa. Refer to table 56b.

In brief, the South African TB problem besides all other facets of health in this country will not change, in fact it will deteriorate, until adequate standards of living are guaranteed for everyone and a national system of Primary Health Care and preventative medicine has been established. The American Association for Advancement of Science in its report (1990) entitled Apartheid Medicine: Health and Human Rights in South Africa states:

"Any measures short of shared and fully representative political power will fail to

correct the serious discrepancies in economic and social status, education, housing, access to health services, basic nutrition, and public health programs which create the marked differences in health conditions among the racial groups in South Africa."[49]

[5] **THE MANAGEMENT AND CONTROL OF TB**

Any programme devised to reduce the spread of TB is doomed to failure if it does not take cognizance of the nature of the socioeconomic problems of South Africa. A TB control programme, without realizing and linking these facts in its efforts to control this TB epidemic, would be an exercise in futility.

This point of view is endorsed by Olsen:[59] "In order to get at the root of tuberculosis it is necessary to understand where its springs arise. From some crater in the depths of society, among the most wretched poverty and misery, unemployment and imbecility, the fountain of tuberculosis infection is thrown up through the community and seizes upon all who are susceptible. Until these social evils can be got under control, we shall never be quite free from tuberculosis. Until then we shall not deserve to be..."

To state that the current TB epidemic in South Africa is a product of social, political and economic conditions would be delving into the realms of academic rhetoric. For these 'products' are but symptoms of a more fundamental political and economic transformation that have been linked to industrial capitalism especially in developing countries and South Africa is no exception.[60] The answer to why then does TB remain such a serious problem in South Africa, lies in the understanding of these transformations.

Thus the Management of TB should be via health centers available at every community center and should have:[58]

(i) an integrated health care facility with a TB programme incorporated

(ii) and a means of rapid diagnosis treatment and prevention.

As for the formation of an integrated health care facility with a TB Programme incorporated, the possible areas in which the health authorities could direct their resources in a concerted effort to combat TB would be:

(a) The chain of transmission from the infected persons to the uninfected persons should be interrupted, identified and treated.

(b) National Tuberculin Registry should be introduced to monitor ARI and the prevalence of infection.

(c) Research findings and a national disease network surveillance system to be implemented.

(d) Therapy to be made effective and increase patient compliance (adherence). Patients should present themselves at clinics earlier. And this could be achieved by introducing an effective TB education campaign in the community in conjunction with the civic organization and unions

(e) TB Educational and awareness programmes should be reviewed with emphasis placed in motivating community at large to seek medical help at the earliest onset or suspicion of TB. More emphasis on behaviour modification with regards to TB.

(f) The TB awareness/education programmes should be targeted within the:

- homes by regular house visits
- clinics by introduction of a regular structured programme in the waiting rooms
- workplace (should be made compulsory)
- schools & tertiary institutions (include in curricula) and nursery schools (form of a games)
- nursing colleges & public health schools
- old age homes, orphanages & other institutions
- civic organizations & unions
- Non Governmental Organizations (NGO)

(g) Health Personnel --- Training of the Health Care Workers should be ongoing process with regular refresher courses and feedback and evaluation

(h) Efficiency of the Laboratory --- to be improved standardized, organized and expanded on a national basis. To introduce rapid, efficient, cost effective and accurate diagnostic test for TB

(i) Case findings and Case Holdings presently are inadequate. The channel of communications should be kept open and improved.

(j) Proper utilization of inadequate and limited resources with regular checks balances prevent wastage and duplication (transparent) should and must be incorporated into the health programme.

(k) A review of a National Health System Scheme (similar to that of Canada and Sweden) to be reviewed to for implementation into the South African context.

(l) Address the socioeconomic problems by empowering the underprivileged communities.

As for the screening and diagnostic methods for TB, the current tests are cumbersome, expensive and time consuming and not very accurate. The confirmation TB diagnosis takes up to 3-6 weeks. Thus there is a dire need for more rapid, accurate, inexpensive tests to identify patients

with TB (especially PTB and TB meningitis) cannot be over emphasized. The TB ELISA in this study has demonstrated all 5 PTB patients in Macassar Camp, with a 100% sensitivity at 94,5% specificity and a predictive value 45,6%. Thus the TB ELISA could be effectively used as a TB screening test as well a rapid TB diagnostic test.

Furthermore the current Enzyme-Immunoassay (EIA) Kit currently being evaluated (based on a similar principle as the TB ELISA test), is believed to be highly specific for the detection of human IgG level in response to infection with *Mycobacterium tuberculosis*. This due to the fact that the test utilizes two highly purified antigens. The first antigen is present in all members of *Mycobacterium* and the second antigen is highly specific 38 kDa antigen derived from *Mycobacterium tuberculosis* is coated to the microtitration wells.[70] The 38 kDa antigen has been reported to as the single most important antigen for the serodiagnosis of TB. If the EIA Kit proves to be as successful as the TB ELISA test, then it raises exciting possibilities in the area of rapid, accurate and inexpensive TB screening test and TB diagnosis (especially if the test is able to distinguish asymptomatic and active TB cases), for future studies in TB especially HIV/TB patients.

CHAPTER 8
EPILOGUE

"What can I wish to the youth of my country who devote themselves to Science? Firstly, gradualness. About this most important condition of fruitful scientific work I can never speak without emotion. Gradualness, gradualness, gradualness ... never begin the subsequent without mastering the preceding ... But do not become the archivist of facts. Try to penetrate the secret of their occurrence, persistently searching for the laws which govern them. Secondly, modesty ... do not allow haughtiness to take you in possession. Due to that you will be obstinate where it is necessary to agree, you will refuse useful and friendly help, you will lose your objectiveness. Thirdly, passion. Remember that Science demands from a man all his life. If you had two lives that would not be enough for you. Be passionate in your work and searching."

Pavlov, IP (1936)
"Bequest to Academic Youth".
Science, 83, 369.

CHAPTER 8

EPILOGUE

On Friday April 23, 1993 the World Health Organization declared TB a global public health emergency.[1] Has the nascent new South Africa acknowledged this fact? Recent reports from the World Trade Center in Johannesburg (months prior to the first democratic elections in South Africa), indicated that discussions dealt exclusively with political matters without much consideration of any other vital issues such as the collapse of the health services due to wasteful fragmentation and incompetent administration.[2]

The worldwide resurgence of TB in the wake of HIV pandemic accompanied by an alarming increase in multi-drug resistance (MDR-TB), high mortality rate, lengthy and expensive treatment (poor rate of success), does not auger well for the future of the new South Africa. The new South Africa will be ill-prepared to deal with these problems unless immediate and drastic action is implemented for the management and control of TB on a nationwide basis. Failure to do so would result in the new South Africa facing a disaster and a national emergency of the magnitude not yet imaginable.

Under the influence of HIV, and other infections, the development and progression of numerous diseases will increase. In 1993, the Center for Disease Control (Atlanta) and Prevention (CDC) expanded its definition of AIDS to include those infected with HIV who also had a severely suppressed immune system, TB, recurrent pneumonia or invasive cervical cancer.[6]

With more than half the South African population harbouring the dormant *Mycobacterium tuberculosis*, the number of TB cases will increase as has been noted since 1986 when the effects of AIDS were first noted in South Africa. The problem will further be compounded by patients receiving irregular or inadequate TB treatment, resulting in the increase of MDR-TB patients. Pressures at hospitals/clinics will result in patients with the resistant *Mycobacterium tuberculosis* not being treated with the proper supervision thus further spreading the infection and paving the way for this deadly TB epidemic.

The Department of Health recently made public a new TB policy and strategy document developed by the National TB advisory group.[3] The aim of this new policy is effectively to decrease the spread and incidence of TB. However, it must be bourne in

mind that no matter how committed the department may be to the implementation of this new policy, without the full cooperation and participation of the community as well as the immediate implementation of this policy, this programme will be doomed to failure.

If TB is indeed curable and preventable with controlled measures in place in South Africa, (the new TB policy and strategy document developed by the National TB advisory group, is confirmation of this fact), then how come TB is on the rise even before the effects AIDS/HIV pandemic could be observed in this country? This question in the past has been posed by Dr P B Fourie.[4] "Since the disease (TB) is totally curable and the available measures are sufficient to combat the disease effectively, the natural course of the epidemic can be altered to a rapid decline. Why then does the problem remain such a serious one?"

Does the answer lie perhaps to the fact that we have been concentrating for too long on the effects of TB rather than the cause of this disease? For too long we have focused and placed our faith on medical science to solve our health problems (especially TB), in the face of adverse social economic conditions.[5] It is perhaps

unlikely that medical science alone is able to halt the onslaught of the TB epidemic, which in fact is a socioeconomic related disease.

This being the case, will the powers that be in the new South Africa, change the direction and emphasis towards combating and eradicating the root cause of this disease (TB), as has been the case in Cuba and Taiwan? Failure to "deliver the goods" by means of the RDP, will only exacerbate this national emergency in the making.

Can the new government of the day allow this holocaust to take place? In one sense we are fortunate in knowing enough, early enough to prevent this holocaust. But the time to act is now? Our silence and inaction on this imminent national emergency would be tantamount to being accomplice to the holocaust.

Perhaps an example from the pages of history (1847) with regards to epidemics, infection, prevention and control, can best explain our attitude, denial of an epidemic as well as complacency in the face of an imminent epidemic of an unprecedented magnitude. Even with the facts, data, statistics, knowledge of infection and disease, methods of prevention and control, ... at

hand, we still remain switched to the 'self denial' mode, because the solution to the problem may not be the acceptable norm of the day.

A valuable lesson from the mistakes of the past with regards to epidemics, infection, prevention and control, could be learnt from Semmelweis who stated:[7] "When I look upon the past, I can only dispel the sadness which falls upon me by gazing into that happy future when the infection will be banished. But if it is not vouchsafed to me to look upon that happy time with my own eyes ... the conviction that such a time must inevitably sooner or later arrive will cheer my dying hour."

Semmelweis, postulated that a disease was transmitted by medical teachers and students coming from the postmortem room. He instituted a strict routine for washing of hands in chlorinated lime before examining patients. As a result of this procedure the mortality from puerperal fever in the obstetric clinic in the General Hospital in Vienna fell immediately from 12% to 3% and later to almost 1%. Although his doctrine was well received by certain quarters, many opposed his ideas which were considered revolutionary at the time, because it was felt that the idea that incriminated obstetricians as carriers of death

was totally unacceptable and this resulted in his post not being renewed.

He proceeded to Budapest where he introduced these methods with the same degree of success, but there too his doctrine was opposed, even by Virchow. Semmelweis, was not even able to 'sell' his idea in his book entitled 'Etiology' and further attempts by him to have his doctrine accepted were met with ridicule.

Tragically, he was institutionalized in a lunatic asylum in 1865, where ironically, a few days after entering the asylum he died from an infected wound he received in a finger during his last gynecological operation. Thus he became a victim of the very infection to which he devoted his whole life in its prevention. He however, died with the faith that his doctrine would ultimately prevail.

About a decade later Tarnie and Pasteur in France and Lister in England forced the world reluctantly to recognize that what Semmelweis had taught was correct. Who knows how many lives Semmelweis could have saved had his doctrine been accepted more than a decade earlier?

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"The Man of Science appears to be the only man who has something to say just now — and the only man who does not know how to say it."

Sir James Barrie

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(a) Table 39
(b) Table 40 & 41
(c) Figure 49 & 50
(d) Table 43

[23] Chapter 6 of this thesis
(e) Table 16 & 17
(f) Table 19
(g) Table 20
(h) Table 21
(i) Table 22
(j) Table 23
(k) Table 30
(l) Table 44
(m) Table 46a, 46b, & 46c
(n) Table 47

CHAPTER 7

CONCLUSIONS AND DISCUSSIONS

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(b) Table 40 & 41
(c) Figure 49 & 50
(d) Table 43
(e) Table 16 & 17
(f) Table 19
(g) Table 20
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(i) Table 22
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CHAPTER 8

EPILOGUE

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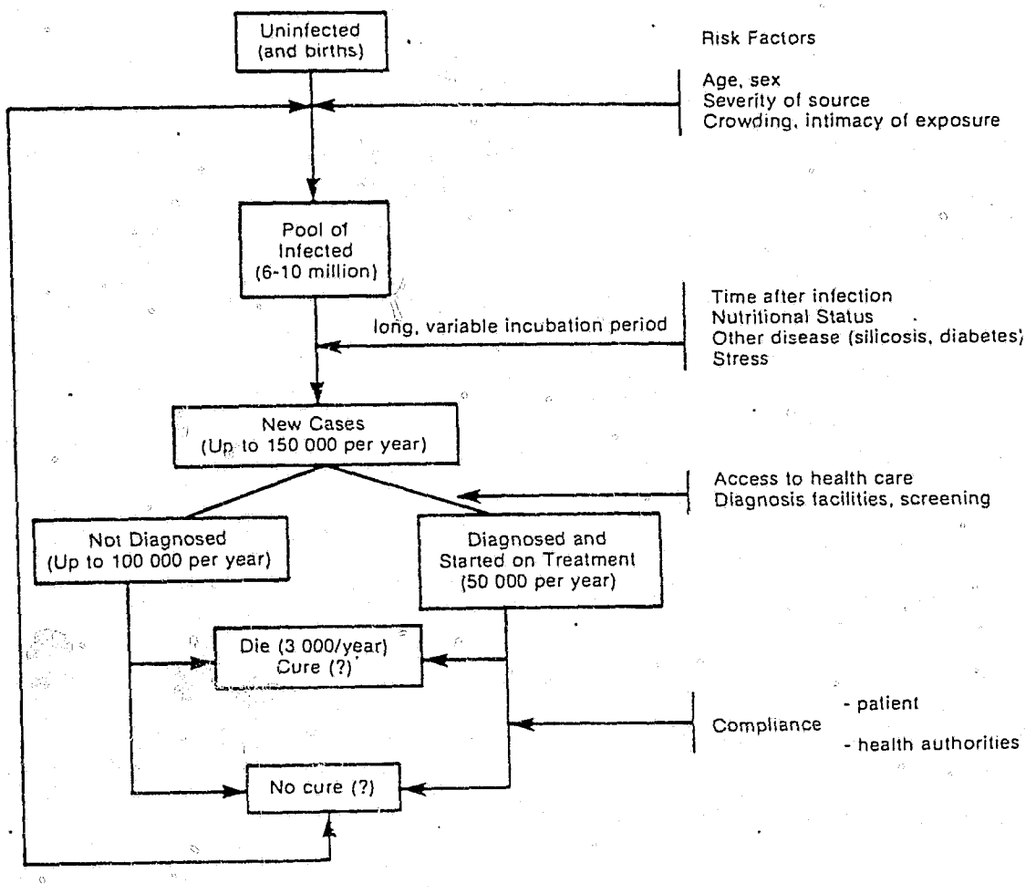
APPENDIXES

"A picture may instantly present,
what a book could set forth,
only in a hundred pages."

Ivan Serzeyevich Turgenev

APPENDIX A1

SIMPLIFIED MODEL OF TUBERCULOSIS



SOURCE:

Medical Research Council, Technical Report No 1, May 1987, Review of South African Mortality (1984) Institute of Biostatistics

APPENDIX A2

PAST TB CASES (KCPTB) OF FACTORY WORKERS AT FOOD FACTORY IN MACASSAR (1981 1986).

TABLE I

RECORD OF PAST TB CASES OF FACTORY WORKERS AT FOOD FACTORY IN MACASSAR

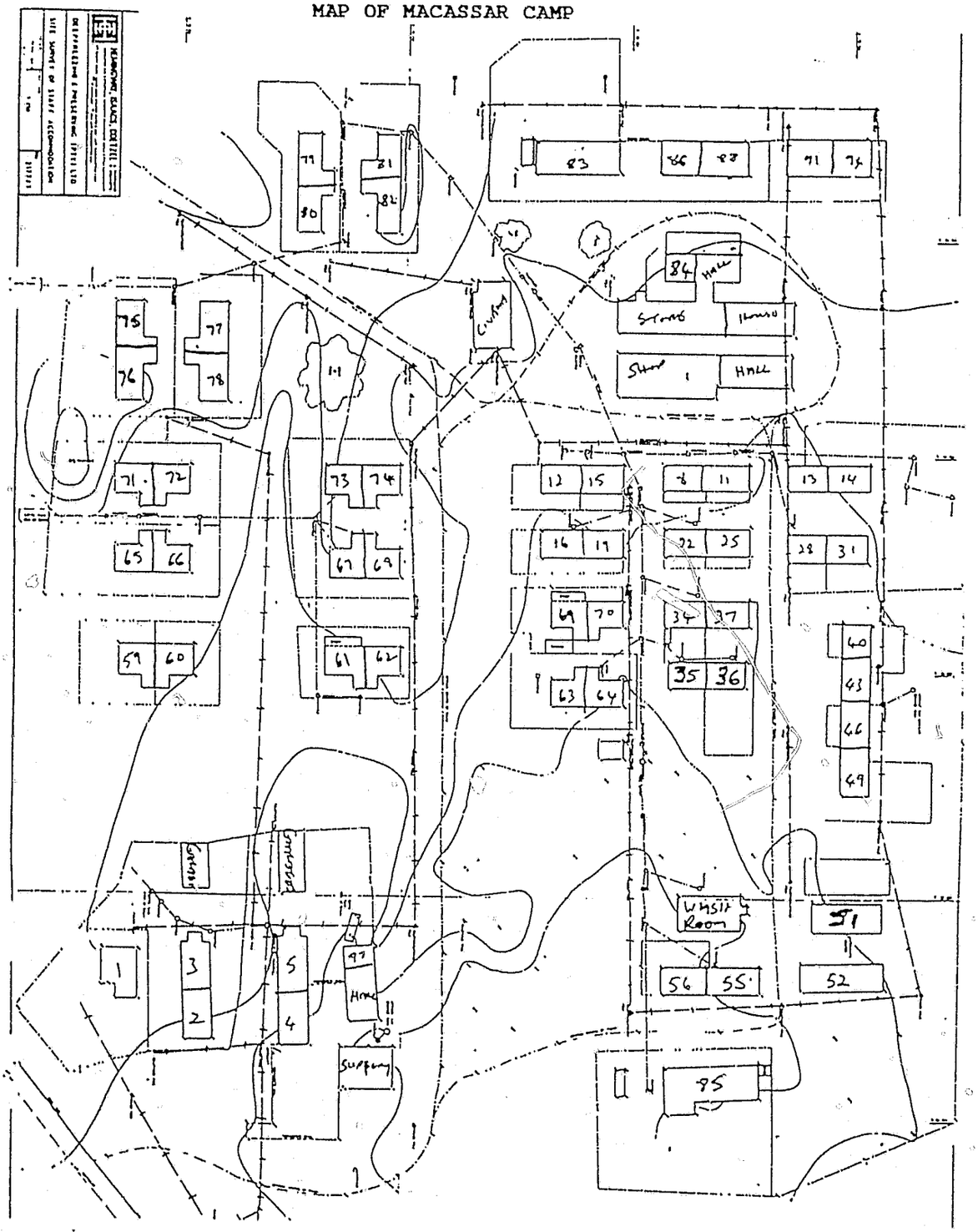
Patient Number	Sex	Date of Report	Staff Status
1	F	??/05/81	(Permanent Staff)
2	F	02/09/81	(Permanent Staff)
3	F	15/09/81	(Permanent Staff)
4	F	18/03/82	(Permanent Staff)
5	M	14/04/82	(Permanent Staff)
6	F	27/05/82	(Temporary Staff)
7	F	01/06/82	(Temporary Staff)
8	M	25/10/82	(Permanent Staff)
9	F	03/09/83	(Temporary Staff)
10	F	04/10/83	(Temporary Staff)
11	M	11/04/84	(Temporary Staff)
12	F	01/08/84	(Temporary Staff)
13	F	05/09/84	(Temporary Staff)
14	F	??/??/84	(Permanent Staff)
15	F	??/??/84	(Permanent Staff)
16	M	11/07/85	(Permanent Staff)
17	M	07/09/85	(Temporary Staff)
18	M	11/04/86	(Temporary Staff)
19	F	18/07/86	(Temporary Staff)
20	F	18/07/86	(Temporary Staff)
21	F	01/08/86	(Temporary Staff)
22	F	22/08/86	(Temporary Staff)

SOURCE:

Mohammed A, Epidemiological study of factory workers' use of the First Aid room in the Food Factory. 1986 - unpublished report to the Management of Food Factory.

APPENDIX B1

MAP OF MACASSAR CAMP



APPENDIX B2

HOUSEHOLD QUESTIONNAIRE

VOORWOORD TOT HUISEHOUDELIKE VRAELYS

Ek gaan U 'n paar vrae vra aangaande TB en ander gesondheids aspekte. Beantwoord asseblief, ja of nee, waar moontlik.

Ek verseker U dat persoonlike beondehede as streng vertroulik beskou sal word.

HUISHOUDELIKE VRAELYS

VOORWOORD:

Ek wil u graag 'n paar vrae stel aangaande T.B. en ander gesondheids aspekte. Beantwoord asseblief ja of nee, waar moontlik. Ek verseker u dat persoonlike besonderhede as streng vetroulik beskou sal word.

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NAAM VAN ONDERHOUDVOERDER: .....

KAART NR.: ..... 1 ☐ 2 ☐☐☐☐☐

DATUM VAN ONDERHOUD: .....

1) HUIS NOMMER ☐☐ 3 ☐☐☐

2) NAAM VAN PERSOON WAT HIERDIE VRAELYS BEANTWOORD .....
(d.i. mees ingeligte persoon rakend huishoudelike sake) ☐ 11 ☐

3) VERWANTSKAP VAN RESPONDENT TOT HUISHOUDING
..... ☐ 12 ☐

4) TIPE GEBOU
1. HUIS
2. KOSHUIS
3. ANDER
☐ 13 ☐

5) IS U TOILET
1. BINNE
2. BUITE
3. BEIDE
☐ 14 ☐

6) AANTAL KAMERS IN HUIS/KOSHUIS
(Uitgesluite toilet, badkamer en kombuis) ☐☐ 15 ☐☐

7) TOTALE AANTAL PERSONE WAT HUIS/KOSHUIS BEWOON ☐☐ 17 ☐☐

8) WAT IS DIE TOTALE INKOMSTE PER WEEK? ☐☐☐ 19 ☐☐☐☐

9) HOEVEEL WORD AAN KRUIDENIERSWARE SPANDEER? ☐☐☐ 23 ☐☐☐

0) VLOEROPPERVLAKTE VAN SLAAPKAMERS

a) _____ 26 ☐☐☐

b) _____ 29 ☐☐☐

c) _____ 32 ☐☐☐

d) _____ 35 ☐☐☐

e) _____ 38 ☐☐☐

1) OOPMAAKBARE AREA VAN VENSTERS IN KAMERS

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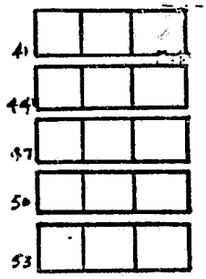
a) _____

b) _____

c) _____

d) _____

e) _____



2) OPSOMMING VAN PERSONE WAT HUIS/KOSHUIS BEWOON

	PERSOON NOMMER	NAAM	GESLAG	OUDERDOM	BEROEPSTATUS	WAAR WERKSAAM/ SKOOL
A						
B						
C						
D						
E						
F						
G						
H						
I						
J						

APPENDIX B3

PERSONAL QUESTIONNAIRE

VOORWOORD TOT HUISEHOUDELIKE VRAELYS

Ek gaan U 'n paar vrae vra aangaande TB en ander gesondheids aspekte. Beantwoord asseblief, ja of nee waar moontlik.

Ek verseker U dat persoonlike beondehede as streng vertroulik beskou sal word

PERSOONLIKE VRAELYS

AAM VAN VRAESTELLER: .....

AART NR.: .....

)ATUM VAN ONDERHOUD: .....

;AAM VAN RESPONDENT: .....

.) HUISNOMMER

2) PERSOONSNOMMER

3) GESLAG

1) OUDERDOM .....

5) IS DIE PERSOON SELF IN STAAT OM TE ANTWOORD?
JA
NEE

6) INDIEN NEE, HOEKOM NIE? .....

WIE SAL ANTWOORD NAMENS DIE RESPONDENT?
(VERWANTSKAP) .....

7) HUWELIKS STATUS:
1. GETROUD
2. GESKEI
3. VERVREEMD
4. WEDUWEE/WEWENAAR
5. SAAMLEEF
6. ENKEL

8) BEROEP STATUS:
1. HUISVROU
2. STUDENT
3. WERKEND
4. WERKLOOS
5. AFGETREE

9) INDIEN WERKEND:
1. PERMANENT
2. TYDELIK
3. DEELTYDS

.0) WAAR WERK U? .....

.1) HOE LANK WOON U IN DIE KAMP? (JARE) .....

.2) BESKRYF DIE TIPE WERK WAT U DOEN .....
.....

.3) WAT IS U POSISIE OF RANG BY DIE WERK? .....
.....

:4) HOE LANK BEKLEE U HIERDIE POSISIE? .....

15) HOE LANK IS U INDIENS VAN DIE MAATSKAPPY?(JARE) [ ][ ] - [ ][ ] 34

16) INDIEN VROULIK: 360

AAN HOEVEEL KINDERS HET U GEBOORTE GESKENK? [ ][ ] 36 [ ][ ]

HOEVEEL MISKRAME HET U GEHAD? [ ] 38 [ ]

HET ENIGE VAN U KINDERS GESTERF VOOR DIE EERSTE VERJAARSDAG? JA [ ]

NEE [ ] 39 [ ]

INDIEN JA, HOEVEEL? [ ] 40 [ ]

17) HET U ENIGE BEHANDELING ONTVANG VIR DIE VOLGENDE SIEKTES IN DIE VERLEDE?

	JA/ NEE	TIPE GESOND-HEIDS-DIENS	WAAR	WANNEER	
HOë BLOEDDRUK					41 [ ][ ][ ][ ]
ASMA/ALLERGIE					45 [ ][ ][ ][ ]
TUBERKULOSE					49 [ ][ ][ ][ ]
SUIKERSIEKTE					53 [ ][ ][ ][ ]
MAAG/KOLON OPERASIE					57 [ ][ ][ ][ ]

18) ROOK U EEN OF MEER SIGARETTE PER DAG JA [ ]

NEE [ ] 61 [ ]

19) INDIEN JA

OP WATTER OUDERDOM HET U BEGIN ROOK? [ ][ ] 62 [ ][ ]

HOEVEEL SIGARETTE ROOK U PER DAG?

1. 1-9
2. 10-19
3. 20-29
4. 30

[ ] 64 [ ]

20) HET U IN AANHOUDENDE HOES VIR MEER AS EEN WEEK GEHAD? JA [ ]

NEE [ ] 65 [ ]

21) INDIEN JA

HET U BLOED GEHOES? JA [ ]

NEE [ ] 66 [ ]

HET U LUSTELOOS EN MOEG GEVOEL IN DIE LAASTE MAAND?

JA [ ]

NEE [ ] 67 [ ]

HET U NAGSWEET ONDERVIND IN DIE LAASTE MAAND

JA [ ]

NEE [ ] 68 [ ]

KAART NR. 1 [2]

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VIND U DAT U KLERE LOSSER AAN U SIT IN DIE LAASTE MAAND? JA ☐ NEE ☐ 2 ☐

VIND U APTYTVERLIES IN DIE LAASTE MAAND? JA ☐ NEE ☐ 3 ☐

22) HET U B.C.G. INENTING ONDERGAAN?. JA ☐ NEF ☐ WEET NIE ☐ 4 ☐

23) INDIEN JA,

BRON VAN INLIGTING:
1. LETSEL
2. KAART ☐ 5 ☐
3. ANDER BRON

24) WAS U AL OOIT VIR X-STRAAL ONDERSOEK VIR T.B.? JA ☐ NEE ☐ 6 ☐

25) INDIEN JA,

WANNEER WAS DIE LAASTE X-STRAAL ONDERSOEK?
MAAND ..... JAAR ..... 7 ☐☐☐☐

WAAR WAS DIE X-STRAAL ONDERSOEK?
..... 11 ☐☐☐☐

26) NEEM U DEEL AAN SPORT/OEFENING TEN MINSTE EEN KEER PER WEEK? JA ☐ NEE ☐ 15 ☐

27) INDIEN JA

HOE LANK NEEM U DEEL AAN SPORT/OEFENINGE GEREELD?
1. MINDER AS 1 JAAR
2. 1-2 JARE ☐ 16 ☐
3. 3-4 JARE
4. 5 JARE OF MEER

28) WAARDEUR DINK U WORD T.B. VEROORSAAK?
1. SOEN
2. KIEME
3. TE VEEL WYN ☐ 17 ☐
4. TE VEEL SIGARETTE
5. VERKEERDE TIPE KOS

29) INDIEN 'n PERSOON T.B. HET WAT DINK U IS DIE GEWONE TEKENS? ..... [ ] 18

30) WAT DINK U WORD GEBRUIK VIR DIE BEHANDELING VAN T.B.? ..... [ ] 19

31) HOE LANK DINK U NEEM DIT OM 'n PASIËNT TE BEHANDEL VOORDAT HY/SY GENEES IS (MAANDE) [ ][ ] 20

32) HET U TEN MINSTE EEN BIER, WYN OF ANDER DRANK GEDRINK IN DIE AFGELOPE JAAR

JA [ ]
NEE [ ] 22

33) INDIEN JA,

IN DIE AFGELOPE TWEE WEKE OP HOEVEEL DAE HET U GEDRINK?

1. NIKS
2. 1-4 DAE
3. 5-9 DAE
4. 10-14 DAE

[ ] 23

34) IS DIT U NORMALE HOEVEELHEID WAT U GEWOONLIK DRINK BINNE TWEE WEKE?

JA [ ]
NEE [ ] 24

35) INDIEN NEE,

WAT IS DIE GEBRUIKLIKE HOEVEELHEID ALKOHOLIESE DRANK PER DAG WAT U GEBRUIK? ..... DAE [ ] 25

36) WANNEER U NORMAALWEG DRINK, HOEVEEL DRINK U?

1. BOTTELS WYN [ ][ ] 26
2. GLASE WYN [ ][ ] 28
3. LITERS BIER [ ][ ] 30
4. DUMPIES/BLIKKIES BIER [ ][ ] 32
5. SOPIES STERK DRANK [ ][ ] 34
6. ANDER [ ][ ] 36

APPENDIX B4

LETTER OF CONSENT FROM MACASSAR CAMP RESIDENT

I, ... , resident of above mentioned Camp, hereby give my consent to Mr A Mohammed to conduct TB Research.

This will involve me (and my children under 18 years), in allowing +-5ml of blood to be drawn and to be tested for a skin allergy. Both blood and skin allergy test will be conducted by a doctor and/or nurse.

In addition to this, I will be expected to submit to a Chest X-Rays, being measured for height and weight and responding to questionnaires.

I participate in this Research Project of my own free will, aware of the minor risks and discomfort involved. I do this in the hope that the outcome of the results will in the long run be of benefit to my community. I have also been assured that any personal details of mine will be kept strictly confidential.

.....
Signature of Camp Resident

Date: .....

APPENDIX B5

FORMULA FOR THE CALCULATION OF THE ANNUAL RISK OF INFECTION (ARI) OF TB

$$ARI = \frac{2[(\text{reactions}) > \text{mode}] + [\text{reactions} = \text{mode}]}{n}$$

Where mode = cut-off point in a specific age group

n = number of persons in specific age group

SOURCE:

[1] Sutherland, I and Fayers P M. The association of the risk of infection with age. Bul Int Union Tuberc 1975; 50: 70-81

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[3] Styblo, K; Meijer, J & Sutherland, I. The transmission of tubercle bacilli. Its trend in a human population. Tuberculosis Surveillance Research Unit Report No 1. Bull Int Union Tuberc 1969; 42: 5-14.

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APPENDIX B6

ACID FAST STAINING PROCEDURE BY ZIEHL-NEELSEN (ZN) STAINING METHOD

Carbol-fuchsin: Dissolve 3,0 gm of basic fuchsin in 10.0 ml of 90% to 95 % ethanol Add 90ml of 5% aqueous solution of phenol.

Acid-alcohol: Add 3,0ml of concentrated HCL slowly to 97ml of 90% to 95% ethanol; in this order. Solution may get hot.

Methylene blue counterstain: Dissolve 0,3gm of methylene blue chloride in 100ml of distilled water.

Procedure:

1. Cover heat-fixed dried smear with a small rectangle (2x3cm) or filter paper *
2. Apply 5 to 7 drops of carbol fuchsin stain to thoroughly moisten filter paper.
3. Heat the stain-covered slide to steaming but do not allow to dry. Heating may be done by gas burner or over an electric staining rack.
4. Remove paper with forceps, rinse with water, and allow to drain.
5 Decolourize with acid-alcohol until no more stain appears in the washing (2 min)
6. Counterstain with methylene blue (1 to 2 min)
7 Rinse, drain and air dry (1 to 2 min)
8. Examine with 100x oil immersion objective. Bacilli are stained red and the background light blue.

* To avoid the transfer of the bacilli from one side to another , never blot the smears on the rack. Do not use jars for staining more than on slide at a time. Discard all the solutions that have been in contact with smears.

SOURCE:

Youmans, GP. (1979) Tuberculosis. Chapter 19, WB Saunders & Co. London.

APPENDIX B7

MICROSCOPIC ANALYSIS

AND

INTERPRETATION OF ZN SMEARS

NUMBER OF BACILLI	REPORT
0	No acid-alcohol bacilli found
1 to 2 in the the entire smear	Report number found & request additional specimen to repeat the test
3 to 9 in the entire smear	Rare, or +
10 or more in the entire smear	Few, or ++
1 or more per oil-immersion field	Numerous, or +++

SOURCE:

Youmans, GP. (1979) Tuberculosis. Chapter 19, WB Saunders & Co. London.

APPENDIX B8

IDENTIFICATION CHARACTERISTICS

OF

Mycobacterium tuberculosis

TESTS	RESULTS
Optimum isolation temperature:	37^0 C
Rate of Growth in Days:	12 - 25 days
Pigmentation outcome;	
growth in light:	buff
growth in dark:	buff
Niacin Test:	+
Nitrate Reduction:	3 to 5 +
Catalase Test:	
Semiquantitative	<40*
pH 7.0 @ 68^0 C	-
Tween 80 Hydrolysis (10 days)	-/+
Arylsulfatase (3 days)	-
Urease	+
Resistance to T2H (1ug/ml)	+
Growth on 5% NaCl	-
Iron Uptake	-

KEY TO RESULTS:

+	= 84% of strains
-/+	= 16% to 49% strains
*	= INH resistant strains may be negative

SOURCE:

Youmans, GP. (1979) Tuberculosis. Chapter 19, WB Saunders & Co. London.

APPENDIX C1

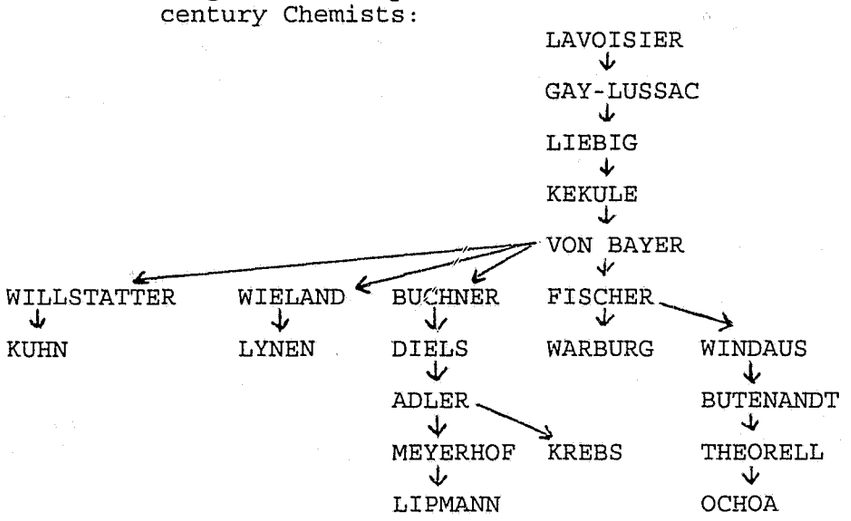
QUOTATION - THE MAKING OF A SCIENTIST

"The making of a scientist is not merely a matter of attending a course of lectures and reading books but of researching together over an extended period of a few years. In this process excellence and distinction develops if nurtured by excellence. Merely constructing a building for the so-called center of excellence does not build excellence - it has never been done in history. In the absence of someone with outstanding ability there is always a good chance that we easily come to believe that we are excellent and much better than others. Mediocre people may appear big to themselves and to others if they are surrounded by small circumstances. By the same token, big people feel dwarfed in the company of giants. From the giants of science what we learn is 'to see ourselves modestly and not to overrate ourselves with vast broadmindedness, free but disciplined imagination, great enthusiasm and deep devotion.' The most important element out of these qualities is the attitude of humility; from it flows a self-critical mind and the continuous effort to learn and to improve."

SOURCE:

Lodhi, MAK. (1987). Islamization of Attitudes & Practices in Science & Technology, Publisher: International Institute of Islamic Thought. p148

FIGURE 1: Diagrammatic Representation of 19th & 20th century Chemists:

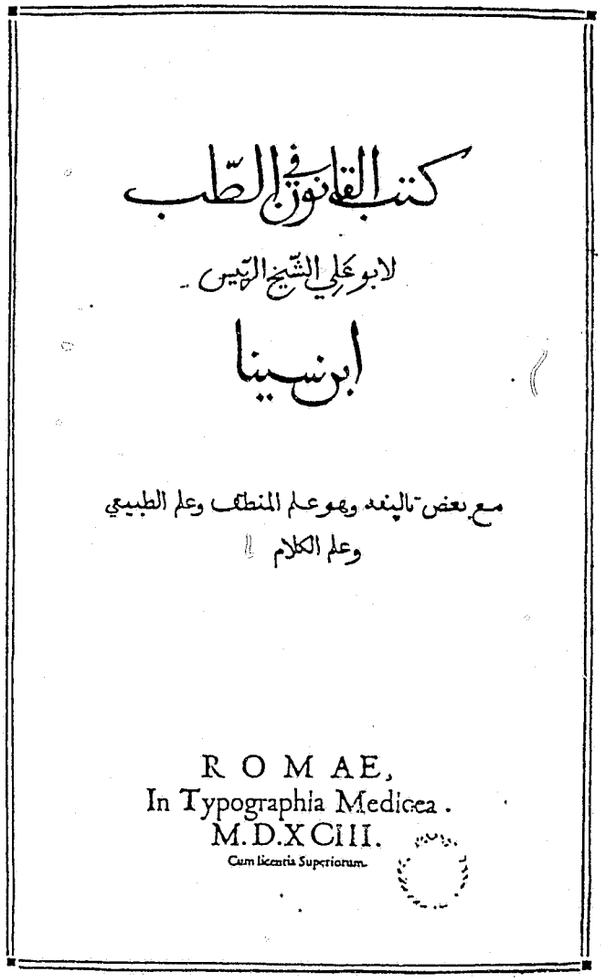


APPENDIX C2

Title-page of the Canon of Medicine by Avicenna printed in Arabic type in Rome, 1593

FIGURE 2:

Title-page of the Canon of Medicine by Avicenna printed in Arabic type in Rome, 1593


كتاب القانون في الطب

لابو علي الشيخ الرئيس

ابن سينا

مع بعض تاليفه وهو علم المنطق وعلم الطبيعي
وعلم الكلام

ROMAE,
In Typographia Medicea.
M.D.XCIII.
Cum licentia Superiorum.

SOURCE:

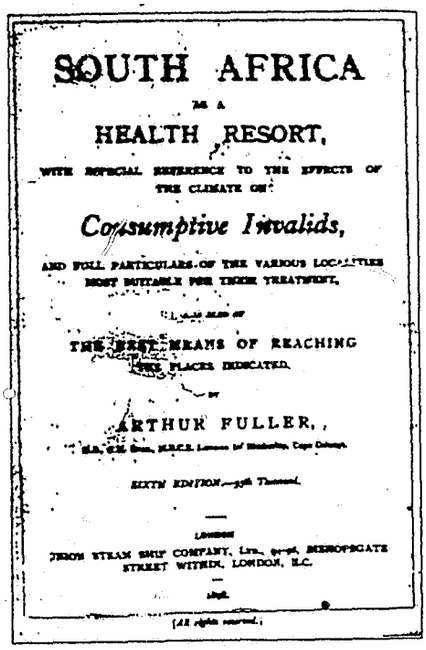
Manfred Ulmann (1978), Islamic Surveys, Islamic Medicine, Chapter 2 (The Age of the Translations), Edinburgh University Press, Edinburgh.

APPENDIX C3

Bookplate of 6th edition of the book South Africa as a health resort, published in 1898.

FIGURE 3:

Bookplate of 6th edition of the book South Africa as a health resort, published in 1898.



SOURCE:

A Century of Tuberculosis. South African Perspectives. Edited by: Coovadia, HM and Benatar, SR, Chapter 1 (History of Tuberculosis: Metcalf, C pP1-31 Oxford University Press (1991), Cape Town.

APPENDIX D1

Cumulative Distribution of Individuals Infected with Tuberculosis and HIV, 15-49 Year-Old Group, Early 1992.

REGION	HIV INFECTED (thousand)	TB INFECTED (%)	HIV/TB INFECTED	
			NUMBER (thousand)	PERCENT OF TOTAL.
Africa[1]	6500	48	3120	77.8
Americas[2]	1000	30	300	7.5
Eastern Mediterranean[1]	50	23	11	0.3
South East Asia[1] & Western Pacific[3]	1020	40	408	10.2
Europe[1] & others[4]	1550	11	170	4.2
ALL REGIONS	10 120	34	4 009	100

[1] Includes all countries of WHO region.

[2] Includes all countries of the American Region of WHO, except USA and Canada.

[3] Includes all countries of the Western Pacific Region of WHO, except Japan, Australia, New Zealand

[4] USA, Canada, Japan, Australia, New Zealand

SOURCE:

WHO/TUB/92-164, (p19)

APPENDIX D2

The average prevalence rates of tuberculosis infection in South Africa (1974-1980) according to ethnic group, geographical region and age.

Ethnic Group and Geographical Location	Age group 5–9 years Mean Age (yrs)	Pre-valence (%)	No. Tested (N)	Mean Survey Year (19...)*	Age Group 10-14 years Mean Age (yrs)	Pre-valence (%)	No. Tested (N)	Mean Survey Year (19...)*	Age Group 15-19 years Mean Age (yrs)	Pre-valence (%)	No. Tested (N)	Mean Survey year (19...)*
Black												
Highland	7,4	11,4	28 531	76,9	11,7	18,6	8 296	76,9	16,6	31,0	3 732	77,6
Lowland	7,3	21,9	8 555	76,8	11,6	31,7	2 302	76,6	16,7	40,3	1 046	76,8
Urban	7,4	14,0	32 875	76,9	11,6	20,9	7 939	77,2	16,7	33,8	3 830	77,8
Rural	6,9	12,3	4 721	76,9	12,0	22,4	2 659	76,0	16,1	30,1	948	76,0
All regions	7,4	13,9	37 086	76,9	11,7	21,4	10 598	76,9	16,6	33,1	4 778	77,4
Coloured												
Highland	7,1	7,6	8 113	76,8	11,8	14,0	3 534	77,5	16,3	19,5	1 427	77,9
Lowland	6,7	9,6	7 448	77,2	12,2	16,9	3 464	77,6	16,1	20,1	2 120	77,8
All regions	6,9	8,6	15 601	77,0	12,0	15,4	6 998	77,5	16,2	19,9	3 547	77,9
Asian												
Highland	7,0	2,5	3 618	77,5	11,6	5,7	1 680	78,9	16,3	11,1	943	78,9
Lowland	6,2	3,2	3 697	76,9	11,5	4,7	465	78,2	16,0	7,5	380	78,0
All regions	6,7	2,8	7 233	77,3	11,6	5,5	2 145	78,7	16,2	10,1	1 323	78,6
White												
Highland	7,1	0,9	11 683	77,3	12,0	1,6	9 667	77,1	16,1	3,3	5 030	77,7
Lowland	7,1	1,5	5 515	77,0	11,9	3,5	5 124	76,5	16,0	4,6	2 245	76,3
All regions	7,1	1,1	17 198	77,2	12,0	2,3	14 791	76,9	16,1	3,7	7 275	77,3

*In this column the first two figures indicate the year, the third the month – e.g. 76,9 = September 1976.

SOURCE:

Fourie, PB, (1983). The prevalence and annual rate of tuberculosis infection in South Africa. Tubercle. 64, 181-92.

APPENDIX D3

The risk of TB infection and estimated annual changes in the risk for South African children aged 5-9 years.

Survey Group	Mean Survey Year (19...)†	Mean Age (yrs)	No. Tested+	Pre-valence (%)	Risk of Infection 1985 (%)	Annual Change in risk (%)
Indian	76,8	6,7	6 148	2,7	0,1	–11,1
	81,2	6,9	2 500	1,7		
Coloured	76,6	6,9	13 713	8,3	0,9	–2,2
	81,4	6,9	4 003	7,5		
Black (inland)	75,9	7,4	23 224	11,7	0,6	–7,9
	81,3	7,4	9 855	7,8		
Black (coastal)	76,2	7,2	8 262	22,8	1,6	–6,2
	82,2	7,2	3 788	16,3		

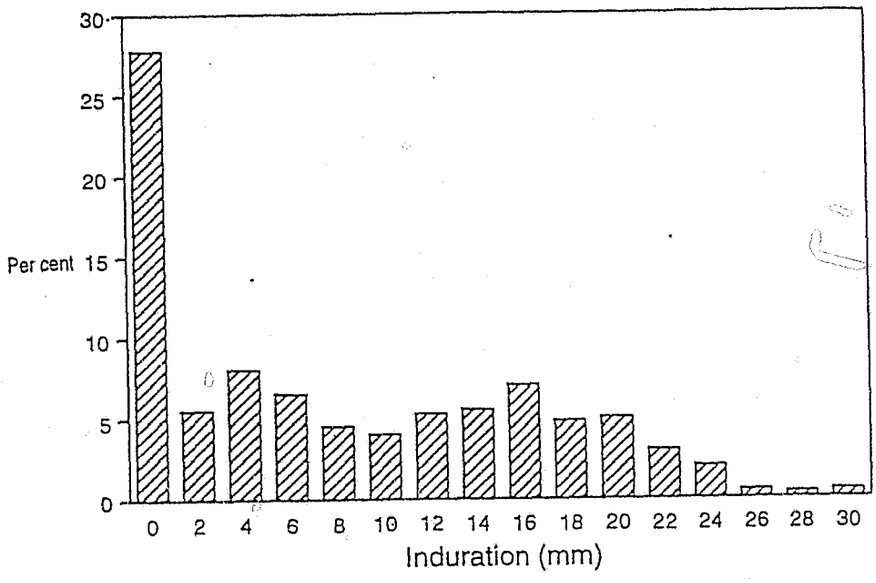
+ Children without BCG scars only.
† In this column the first two figures indicate the year, the third the month, e.g. 76,8 = August 1976

SOURCE:

Fourie, PB, (1986'. Tuberculosis prevalence and risk of infection in Southern Africa. S Afr J Sci. 82,387.

APPENDIX D4a

Tuberculin hypersensitivity patterns in black children 10-14 years old without BCG scars from Transkei and Ciskei showing the effect of non-specific (MOTT) sensitivity.

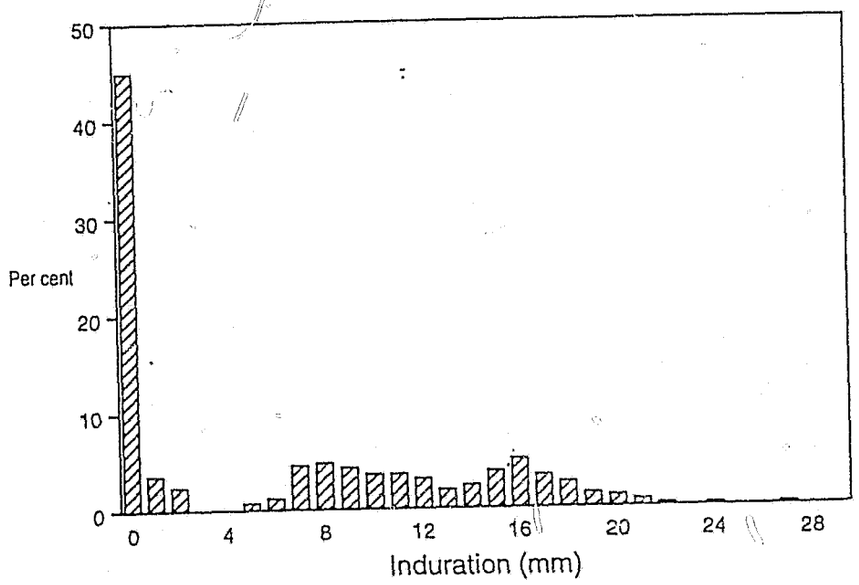


SOURCE:

Fourie, PB. (1983). The prevalence and annual rate of tuberculosis infection in South Africa. Tubercle. 64, 181-92.

APPENDIX D4b

Tuberculin hypersensitivity patterns in coloured children 5-9 years old from Cape Town who had been given BCG within the previous 12 months.



SOURCE:

Seager, JR; Felten, MK; Collins, TFB; & Kerr, J (1987). Screening successfully treated tuberculosis patients for signs of chronic respiratory failure. Paper presented at the 2nd TBRI Symposium on Tuberculosis in Southern Africa, Cape Town, 1987.

DEPARTMENT OF NATIONAL HEALTH AND POPULATION DEVELOPMENT

No. R. 327 22 February 1991

FOODSTUFFS, COSMETICS AND DISINFECTANTS ACT, 1972 (ACT No. 54 OF 1972)

ENFORCEMENT BY LOCAL AUTHORITIES.—COLIGNY AND SANNIESHOF

I, Elizabeth Hendrina Venter, Minister of National Health, hereby authorise under section 23 (1) of the Foodstuffs, Cosmetics and Disinfectants Act, 1972 (Act No. 54 of 1972), the Municipalities of Coligny and Sannieshof to enforce the relevant provisions of the said Act within their areas of jurisdiction and through their duly authorised officers.

E. H. VENTER,
Minister of National Health.

No. R. 328 22 February 1991

HEALTH ACT, 1977 (ACT No. 63 OF 1977)

DECLARATION OF MEDICAL CONDITIONS TO BE NOTIFIABLE MEDICAL CONDITIONS IN TERMS OF SECTION 45 OF THE HEALTH ACT, 1977 (ACT No. 63 OF 1977)

I, Elizabeth Hendrina Venter, Minister of National Health, acting under and by virtue of section 45 of the Health Act, 1977 (Act No. 63 of 1977)—

(a) hereby declare the medical conditions specified in the Schedule hereto to be notifiable medical conditions;

(b) hereby withdraw Government Notice No. R. 2708 of 15 December 1989.

SCHEDULE

Acute rheumatic fever;
Anthrax;
Brucellosis;
Cholera;
Congenital syphilis;
Diphtheria;
Food poisoning (outbreaks of more than four persons);
Haemorrhagic fevers of Africa (Congo fever, Dengue fever, Ebola fever, Lassa fever, Marburg fever, Rift Valley fever);
Lead poisoning;
Legionellosis;
Leprosy;
Malaria;
Measles (rubeola);
Meningococcal infections;
Paratyphoid fever;
Plague;

DEPARTEMENT VAN NASIONALE GESONDHEID EN BEVOLKINGS-ONTWIKKELING

No. R. 327 22 Februarie 1991

WET OP VOEDINGSMIDDELS, SKOONHEIDSMIDDELS EN ONTSMETTINGSMIDDELS, 1972 (WET No. 54 VAN 1972)

TOEPASSING DEUR PLAASLIKE BESTURE.—COLIGNY EN SANNIESHOF

Ek, Elizabeth Hendrina Venter, Minister van Nasionale Gesondheid, magtig hierby kragtens artikel 23 (1) van die Wet op Voedingsmiddels, Skoonheidsmiddels en Ontsmettingsmiddels, 1972 (Wet No. 54 van 1972), die Munisipaliteite van Coligny en Sannieshof om binne hulle regsgebied en deur middel van hulle behoorlik gemagtigde beamptes die toepaslike bepalings van genoemde Wet uit te voer.

E. H. VENTER,
Minister van Nasionale Gesondheid.

No. R. 328 22 Februarie 1991

WET OP GESONDHEID 1977 (WET No. 63 VAN 1977)

VERKLARING VAN MEDIESE TOESTANDE TOT AANMELDBARE MEDIESE TOESTANDE KRAGTENS ARTIKEL 45 VAN DIE WET OP GESONDHEID, 1977 (WET No. 63 VAN 1977)

Ek, Elizabeth Hendrina Venter, Minister van Nasionale Gesondheid, handelende kragtens artikel 45 van die Wet op Gesondheid, 1977 (Wet No. 63 van 1977)—

(a) verklaar hierby die mediese toestande vermeld in die bylae hiervan tot aanmeldbare mediese toestande;

(b) herroep hierby Goewermentskennisgewing No. R. 2708 van 15 Desember 1989.

BYLAE

Akute rumatiese koors;
Antraks;
Brusellose;
Cholera;
Difterie;
Geelkoors;
Hemoragiese koorssiektes van Afrika (Denguekoors, Ebolakoors, Kongokoors, Lassakoors, Marburgkoors, Slenkdalkoors);
Hondsdolheid (spesifiseer of menslike geval of menslike kontak);
Kongenitale sifilis;
Legionellose;
Leprose;
Loodvergiftiging;
Malaria;
Masels (rubeola);

Poisoning from any agricultural or stock remedy registered in terms of the Fertilizers, Farm Feeds, Agricultural Remedies and Stock Remedies Act, 1947 (Act No. 36 of 1947);
Poliomyelitis;
Rabies (specify whether human case or human contact);
Smallpox and any smallpox-like disease, excluding chickenpox;
Tetanus;
Tetanus neonatorum;
Trachoma;
Tuberculosis—
(i) pulmonary and other forms, except cases diagnosed solely on the basis of clinical signs and symptoms;
(ii) in the case of any child younger than 5 years with a significant reaction following tuberculin testing;
Typhoid fever;
Typhus fever (epidemic louse-borne typhus fever, endemic flea-borne typhus fever);
Viral hepatitis A, B, non-A, non-B and undifferentiated;
Yellow fever.

E. H. VENTER,
Minister of National Health.

No. R. 329 22 February 1991

FOODSTUFFS, COSMETICS AND DISINFECTANTS ACT, 1972 (ACT No. 54 OF 1972)

REGULATIONS GOVERNING EMULSIFIERS, STABILISERS AND THICKENERS AND THE AMOUNTS THEREOF THAT FOODSTUFFS MAY CONTAIN.—AMENDMENT

The Minister of National Health has, in terms of section 15 (1) of the Foodstuffs, Cosmetics and Disinfectants Act, 1972 (Act No. 54 of 1972), made the regulations contained in the Schedule hereto.

Interested persons are invited to submit any substantiated comments on the proposed regulations, or representations they wish to make in regard thereto, to the Director-General: National Health and Population Development, Private Bag X828, Pretoria, 0001 (for the attention of the Director: Foodstuffs, Cosmetics, Disinfectants and Hazardous Substances), within three months of the date of publication of this notice.

Meningokokkale infeksies;
Paratifoïedkoors;
Pes;
Pokke en soortgelyke siektes, uitgesonderd waterpokkies;
Poliomielitis;
Tetanus;
Tetanus neonatorum;
Tifoïedkoors;
Tifuskoors (epidemiese luis-gedraagde tifuskoors, endemiese vlooi-gedraagde tifuskoors);
Tragoom;
Tuberkulose—
(i) long en ander vorme, behalwe gevalle gediagnoseer op grond van kliniese tekens en simptome alleen;
(ii) in die geval van 'n kind jonger as 5 jaar met 'n betekenisvolle reaksie na tuberkulientoetsing;
Vergiftiging weens enige landbou- of veemiddel wat kragtens die Wet op Misstowwe, Veevoedsel, Landboumiddels en Veemiddels, 1947 (Wet No. 36 van 1947), geregistreer is;
Voedselvergiftiging (uitbrekings van meer as vier persone);
Virushepatitis A, B, nie-A, nie-B en ongedifferensieerd.

E. H. VENTER,
Minister van Nasionale Gesondheid.

No. R. 329 22 Februarie 1991

WET OP VOEDINGSMIDDELS, SKOONHEIDSMIDDELS EN ONTSMETTINGSMIDDELS, 1972 (WET No. 54 VAN 1972)

REGULASIES BETREFFENDE EMULGEERMIDDELS, STABILISEERDERS EN VERDIKKERS EN DIE HOEVEELHEDE DAARVAN WAT VOEDINGSMIDDELS MAG BEVAT.—WYSIGING

Die Minister van Nasionale Gesondheid het kragtens artikel 15 (1) van die Wet op Voedingsmiddels Skoonheidsmiddels en Ontsmettingsmiddels, 1972 (Wet No. 54 van 1972), die regulasies in die Bylae hiervan vervat, uitgevaardig.

Belanghebbende persone word versoek om binne drie maande na die datum van publikasie van hierdie kennisgewing enige gemotiveerde kommentaar oor of vertoë in verband met die voorgestelde regulasies in te dien by die Direkteur-generaal: Nasionale Gesondheid en Bevolkingsontwikkeling, Privaatsak X828, Pretoria, 0001 (vir die aandag van die Direkteur: Voedsel, Kosmetika, Ontsmettingsmiddels en Gevaarhoudende Stowwe).

APPENDIX D5 - 376

List of Notifiable Medical Conditions by Department of National Health & Population Development

SOURCE: Government Gazette. No R 328

APPENDIX D6

Number of Notifications by Disease, Year and Type in South African Coloured Population, 1971-1988

1 PULMONARY TB

YEAR	NUMBER OF DEATHS	NUMBER OF CASES	C.F.R.
71	445	5657	7,8
72	435	6186	7,0
73	668	6489	10,3
74	561	7161	7,8
75	495	6850	7,2
76	547	6836	8,0
77	515	7139	7,2
78	516	7741	6,7
79	491	8312	5,9
80	499	8291	6,0
81	507	8217	6,2
82	502	9265	5,4
83	468	9647	4,9
84	535	10574	5,1
85	476	10971	4,3
86	588	11031	5,3
87	765	12020	6,4
88	622	13954	4,5
Total	9635	156341	6,2

2 TB MENINGITIS

YEAR	NUMBER OF DEATHS	NUMBER OF CASES	C.F.R.
71	30	62	48,4
72	25	57	43,9
73	34	108	35,2
74	36	85	42,4
75	40	102	39,2
76	28	73	38,4
77	34	67	50,8
78	23	57	40,4
79	29	49	59,2
80	26	48	54,2
81	22	101	21,8
82	15	60	25,0
83	23	88	26,1
84	18	71	25,4
85	17	86	19,8
86	14	79	17,7
87	24	65	36,9
88	11	61	18,0
Total	449	1319	34,0

Note: C.F.R. is the case fatality rate, given as a percentage.

SOURCE:

Epidemiological Comments, (July, 1989). 16(7):5

Table 1

Estimated Percentage Coverage per Population Group and Health Region, RSA, 1988

BCG

	ASIAN		BLACK		COLOURED		WHITE		ALL GROUPS	
DOSE	1	2	1	2	1	2	1	2	1	2
HEALTH REGION										
WESTERN CAPE										
Vaccinations	46	0	2 341	1 084	14 924	3 488	4 507	412	21 818	4 984
Target	380	380	15 220	15 220	43 918	43 918	12 755	12 755	72 273	72 273
% Coverage	12	0	15	7	34	8	35	3	30	7
NORTHERN CAPE										
Vaccinations	6	5	3 293	795	5 523	675	436	63	9 258	1 539
Target	37	37	9 897	9 897	6 617	6 617	1 883	1 883	18 434	18 434
% Coverage	16	14	33	8	83	10	23	3	50	8
EASTERN CAPE										
Vaccinations	51	47	20 106	9 637	7 974	2 177	3 216	1 360	31 388	13 221
Target	226	226	34 330	34 330	8 763	8 763	5 863	5 863	49 182	49 182
% Coverage	41	21	59	28	91	25	55	23	64	27
ORANGE FREE STATE										
Vaccinations	3	116	21 877	3 226	442	248	2 859	462	25 167	4 052
Target			58 561	58 561	1 561	1 561	5 633	5 633	65 755	65 755
% Coverage			37	6	28	16	51	8	38	6
NATAL										
Vaccinations	9 039	12 859	59 833	61 569	499	2 223	4 606	3 866	73 979	80 567
Target	13 258	13 258	35 166	35 166	2 368	2 368	9 493	9 493	60 285	60 285
% Coverage	68	97	170	175	21	94	49	41	123	134
SOUTHERN TRANSVAAL										
Vaccinations	2 285	696	77 016	19 778	4 659	1 144	34 220	7 320	118 080	28 938
Target	2 437	2 437	171 693	171 693	6 483	6 483	38 621	38 621	219 234	219 234
% Coverage	90	29	45	12	72	18	89	19	54	13
NORTHERN TRANSVAAL										
Vaccinations	178	8	16 416	2 412	161	4	3 077	187	19 832	2 611
Target	100	100	26 462	26 462	203	203	3 102	3 102	29 867	29 867
% Coverage	178	8	62	9	79	2	99	6	66	9
ALL REGIONS										
Vaccinations	11 549	13 731	209 877	98 502	34 182	10 009	52 91	13 679	293 522	135 912
Target	16 438	16 438	351 329	351 329	69 913	69 913	77 350	77 350	515 030	515 030
% Coverage	70	84	57	28	49	14	68	18	58	26

Table 2

Estimated Percentage Coverage per Population Group and Health Region, RSA, 1988

Measles

	ASIAN		BLACK		COLOURED		WHITE		ALL GROUPS	
DOSE	1	2	1	2	1	2	1	2	1	2
HEALTH REGION										
WESTERN CAPE										
Vaccinations	190	1	16 295	7 679	53 892	3 978	8 042	914	78 420	12 572
Target	380	380	15 220	15 220	43 918	43 918	12 755	12 755	72 273	72 273
% Coverage	50	0	107	50	123	9	63	7	109	17
NORTHERN CAPE										
Vaccinations	22	1	5 359	481	6 935	991	850	31	13 177	1 504
Target	37	37	9 897	9 897	6 617	6 617	1 883	1 883	18 434	18 434
% Coverage	59	3	54	5	105	15	45	2	71	8
EASTERN CAPE										
Vaccinations	99	0	25 682	2 199	10 653	550	3 971	237	40 105	2 986
Target	226	226	34 330	34 330	8 763	8 763	5 863	5 863	49 182	49 182
% Coverage	44	0	75	6	122	6	68	4	82	6
ORANGE FREE STATE										
Vaccinations	58	0	18 906	373	600	1	1 537	5	21 101	379
Target			58 561	58 561	1 561	1 561	5 633	5 633	65 755	65 755
% Coverage			32	1	38	0	27	0	32	1
NATAL										
Vaccinations	15 287	274	79 384	10 489	2 574	141	5 009	309	102 254	11 212
Target	13 258	13 258	35 166	35 166	2 368	2 368	9 493	9 493	60 285	60 285
% Coverage	115	2	226	30	109	6	53	3	170	19
SOUTHERN TRANSVAAL										
Vaccinations	2 961	407	89 055	16 798	6 602	2 337	26 702	4 770	125 320	24 322
Target	2 437	2 437	171 693	171 693	6 483	6 483	38 621	38 621	219 234	219 234
% Coverage	122	17	52	10	102	37	69	12	57	11
NORTHERN TRANSVAAL										
Vaccinations	79	17	13 804	1 268	124	18	2 134	327	16 141	1 630
Target	100	100	26 462	26 462	203	203	3 102	3 102	29 867	29 867
% Coverage	79	17	52	5	61	9	69	11	54	5
ALL REGIONS										
Vaccinations	18 696	700	248 196	39 287	81 381	8 076	48 245	6 542	396 818	54 605
Target	16 438	16 438	351 329	351 329	69 913	69 913	77 350	77 350	515 030	515 030
% Coverage	114	4	71	11	116	12	62	8	77	11

SOURCE: Epidemiological Comments, (Dec, 1989). 16(12):12

Estimated Percentage Coverage per Population Group and Health Region, RSA, 1988.

NOTIFIABLE MEDICAL CONDITIONS

Table 1 : Number of notified cases by condition and region for the reporting period January to December 1991 (as on 27/01/92)

Medical condition		Health regions of RSA								Self-governing national states										Independent national states							Other (1)	Grand Total
Code	Name	Cape East	Province West	Natal North	OFS	Transvaal South	Transvaal North	Total		Gazankulu	KaNgwane	KwaNdebele	Kwa Zulu	Lebowa	Qwa Qwa	Boplu thatswana	Ciskei	Transkei	Venda	Total								
084	Malaria	4	10	4	189	38	242	962	1449	436	690	0	1615	47	0	0	0	0	133	2923							163	4535
055	Measles	146	214	20	130	220	216	18	964	21	10	65	498	24	133	1	32	2084	13	2881							2	3847
036	Meningococcal infection	73	304	35	10	18	75	55	570	26	20	1	27	14	9	0	12	36	1	146							0	716
037	Tetanus	7	5	8	1	5	5	3	34	19	2	2	4	5	0	0	0	8	2	42							0	76
7713	Tetanus neonatorum	1	0	0	0	3	0	0	4	0	0	0	0	3	0	0	0	0	0	3							0	7
010	Tuberculosis primary	486	2459	34	0	302	45	0	3326	0	74	0	2		1	3	201	1	0	282							2	3610
011	Tuberculosis pulmonary	8558	16433	2245	2692	7523	10501	314	48266	568	575	103	6280	708	335	115	1474	517	266	10941							9	59216
013	Tuberculosis of meninges	41	113	32	24	21	31	1	263	0	3	1	20	0	1	1	9	5	0	40							0	303
010-8	Tuberculosis total	9407	19667	2379	2757	7893	11141	322	53566	571	733	104	6351	709	337	121	1828	565	278	11597							11	65174
0020	Typhoid fever	43	23	2	91	30	99	19	307	239	109	25	227	105	3	0	6	315	68	1097							0	1404
0701	Viral hep fever type A	72	295	21	222	206	481	9	1306	1	3	0	6	3	0	0	8	2	2	25							1	1332
0703	Viral hep fever type B	148	140	23	75	30	93	7	516	10	21	0	54	2	0	0	12	4	2	105							2	623
0705	Viral hep non-A non-B	1	4	0	2	0	7	1	15	0	0	0	1	0	0	0	0	0	0	1							0	16
0709	Viral hep unspecified type	12	71	0	23	24	70	11	211	32	3	3	76	17	0	0	1	98	19	249							0	460
070	Viral hep total	233	510	44	322	260	651	28	2048	43	27	3	137	22	0	0	21	104	23	380							3	2431
	Total	9914	20733	2192	3500	8467	12429	1407	58942	1357	1591	200	8859	929	482	122	1899	3112	518	19069							179	78190
(2) Estimated population (x1000)		1998	3489	780	2739	2454	8954	1130	21544	716	566	340	5399	2646	262	1735	1031	3403	523	16621								38165

Cumulative annual total of remaining notifiable conditions

022	Anthrax	0	065L	Lassa fever	0	071	Rabies	9
023	Brucellosis	13	984	Lead poisoning	4	390	Rheumatic fever	34
001	Cholera	1	040L	Legionellosis	13	0663	Rift Valley fever	0
090	Congenital syphilis	98	030	Leprosy	28	050	Smallpox	0
065D	Crimean Congo haemorrhagic fever	4	065M	Marburg fever	0	076	Trachoma	221
061	Dengue fever	0	0025	Paratyphoid fever	0	090	Typhus fever (lice)	1
032	Diphtheria	12	020	Plague	0	081	Typhus fever (ratflea)	1
065E	Ebola fever	0	989	Poisoning agric. stock remedies	127	060	Yellow fever	0
005	Food poisoning	122	045	Poliomyelitis	2			

(1) Source of infection outside the borders of RSA and national states (2) Source : Epidemiological Comments Vol 14 No 1

SOURCE: Epidemiological Comments, (Jan, 1992). 19(1):13

Notifiable Medical Conditions: Number of notified cases by condition and region for the reporting period Jan- Dec 1991

APPENDIX E1

HSRC PAMPHLET OF SOUTH AFRICA AND ITS PROVINCES (1994)

Nuwe Provinsies	Noord-Kaap	Noord-wes	Wes-Kaap	Oranje-Vrystaat	Oos-Kaap	PWV	Noord-Transvaal	Oos-Kaap	KwaZulu /Natal	Totaal
Bevolking (1994, miljoen)[2]	0,7	3,3	3,6	2,7	6,4	6,9	5,2	2,9	8,5	40,3
Gebied ('000 km^2)	358	117	131	131	172	17	122	82	93	1 223
Skoolgaande kinders (1992 of 1993, miljoen)[3]	0,2	0,9	9.8	0.7	2,1	1,4	1,8	0,7	2,1	10,7
Tersiêre studente (1991, '000)[4]	-	12.3	53,9	13,0	23,9	77,6	18,8	-	40.0	239,5
Kiesers (1994, miljoen)[5]	0,4	1,7	2,4	1.6	3,2	4,9	2,3	1,6	4,6	22,7
Setels in Nasionale vergadering[5]	7	32	42	28	56	86	40	28	81	400
Setels in provinsiale parlemente[1]	30	34	34	30	52	86	40	30	80	424

Bronne:
1. Grondwet van die Republiek van Suid-Afrika, (Wet No. 200, 1993), en Wysigingswet op die Grondwet van Suid-Afrika (Wet No. 2, 1994), SA Reserwebank, Kwartaalblad
2. Sentrale Statistiekdiens, Middeljaarramings, 1970-1994, P0302
3. Edusource Data News, Maart 1994
4. DSA in Depth, Jan/Feb 1994
5. Ramings gebaseer op die aanname dat dieselfde persentasie kiesers in elke provinsie

APPENDIX E2

Estimated Population (1992) - Western Cape (Development Region A). Based on the 1985 census

Estimated population - 1992 — *Western Cape (Development region A)*

Map Ref.	District Name	Asian	Black	Coloured	White	Total
1	Beaufort West	10	5 122	24 172	6 404	35 708
2	Bellville	752	4 831	129 013	132 896	267 492
3	Bredasdorp	4	765	15 774	5 314	21 857
4	Caledon	22	12 316	55 786	13 958	82 082
5	Calitzdorp	1	153	6 135	1 361	7 650
6	Calvinia	0	245	16 773	4 345	21 363
7	Cape	2 202	6 582	66 389	116 211	191 384
8	Ceres	7	6 066	36 945	5 695	48 713
9	Clanwilliam	2	866	22 777	5 409	29 054
10	Fraserburg	0	38	3 895	999	4 932
11	George	48	7 715	55 497	24 854	88 114
12	Goodwood	2 906	1 243	195 243	73 250	272 642
13	Heidelberg	1	422	9 935	2 294	12 652
14	Hermanus	12	2 004	11 031	7 938	20 985
15	Hopefield	2	143	6 066	2 386	8 597
16	Knysna	36	8 427	26 333	14 099	48 895
17	Kuilsriver	62	6 210	52 181	24 230	82 683
18	Ladismith	1	462	10 612	1 979	13 054
19	Laingsburg	23	375	5 730	1 289	7 417
20	Malmesbury	67	4 063	90 826	19 422	114 378
21	Montagu	37	2 607	14 831	4 028	21 503
22	Mosselbay	12	5 005	28 095	11 810	44 922
23	Murraysburg	2	901	4 549	826	6 278
24	Namakwaland	10	4 255	56 589	10 875	71 729
25	Oudtshoorn	34	4 582	51 446	15 582	71 644
26	Paarl	107	20 390	95 418	27 358	143 273
27	Piketberg	8	1 046	27 738	8 139	36 931
28	Prince Albert	0	53	7 967	1 540	9 560
29	Riversdale	2	785	18 053	7 481	26 321
30	Robertson	13	3 528	25 307	6 481	35 329
31	Simons Town	156	1 233	15 573	30 623	47 585
32	Somerset West	61	3 942	33 134	23 205	60 342
33	Stellenbosch	69	6 424	46 021	26 580	79 093
34	Strand	113	2 458	18 021	20 421	41 013
35	Sutherland	1	130	3 368	1 022	4 521
36	Swellendam	13	2 486	24 544	7 416	34 459
37	Tulbagh	5	1 596	22 210	3 517	27 328
38	Uniondale	2	599	8 878	1 332	10 811
39	Vanrhynsdorp	4	168	10 358	2 901	13 431
40	Victoria-West	5	1 746	9 253	1 809	12 813
41	Vredenburg	35	1 059	25 996	8 697	35 787
42	Vredendal	1	1 031	23 563	6 275	30 870
43	Wellington	55	773	29 709	8 313	38 850
44	Williston	0	18	3 939	1 038	4 995
45	Worcester	179	21 911	80 016	25 410	127 516
46	Wynberg	15 571	361 074	593 984	159 311	1 129 940
	Walvisbay	8	10 675	5 255	5 918	21 856
	Total	22 660	528 523	2 124 928	892 241	3 568 352

Source: Directorate: Epidemiology, based on the 1985-census

SOURCE:

Epidemiological Comments, Vol 19 (4) April 1992

APPENDIX E3

POOR NUTRITION LINKED TO INCREASED TB INCIDENCE

LIST OF REFERENCES:

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[iv] Barnes, P F; Modlin, R L; Bikle D D & Adams J S. Transpleural gradient of 1,25-dihydroxy vitamin D in tuberculous pleuritis. Journal of Clinical Investigation. 1989; 83, 1527-32.

[v] Lagrange, P H. Tuberculosis: Immunological and clinical aspects. In: Humber D P, ed. Immunological Aspects of Leprosy. Tuberculosis and Leishmaniasis. Amsterdam: 1981; Exerpta Medica, International Congress Series 574.

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APPENDIX E4

TB CASE HISTORY: THE SEFATSE FAMILY

Mr Sefatse, his wife and seven children comprised a household living on a closer settlement site. Since 1974 when they arrived in Qwa Qwa from a Free State farm, Sefatse has had intermittent labour contracts. In 1981 he secured a labour contract at Sasol II which he, in common with many others in Qwa Qwa, did not realize was not renewable. He also became ill at this time, which made it difficult to find another job. In mid-1982 he secured work in Bethlehem but after four months, he was so ill that he returned home. Diagnosed then as having TB, he had not worked since then. During 1982 Sefatse's eldest son secured a contract in Welkom. Since then his son has become seriously ill but has clung to his job because it is the only source of income to the site. The second son was removed from school in mid-1982 because his fees could not be met. He was still too young to be issued with a reference book and has been unable to find a job within Qwa Qwa. By 1983 the household was in a deep crisis. Sefatse's wife and oldest daughter had been diagnosed as suffering from pellagra and wife had suspected TB as well. A younger child had been sent home from school because she was passing out in class as result of hunger. A combination of drought and illness and hunger in the household had meant that even the small site garden was no longer in use. Sefatse and his wife said that they had exhausted the possibilities for begging or borrowing in the neighbourhood.

Owing to the vagaries of relocation neither Sefatse or his wife had any kin in Qwa Qwa on whose support they could depend. [They] expressed their anxiety for and guilt about their children and repeatedly said that they had failed as parents. 'I cannot seep at night any longer,' said Mrs Sefatse, 'because my son was so ill when he last came home [at Easter] but I sent him back to work [in Welkom] because his is the only income we have. I am forced to kill one child in order to feed the others.'

A neighbour to the household noted that she and others were appalled and ashamed of seeing Mrs Sefatse who was literally starving to death and of hearing her cries where she lay. She was, however, unable to do anything more to help as she also had no food nor money in her house. (52:15 and 24; 297:34-5)

SOURCE:
Francis, W; Rampele, M; (1989): Uprooting Poverty, The South Africa Challenge, p119, Creda Press, South Africa

1985 POPULATION					1985
LOCATION	WHITE	COLOURED	BLACK	ASIAN	TOT POP
DIV COUNCIL OF CAPE	240950	370990	274690	10880	897510
DIV COUNCIL OF STELLENBOSCH	9453	118512	13552	0	141547
DIV COUNCIL OF PAARL	10526	45741	6154	0	62151
STELLENBOSCH MUNICIPALITY	20597	20649	3346	40	45132
SOMERSET WEST MUNICIPALITY	19750	3260	360	60	23130
STRAND MUNICIPALITY	16013	14955	250	0	31221
GORDONS BAY MUNICIPALITY	0	0	0	0	0
KRAAIFONTEIN MUNICIPALITY	15100	15600	70	0	30770
BRACKENFELL MUNICIPALITY	13200	600	600	0	14400
KUILS RIVER MUNICIPALITY	11024	10040	0	0	21064
PAARL MUNICIPALITY	17261	46413	12909	0	76672
WELLINGTON MUNICIPALITY	6000	17300	70	0	23370
CAPE TOWN MUNICIPALITY	273780	566560	136186	14349	990875
TOTAL	656957	1233825	448328	25329	2364442

YEAR 1986	1986 POPULATION				1986
LOCATION	WHITE	COLOURED	BLACK	ASIAN	TOT POP
DIV COUNCIL OF CAPE	244350	382050	350700	10910	988040
DIV COUNCIL OF STELLENBOSCH	9517	122231	18582	0	150330
DIV COUNCIL OF PAARL	10649	46976	6313	0	63938
STELLENBOSCH MUNICIPALITY	21477	21271	3392	40	46180
SOMERSET WEST MUNICIPALITY	20330	3329	500	60	24210
STAND MUNICIPALITY	17807	15038	250	0	33095
GORDONS BAY MUNICIPALITY	2517	567	57	1	3142
KRAAIFONTEIN MUNICIPALITY	19700	19000	80	3	37783
BRACKENFELL MUNICIPALITY	13800	600	600	0	15000
KUILS RIVER MUNICIPALITY	12150	11050	0	0	23200
PAARL MUNICIPALITY	17411	47374	13389	0	78174
WELLINGTON MUNICIPALITY	6160	17830	70	0	24060
CAPE TOWN MUNICIPALITY	277824	561387	140652	14774	1014637
TOTAL	672722	1268694	534585	25788	2501789

YEAR 1987	1987 POPULATION				1987
LOCATION	WHITE	COLOURED	BLACK	ASIAN	TOT POP
DIV COUNCIL OF CAPE	255030	393310	299210	10970	958520
DIV COUNCIL OF STELLENBOSCH	9595	132057	18851	0	160533
DIV COUNCIL OF PAARL	10861	45385	6449	0	66695
STELLENBOSCH MUNICIPALITY	22034	22100	10158	40	54332
SOMERSET WEST MUNICIPALITY	19030	3383	560	15	22991
STAND MUNICIPALITY	15635	15511	280	0	34426
GORDONS BAY MUNICIPALITY	2691	597	82	1	3271
KRAAIFONTEIN MUNICIPALITY	19200	19700	90	0	38990
BRACKENFELL MUNICIPALITY	15000	400	600	0	16000
KUILS RIVER MUNICIPALITY	12670	12460	0	0	25130
PAARL MUNICIPALITY	17576	48425	15000	0	81001
WELLINGTON MUNICIPALITY	6300	18450	70	0	24820
CAPE TOWN MUNICIPALITY	251927	590602	315425	15212	1212166
TOTAL	690419	1311370	669505	26241	2697865

YEAR 1988	1988 POPULATION				1988
LOCATION	WHITE	COLOURED	BLACK	ASIAN	TOT POP
DIV COUNCIL OF CAPE	262690	411090	334860	12900	1021540
DIV COUNCIL OF STELLENBOSCH	9650	144100	19233	0	172983
DIV COUNCIL OF PAARL	10955	49691	6623	0	67302
STELLENBOSCH MUNICIPALITY	22744	23062	10068	40	55914
SOMERSET WEST MUNICIPALITY	19594	3437	600	18	23649
STAND MUNICIPALITY	19255	15960	280	0	35525
GORDONS BAY MUNICIPALITY	2695	611	93	1	3403
KRAAIFONTEIN MUNICIPALITY	19660	20142	109	0	39911
BRACKENFELL MUNICIPALITY	15000	650	800	0	16450
KUILS RIVER MUNICIPALITY	13011	12989	0	0	26000
PAARL MUNICIPALITY	17706	45476	15399	0	81581
WELLINGTON MUNICIPALITY	6450	19100	0	0	25550
CAPE TOWN MUNICIPALITY	283975	604209	192898	15431	1096516
TOTAL	703454	1353517	580963	28390	2666324

YEAR 1989	1989 POPULATION				1989
LOCATION	WHITE	COLOURED	BLACK	ASIAN	TOT POP
DIV COUNCIL OF CAPE	275800	438659	502140	13277	1230076
DIV COUNCIL OF STELLENBOSCH	9711	171295	19539	0	200545
DIV COUNCIL OF PAARL	11108	51251	6736	0	69125
STELLENBOSCH MUNICIPALITY	23217	23416	10213	40	56886
SOMERSET WEST MUNICIPALITY	20583	3562	707	19	24871
STAND MUNICIPALITY	20657	16899	400	0	37956
GORDONS BAY MUNICIPALITY	2798	638	101	1	3538
KRAAIFONTEIN MUNICIPALITY	21820	20587	115	0	42522
BRACKENFELL MUNICIPALITY	16000	650	800	0	17450
KUILS RIVER MUNICIPALITY	13700	13640	0	0	27340
PAARL MUNICIPALITY	17930	49892	15946	0	83768
WELLINGTON MUNICIPALITY	6604	19800	70	0	26474
CAPE TOWN MUNICIPALITY	288172	620021	194038	15888	1118119
TOTAL	728100	1430540	750805	29225	2938670

APPENDIX E5a(i)

POPULATION OF WCHSR:
VARIOUS DEMARCATED AREAS
& MACASSAR

LOCATION	1985 PULMONARY TB NOTIFICATIONS WHITE	COLOURED	BLACK	ASIAN	TOTAL NOTIFI
DIV COUNCIL OF CAPE	36	1820	1555	2	3413
DIV COUNCIL OF STELLENBOSCH	0	401	102	0	503
DIV COUNCIL OF PAARL	4	159	27	0	216
STELLENBOSCH MUNICIPALITY	2	39	29	0	70
SOMERSET WEST MUNICIPALITY	4	17	0	0	21
STRAND MUNICIPALITY	5	19	3	0	27
GORDONS BAY MUNICIPALITY	0	0	0	0	0
KRAAIFONTEIN MUNICIPALITY	1	66	5	0	72
BRACKENFELL MUNICIPALITY	1	2	9	0	12
KUILS RIVER MUNICIPALITY	1	34	0	0	35
PAARL MUNICIPALITY	3	269	104	0	376
WELLINGTON MUNICIPALITY	0	33	3	0	36
CAPE TOWN MUNICIPALITY	61	1715	1493	7	3276
TOTAL	118	4604	3330	9	8057

LOCATION	1986 PULMONARY TB NOTIFICATIONS WHITE	COLOURED	BLACK	ASIAN	TOTAL NOTIFI
DIV COUNCIL OF CAPE	31	2570	2041	5	4650
DIV COUNCIL OF STELLENBOSCH	3	448	120	0	568
DIV COUNCIL OF PAARL	1	196	37	0	233
STELLENBOSCH MUNICIPALITY	3	29	37	0	69
SOMERSET WEST MUNICIPALITY	3	26	4	0	33
STRAND MUNICIPALITY	4	62	3	0	69
GORDONS BAY MUNICIPALITY	0	0	0	0	0
KRAAIFONTEIN MUNICIPALITY	0	48	0	0	48
BRACKENFELL MUNICIPALITY	0	3	5	0	8
KUILS RIVER MUNICIPALITY	1	46	0	0	47
PAARL MUNICIPALITY	9	316	119	0	444
WELLINGTON MUNICIPALITY	1	30	0	0	31
CAPE TOWN MUNICIPALITY	51	1978	1540	8	3577
TOTAL	107	5752	3909	13	9777

LOCATION	1987 PULMONARY TB (PTB) NOTIFICATIONS WHITE	COLOURED	BLACK	ASIAN	TOTAL NOTIFI
DIV COUNCIL OF CAPE	48	2374	2219	9	4650
DIV COUNCIL OF STELLENBOSCH	3	481	117	0	598
DIV COUNCIL OF PAARL	2	223	38	0	261
STELLENBOSCH MUNICIPALITY	1	33	35	0	71
SOMERSET WEST MUNICIPALITY	9	23	8	0	31
STRAND MUNICIPALITY	4	51	3	0	54
GORDONS BAY MUNICIPALITY	0	1	0	0	1
KRAAIFONTEIN MUNICIPALITY	0	73	2	0	75
BRACKENFELL MUNICIPALITY	0	3	6	0	9
KUILS RIVER MUNICIPALITY	4	49	0	0	49
PAARL MUNICIPALITY	5	368	146	0	514
WELLINGTON MUNICIPALITY	2	31	0	0	31
CAPE TOWN MUNICIPALITY	63	1940	1685	5	3693
TOTAL	147	5650	4262	14	10073

LOCATION	1988 PULMONARY TB NOTIFICATIONS WHITE	COLOURED	BLACK	ASIAN	TOTAL NOTIFI
DIV COUNCIL OF CAPE	47	2512	2362	14	4935
DIV COUNCIL OF STELLENBOSCH	3	621	186	0	807
DIV COUNCIL OF PAARL	2	275	41	0	316
STELLENBOSCH MUNICIPALITY	2	47	61	0	110
SOMERSET WEST MUNICIPALITY	3	34	1	0	38
STRAND MUNICIPALITY	2	47	2	0	51
GORDONS BAY MUNICIPALITY	0	2	0	0	2
KRAAIFONTEIN MUNICIPALITY	7	122	3	0	132
BRACKENFELL MUNICIPALITY	1	6	8	0	15
KUILS RIVER MUNICIPALITY	2	91	0	0	93
PAARL MUNICIPALITY	11	430	215	0	656
WELLINGTON MUNICIPALITY	0	40	0	0	40
CAPE TOWN MUNICIPALITY	70	2245	1744	7	4066
TOTAL	150	6472	4623	21	11261

LOCATION	1989 PULMONARY TB NOTIFICATIONS WHITE	COLOURED	BLACK	ASIAN	TOTAL NOTIFI
DIV COUNCIL OF CAPE	54	2443	2545	6	5048
DIV COUNCIL OF STELLENBOSCH	3	672	197	0	869
DIV COUNCIL OF PAARL	4	301	36	0	337
STELLENBOSCH MUNICIPALITY	1	78	77	0	156
SOMERSET WEST MUNICIPALITY	0	36	4	0	40
STRAND MUNICIPALITY	8	119	3	0	130
GORDONS BAY MUNICIPALITY	1	2	1	0	4
KRAAIFONTEIN MUNICIPALITY	13	116	3	0	132
BRACKENFELL MUNICIPALITY	1	4	8	0	13
KUILS RIVER MUNICIPALITY	4	74	0	0	78
PAARL MUNICIPALITY	9	419	195	0	623
WELLINGTON MUNICIPALITY	0	35	3	0	38
CAPE TOWN MUNICIPALITY	52	2587	1845	12	4496
TOTAL	150	6856	4917	18	11964

APPENDIX E5a(ii)

DATA OF PTB NOTIFICATIONS OF WCHSR: VARIOUS DEMARCATED AREAS & MACASSAR

APPENDIX E5b

RECORD OF MACASSAR/FIGROVE POPULATION

AND

PAST TB CASES FROM CLINIC (1985 - 1989)

MACASSAR/FIRGROVE POPULATION
(1985 - 1989)

LOCATION MACASSAR/FIRGROVE	YEAR	RACE WHITE	COLOURED	BLACK	ASIAN	TOT POP
	1985	0	20000	1	4	20005
	1986	0	22700	1	4	22705
	1987	0	25200	1	4	25205
	1988	0	27500	1	4	27505
	1989	0	30300	20	4	30324

MACASSAR/FIRGROVE PULMONARY TB NOTIFICATIONS
(1985 - 1989)

LOCATION MACASSAR/FIRGROVE	YEAR	RACE WHITE	COLOURED	BLACK	ASIAN	TOTAL NOTIFICA
	1985	0	45	0	0	45
	1986	0	96	0	0	96
	1987	0	110	0	0	110
	1988	0	115	0	0	115
	1989	0	126	0	0	126

MACASSAR TB (PULMONARY) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
1	21	F	55/01/05	
5	41	M	55/01/22	
	45	F	55/02/09	
	37	F	55/03/21	
	27	F	55/04/15	
	23	M	55/06/01	
0	42	M	55/06/01	
	27	M	55/07/05	
5	54	M	55/07/08	
0	45	M	55/07/11	
2	60	M	55/07/15	
4	35	M	55/07/16	
	24	F	55/07/22	
	50	M	55/07/25	
	25	M	55/07/27	
3	35	M	55/05/02	
9	28	F	55/09/12	
3	15	M	55/08/26	
7	40	M	55/08/26	
9	48	M	55/09/29	DOD 56/02/25
	60	M	55/09/02	
1	30	F	55/09/28	
0	22	F	55/09/30	
3	25	M	55/09/30	
	35	M	55/10/07	
4	20	F	55/10/21	
2	22	M	55/10/21	
3	24	F	55/10/21	
2	25	M	55/10/21	
0	35	F	55/10/21	
6	38	F	55/10/22	
	47	F	55/10/23	
	30	F	55/10/25	
	52	M	55/10/29	
4	30	F	55/11/04	
6	24	F	55/11/11	
5	25	F	55/11/18	
9	40	F	55/11/18	
	29	M	55/11/22	
7	31	F	55/12/02	
5	20	F	55/12/11	
7	15	M	55/12/17	
9	15	M	55/12/17	REACTIVATED
3	19	M	55/12/17	
0	70	M	55/12/21	
4	33	M	56/01/05	
2	44	M	56/01/10	
9	15	M	56/01/13	
4	25	F	56/01/20	
15	38	F	56/01/20	
15	50	M	56/01/24	
15	26	M	56/01/26	
4	25	F	56/01/27	
6	30	M	56/01/27	
9	25	M	56/02/04	
19	15	F	56/02/10	
41	25	M	56/02/11	
16	25	M	56/02/13	
156	25	M	56/02/17	
57	47	M	56/02/17	
15	27	M	56/02/18	
17	22	F	56/02/24	
18	23	M	56/02/24	
167	51	M	56/02/24	
9	22	F	56/03/03	
	31	F	56/03/03	
56	25	F	56/03/17	
12	34	F	56/03/17	
37	48	F	56/03/17	
321	29	F	56/03/24	
45	45	M	56/04/01	
49	43	M	56/04/02	
78	19	M	56/04/11	
34	25	M	56/04/14	
40	22	F	56/04/15	
56	62	M	56/04/21	
	26	F	56/05/06	
73	42	M	56/05/05	
67	31	F	56/05/07	
15	22	M	56/05/12	
43	32	M	56/05/12	
54	66	M	56/05/12	
39	27	F	56/05/21	
156	25	M	56/05/25	
34	24	F	56/05/26	
151	30	M	56/05/26	
33	37	M	56/06/05	
	63	F	56/06/06	
10	18	F	56/06/12	
25	36	F	56/06/26	
44	44	F	56/06/26	
34	25	F	56/07/03	
14	27	M	56/07/08	
57	23	M	56/07/10	
75	24	F	56/07/16	
22	52	F	56/07/16	
29	32	F	56/07/17	REACTIVATED
36	32	F	56/07/17	
59	60	M	56/07/22	
4	30	M	56/07/24	DOD 57/01/17
31	32	M	56/07/25	
23	57	F	56/07/30	
42	20	F	56/07/31	
62	26	F	56/05/01	
40	24	F	56/08/12	
27	61	M	56/08/21	
50	30	F	56/08/25	
19	40	M	56/08/28	REACTIVATED
52	24	M	56/09/04	
35	26	M	56/09/11	
9	27	F	56/09/19	
54	35	M	56/09/19	
16	64	M	56/09/19	
14	21	F	56/10/02	
25	25	F	56/10/02	
69	29	F	56/10/02	
1	19	M	56/10/09	
14	25	F	56/10/09	REACTIVATED
13	25	F	56/10/09	
56	61	M	56/10/10	
73	20	F	56/10/23	
74	21	F	56/10/23	
61	25	F	56/10/23	
54	29	M	56/10/23	
41	25	M	56/10/23	REACTIVATED
76	15	F	56/10/30	
47	29	M	56/10/30	
36	32	F	56/10/30	
29	20	F	56/11/06	
63	27	F	56/11/06	
30	27	F	56/11/20	
12	19	F	56/11/22	
49	65	F	56/11/25	DOD 57/05/11
30	15	M	56/11/27	
40	24	M	56/11/27	
105	20	M	56/12/04	
23	23	M	56/12/04	
84	31	M	56/12/18	REACTIVATED
23	27	M	56/12/22	
53	29	F	56/12/22	
61	46	M	56/12/22	
15	20	F	57/01/07	
79	41	M	57/01/08	
31	61	M	57/01/05	
96	21	M	57/01/15	
57	23	F	57/01/15	
24	21	F	57/01/22	
90	21	F	57/01/22	
	30	F	57/01/29	
19	42	M	57/01/29	
3	18	M	57/02/05	
62	25	F	57/02/05	
22	18	M	57/02/09	REACTIVATED
2	30	F	57/02/09	
50	30	F	57/02/16	
34	70	M	57/02/19	REACTIVATED
3	32	M	57/02/20	
5	47	F	57/02/26	
31	51	M	57/02/26	
19	52	M	57/02/26	
24	50	M	57/03/05	
2	25	F	57/01/12	REACTIVATED
1	26	F	57/03/12	
3	30	F	57/04/19	
17	23	F	57/03/26	REACTIVATED
54	37	F	57/04/26	
29	34	M	57/04/01	
43	25	M	57/01/02	
27	63	M	57/04/02	REACTIVATED
9	21	F	57/01/07	
42	23	F	57/01/09	
40	24	M	57/04/09	REACTIVATED
9	15	M	57/04/21	REACTIVATED
9	33	F	57/04/23	
37	35	M	57/04/23	
37	49	F	57/04/23	
11	15	F	57/04/30	
29	22	F	57/04/30	
27	18	M	57/05/07	
12	23	M	57/05/07	
63	27	M	57/05/07	
8	48	F	57/05/08	
35	27	F	57/05/14	
15	31	M	57/05/18	
16	34	M	57/05/21	
130	30	F	57/05/26	
134	27	M	57/05/27	
14	30	M	57/06/01	
12	24	M	57/06/04	REACTIVATED
60	30	M	57/06/04	
17	40	F	57/06/04	REACTIVATED
43	22	F	57/06/18	
38	43	F	57/06/18	
39	38	F	57/06/19	DOD 58/01/23
32	49	M	57/06/24	
145	28	M	57/06/25	
35	30	M	57/07/02	
10	57	M	57/07/02	
16	26	M	57/07/09	REACTIVATED
13	46	M	57/07/10	
44	20	M	57/07/15	
10	24	F	57/07/16	REACTIVATED
152	34	F	57/07/16	
130	34	M	57/07/23	
47	22	M	57/07/28	
28	35	M	57/07/28	REACTIVATED
21	49	M	57/07/28	
25	23	M	57/08/06	
32	21	F	57/08/12	
177	26	M	57/08/12	
84	32	F	57/08/17	
22	21	F	57/08/27	
92	27	F	57/08/27	
51	20	F	57/08/27	
59	50	F	57/08/27	
90	26	M	57/09/03	
96	45	M	57/09/03	
36	57	F	57/09/03	DOD 58/04/11
29	22	F	57/09/17	
	26	F	57/09/24	

MACASSAR TB (PULMONARY) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
1	21	F	55/01/05	
5	41	M	55/01/22	
	45	F	55/02/09	
	37	F	55/03/21	
	27	F	55/04/15	
	23	M	55/06/01	
0	42	M	55/06/01	
	27	M	55/07/05	
5	54	M	55/07/08	
0	45	M	55/07/11	
2	60	M	55/07/15	
4	35	M	55/07/16	
	24	F	55/07/22	
	50	M	55/07/25	
	25	M	55/07/27	
3	35	M	55/05/02	
9	28	F	55/09/12	
3	15	M	55/08/26	
7	40	M	55/08/26	
9	48	M	55/09/29	DOD 56/02/25
	60	M	55/09/02	
1	30	F	55/09/28	
0	22	F	55/09/30	
3	25	M	55/09/30	
	35	M	55/10/07	
4	20	F	55/10/21	
2	22	M	55/10/21	
3	24	F	55/10/21	
2	25	M	55/10/21	
0	35	F	55/10/21	
6	38	F	55/10/22	
	47	F	55/10/23	
	30	F	55/10/25	
	52	M	55/10/29	
4	30	F	55/11/04	
6	24	F	55/11/11	
5	25	F	55/11/18	
9	40	F	55/11/18	
	29	M	55/11/22	
7	31	F	55/12/02	
5	20	F	55/12/11	
7	15	M	55/12/17	
9	15	M	55/12/17	REACTIVATED
3	19	M	55/12/17	
0	70	M	55/12/21	
4	33	M	56/01/05	
2	44	M	56/01/10	
9	15	M	56/01/13	
4	25	F	56/01/20	
15	38	F	56/01/20	
15	50	M	56/01/24	
15	26	M	56/01/26	
4	25	F	56/01/27	
6	30	M	56/01/27	
9	25	M	56/02/04	
19	15	F	56/02/10	
41	25	M	56/02/11	
16	25	M	56/02/13	
156	25	M	56/02/17	
57	47	M	56/02/17	
15	27	M	56/02/18	
17	22	F	56/02/24	
18	23	M	56/02/24	
167	51	M	56/02/24	
9	22	F	56/03/03	
	31	F	56/03/03	
56	25	F	56/03/17	
12	34	F	56/03/17	
37	48	F	56/03/17	
321	29	F	56/03/24	
45	45	M	56/04/01	
49	43	M	56/04/02	
78	19	M	56/04/11	
34	25	M	56/04/14	
40	22	F	56/04/15	
56	62	M	56/04/21	
	26	F	56/05/06	
73	42	M	56/05/05	
67	31	F	56/05/07	
15	22	M	56/05/12	
43	32	M	56/05/12	
54	66	M	56/05/12	
39	27	F	56/05/21	
156	25	M	56/05/25	
34	24	F	56/05/26	
151	30	M	56/05/26	
33	37	M	56/06/05	
	63	F	56/06/06	
10	18	F	56/06/12	
25	36	F	56/06/26	
44	44	F	56/06/26	
34	25	F	56/07/03	
14	27	M	56/07/08	
57	23	M	56/07/10	
75	24	F	56/07/16	
22	52	F	56/07/16	
29	32	F	56/07/17	REACTIVATED
36	32	F	56/07/17	
59	60	M	56/07/22	
4	30	M	56/07/24	DOD 57/01/17
31	32	M	56/07/25	
23	57	F	56/07/30	
42	20	F	56/07/31	
62	26	F	56/05/01	
40	24	F	56/08/12	
27	61	M	56/08/21	
50	30	F	56/08/25	
19	40	M	56/08/28	REACTIVATED
52	24	M	56/09/04	
35	26	M	56/09/11	
9	27	F	56/09/19	
54	35	M	56/09/19	
16	64	M	56/09/19	
14	21	F	56/10/02	
25	25	F	56/10/02	
69	29	F	56/10/02	
1	19	M	56/10/09	
14	25	F	56/10/09	REACTIVATED
13	25	F	56/10/09	
56	61	M	56/10/10	
73	20	F	56/10/23	
74	21	F	56/10/23	
61	25	F	56/10/23	
54	29	M	56/10/23	
41	25	M	56/10/23	REACTIVATED
76	15	F	56/10/30	
47	29	M	56/10/30	
36	32	F	56/10/30	
29	20	F	56/11/06	
63	27	F	56/11/06	
30	27	F	56/11/20	
12	19	F	56/11/22	
49	65	F	56/11/25	DOD 57/05/11
30	15	M	56/11/27	
40	24	M	56/11/27	
105	20	M	56/12/04	
23	23	M	56/12/04	
84	31	M	56/12/18	REACTIVATED
23	27	M	56/12/22	
53	29	F	56/12/22	
61	46	M	56/12/22	
15	20	F	57/01/07	
79	41	M	57/01/08	
31	61	M	57/01/05	
96	21	M	57/01/15	
57	23	F	57/01/15	
24	21	F	57/01/22	
90	21	F	57/01/22	
	30	F	57/01/29	
19	42	M	57/01/29	
3	18	M	57/02/05	
62	25	F	57/02/05	
22	18	M	57/02/09	REACTIVATED
2	30	F	57/02/09	
50	30	F	57/02/16	
34	70	M	57/02/19	REACTIVATED
3	32	M	57/02/20	
5	47	F	57/02/26	
31	51	M	57/02/26	
19	52	M	57/02/26	
24	50	M	57/03/05	
2	25	F	57/01/12	REACTIVATED
1	26	F	57/03/12	
3	30	F	57/04/19	
17	23	F	57/03/26	REACTIVATED
54	37	F	57/04/26	
29	34	M	57/04/01	
43	25	M	57/01/02	
27	63	M	57/04/02	REACTIVATED
9	21	F	57/01/07	
42	23	F	57/01/09	
40	24	M	57/04/09	REACTIVATED
9	15	M	57/04/21	REACTIVATED
9	33	F	57/04/23	
37	35	M	57/04/23	
37	49	F	57/04/23	
11	15	F	57/04/30	
29	22	F	57/04/30	
27	18	M	57/05/07	
12	23	M	57/05/07	
63	27	M	57/05/07	
8	48	F	57/05/08	
35	27	F	57/05/14	
15	31	M	57/05/18	
16	34	M	57/05/21	
130	30	F	57/05/26	
134	27	M	57/05/27	
14	30	M	57/06/01	
12	24	M	57/06/04	REACTIVATED
60	30	M	57/06/04	
17	40	F	57/06/04	REACTIVATED
43	22	F	57/06/18	
38	43	F	57/06/18	
39	38	F	57/06/19	DOD 58/01/23
32	49	M	57/06/24	
145	28	M	57/06/25	
35	30	M	57/07/02	
10	57	M	57/07/02	
16	26	M	57/07/09	REACTIVATED
13	46	M	57/07/10	
44	20	M	57/07/15	
10	24	F	57/07/16	REACTIVATED
152	34	F	57/07/16	
130	34	M	57/07/23	
47	22	M	57/07/28	
28	35	M	57/07/28	REACTIVATED
21	49	M	57/07/28	
25	23	M	57/08/06	
32	21	F	57/08/12	
177	26	M	57/08/12	
84	32	F	57/08/17	
22	21	F	57/08/27	
92	27	F	57/08/27	
51	20	F	57/08/27	
59	50	F	57/08/27	
90	26	M	57/09/03	
96	45	M	57/09/03	
36	57	F	57/09/03	DOD 58/04/11
29	22	F	57/09/17	
	26	F	57/09/24	

MACASSAR TB (ALL FORMS) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
31	21	F	55/01/05	
16	11	M	55/01/23	
	45	F	55/02/09	
	37	F	55/03/21	
	27	F	55/01/15	
	23	M	55/06/01	
10	42	M	55/06/01	
41	17	F	55/06/19	
	27	M	55/07/08	
45	54	M	55/07/08	
60	45	M	55/07/11	
42	60	M	55/07/15	
124	38	M	85/07/16	
	24	F	85/07/22	
	50	M	85/07/25	
	25	M	55/07/27	
53	35	M	85/08/02	
	5	F	85/08/12	
129	28	F	55/05/12	
67	15	M	55/05/20	
27	40	M	85/08/26	
29	48	M	85/08/29	DOD 56/02/28
	60	M	55/09/02	
51	30	F	85/09/28	
30	22	F	55/09/30	
33	28	M	85/09/30	
	15	M	55/10/07	
	38	M	56/10/07	
	6	M	55/10/14	
3	16	M	55/10/21	
14	20	F	55/10/21	
52	22	M	56/10/21	
43	24	F	55/10/21	
52	25	M	55/10/21	
50	38	F	55/10/21	
36	38	F	55/10/22	
	47	F	56/10/23	
	30	F	55/10/28	
	52	M	55/10/29	
64	30	F	55/11/04	
60	14	F	55/11/11	
96	24	F	55/11/11	
5	25	F	55/11/15	
9	40	F	55/11/18	
	29	M	85/12/02	
57	31	F	56/12/02	
55	17	F	55/12/06	
26	2	M	55/12/09	
15	20	F	85/12/11	
1	15	F	85/12/17	
17	18	M	85/12/17	
29	15	M	58/12/17	REACTIVATED
12	19	M	55/12/17	
70	70	M	56/12/21	
11	2	F	56/01/05	
111	33	M	56/01/05	
22	41	M	56/01/10	
59	15	M	56/01/13	
14	25	F	56/01/20	
145	38	F	56/01/20	
35	50	M	56/01/24	
15	20	M	56/01/26	
154	25	F	56/01/27	
6	30	M	56/01/27	
5	12	M	56/02/04	
9	25	M	56/02/04	
19	15	F	56/02/10	
41	25	M	56/02/11	
16	20	M	56/02/13	
156	25	M	56/02/17	
57	37	M	56/02/17	
15	27	M	56/02/18	
17	22	F	56/02/24	
15	23	M	56/02/24	
167	31	M	56/02/24	
9	22	F	56/03/03	
	31	F	56/03/03	
	3	M	56/03/10	
67	2	F	56/03/17	
56	28	F	56/03/17	
12	34	F	56/03/17	
37	48	F	56/03/17	
321	29	F	56/03/24	
45	45	M	56/04/01	
49	43	M	56/04/02	
78	19	M	56/04/11	
34	25	M	56/04/14	
40	22	F	56/04/15	
56	62	M	56/04/21	
	26	F	56/05/05	
73	42	M	56/05/05	
67	31	F	56/05/07	
15	22	M	56/05/12	
13	32	M	56/05/12	
54	66	M	56/05/12	
53	16	F	56/05/15	
15	3	M	56/05/19	
39	27	F	56/05/21	
156	25	M	56/05/25	
34	24	F	56/05/26	
151	30	M	56/05/26	
21	17	M	56/06/05	
33	37	M	56/06/05	
	63	F	56/06/06	
10	15	F	56/06/12	
	16	M	56/06/26	
25	36	F	56/06/26	
44	41	F	56/06/26	
43	2	M	56/07/03	
34	25	F	56/07/03	

MACASSAR TB (ALL FORMS) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
100	8	F	56/07/05	
14	27	M	56/07/05	
57	23	M	56/07/10	
75	24	F	56/07/16	
22	52	F	56/07/16	
29	32	F	56/07/17	REACTIVATED
36	32	F	56/07/17	
59	60	M	56/07/22	
1	30	M	56/07/24	DOD 57/01/17
48	17	M	56/07/25	
31	32	M	56/07/28	
23	57	F	56/07/30	
42	20	F	56/07/31	
62	26	F	56/08/01	
1	3	F	56/08/08	
40	24	F	56/08/12	
27	61	M	56/08/21	
50	30	F	56/08/25	
19	40	M	56/08/28	REACTIVATED
61	17	F	56/09/04	
52	24	M	56/09/04	
6	17	F	56/09/11	
38	26	M	56/09/11	
9	27	F	56/09/19	
74	35	M	56/09/19	
6	64	M	56/09/19	
29	1	M	56/10/02	
14	21	F	56/10/02	
25	25	F	56/10/02	
69	29	F	56/10/02	
29	1	M	56/10/03	
10	2	M	56/10/09	
1	19	M	56/10/09	
14	28	F	56/10/09	REACTIVATED
43	25	F	56/10/09	
56	61	M	56/10/10	
73	20	F	56/10/23	
74	21	F	56/10/23	
61	28	F	56/10/23	
54	29	M	56/10/23	
41	25	M	56/10/23	REACTIVATED
42	3	M	56/10/24	
71	17	F	56/10/30	
76	15	F	56/10/30	
47	29	M	56/10/30	
36	32	F	56/10/30	
29	20	F	56/11/06	
63	27	F	56/11/06	
30	27	F	56/11/20	
12	19	F	56/11/22	
49	65	F	56/11/25	DOD 57/05/11
30	15	M	56/11/27	
40	24	M	56/11/27	
73	16	F	56/12/04	
105	20	M	56/12/04	
23	23	M	56/12/04	
51	31	M	56/12/15	REACTIVATED
23	27	M	56/12/22	
53	29	F	56/12/22	
51	46	M	56/12/22	
18	20	F	57/01/07	
79	41	M	57/01/08	
31	61	M	57/01/08	
86	21	M	57/01/15	
87	23	F	57/01/15	
24	21	F	57/01/22	
90	21	F	57/01/22	
	30	F	57/01/29	
19	42	M	57/01/29	
3	18	M	57/02/05	
62	28	F	57/02/05	
22	18	M	57/02/09	REACTIVATED
2	30	F	57/02/09	
60	30	F	57/02/16	
3	17	M	57/02/19	
34	70	M	57/02/19	REACTIVATED
3	32	M	57/02/20	
5	47	F	57/02/26	
31	51	M	57/02/26	
19	52	M	57/02/26	
92	1	F	57/03/05	
3	4	F	57/03/05	
24	50	M	57/03/05	
2	25	F	57/03/12	REACTIVATED
1	26	F	57/03/12	
5	17	F	57/03/19	
3	30	F	57/03/19	
16	13	M	57/03/26	
17	23	F	57/03/26	REACTIVATED
54	37	F	57/03/26	
29	24	M	57/04/01	
43	26	M	57/04/02	
27	63	M	57/04/02	REACTIVATED
9	24	F	57/04/07	
30	16	F	57/04/09	
42	23	F	57/04/09	
40	24	M	57/04/09	REACTIVATED
55	15	F	57/04/16	
9	18	M	57/04/21	REACTIVATED
9	33	F	57/04/23	
37	38	M	57/04/23	
37	49	F	57/04/23	
22	7	M	57/04/30	
11	15	F	57/04/30	
29	22	F	57/04/30	
23	2	M	57/05/05	
27	18	M	57/05/07	
12	23	M	57/05/07	

MACASSAR TB (ALL FORMS) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
63	27	M	57/05/07	
5	45	F	57/05/05	
10	15	F	57/05/14	
35	27	F	57/05/14	
17	31	M	57/05/15	
16	34	M	57/05/21	
130	30	F	57/05/26	
131	27	M	57/05/27	
14	30	M	57/06/01	
12	24	M	57/06/04	REACTIVATED
60	30	M	57/06/04	
17	46	F	57/06/04	REACTIVATED
13	22	F	57/06/13	
35	43	F	57/06/15	
39	25	F	57/06/19	DOD 55/01/23
32	49	M	57/06/24	
115	23	M	57/06/25	
38	30	M	57/07/02	
10	57	M	57/07/02	
16	25	M	57/07/09	REACTIVATED
18	46	M	57/07/10	
44	20	M	57/07/15	
19	15	F	57/07/16	
10	24	F	57/07/16	REACTIVATED
152	32	F	57/07/16	
157	15	F	57/07/23	
130	31	M	57/07/23	
47	22	M	57/07/25	
25	35	M	57/07/25	REACTIVATED
21	49	M	57/07/26	
48	17	M	57/07/30	
25	23	M	57/05/06	
32	21	F	57/05/12	
177	26	M	57/05/12	
51	32	F	57/08/17	
22	21	F	57/05/27	
92	27	F	57/08/27	
51	29	F	57/05/27	
59	50	F	57/05/27	
95	13	M	57/09/03	
90	26	M	57/09/03	
96	45	M	57/09/03	
36	57	F	57/09/03	DOD 55/04/11
	2	M	57/09/07	
29	22	F	57/09/17	
	25	F	57/09/24	
54	25	F	57/09/24	
35	37	M	57/09/24	
2	16	F	57/10/01	
9	42	M	57/10/02	
5	10	F	57/10/08	
14	26	M	57/10/08	
16	15	F	57/10/14	REACTIVATED
15	41	F	57/10/14	
31	41	F	57/10/15	
	30	F	57/10/19	
13	24	M	57/10/21	
92	2	M	57/10/22	REACTIVATED
30	15	F	57/10/22	
21	20	F	57/10/22	
29	30	F	57/10/22	REACTIVATED
17	17	F	57/10/25	
4	15	M	57/10/29	
76	19	F	57/10/29	REACTIVATED
14	22	M	57/10/29	
10	27	M	57/10/29	
16	29	M	57/11/05	
19	37	M	57/11/05	
57	61	M	57/11/05	REACTIVATED
34	29	M	57/11/07	
15	5	F	57/11/11	
	45	F	57/11/12	
20	51	F	57/11/12	
15	22	F	57/11/19	
12	33	M	57/11/19	
5	52	M	57/11/19	
	65	M	57/11/19	
70	20	M	57/11/24	
15	21	M	57/11/24	
24	30	F	57/11/27	
97	15	F	57/12/03	
	19	M	57/12/09	
2	33	F	57/12/09	REACTIVATED
32	18	M	57/12/10	
21	18	F	57/12/14	
19	19	F	57/12/14	
24	33	M	55/01/04	
8	24	F	55/01/07	
37	15	F	55/01/05	
36	24	F	58/01/12	
16	1	M	55/01/20	
37	24	F	58/01/20	
54	38	M	58/01/21	DOD 55/03/12
41	41	M	58/01/21	
41	45	M	55/01/21	
34	60	F	58/01/21	
7	5	M	55/01/26	REACTIVATED
20	7	F	55/01/25	
110	40	M	58/01/25	
	63	M	58/01/29	DOD 55/02/05 REAC
45	34	F	58/02/04	
70	15	M	55/02/11	
40	22	F	55/02/11	
23	35	M	58/02/11	
41	37	M	55/02/11	
44	52	F	58/02/11	
119	34	F	55/02/18	
45	45	M	55/02/25	
125	15	F	55/03/03	
18	21	F	58/03/03	REACTIVATED

MACASSAR TB (ALL FORMS) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
131	39	F	58/03/03	REACTIVATED
	66	M	55/03/03	
61	36	F	58/03/10	
	33	M	55/03/17	
14	52	M	58/03/17	REACTIVATED
50	60	M	55/03/17	REACTIVATED
42	21	F	58/03/31	
71	65	M	55/03/31	
145	31	F	55/04/14	
91	49	M	58/04/14	
11	35	F	55/04/21	
59	19	M	58/04/28	
58	1	F	58/05/10	
54	30	F	58/05/10	
56	45	M	58/05/10	
38	45	M	58/05/10	
29	30	M	58/05/18	
46	23	F	58/05/19	
60	26	M	58/05/19	
11	27	F	58/05/19	
63	33	F	58/05/19	
9	55	M	58/05/19	
62	59	M	55/05/19	
60	62	M	58/05/19	
61	15	F	58/05/27	REACTIVATED
55	17	M	58/05/27	
32	19	M	58/06/02	
61	20	F	58/06/02	
55	21	M	58/06/02	
70	21	M	58/06/02	
12	3	M	58/06/09	
66	5	F	58/06/09	
35	32	M	58/06/09	
21	60	M	55/06/09	
17	21	F	58/06/16	
24	30	F	58/06/16	
10	57	M	58/06/16	REACTIVATED
38	69	M	55/06/22	
62	18	M	58/06/23	
40	25	F	55/06/23	
18	36	F	58/06/23	
68	36	M	55/06/23	
14	29	F	58/06/30	REACTIVATED
19	46	M	55/06/30	
37	19	F	58/07/14	
22	65	F	55/07/14	
112	22	M	58/07/21	
2	46	F	55/07/21	
1	20	M	55/07/21	REACTIVATED
21	19	F	58/07/28	REACTIVATED
36	22	M	55/07/28	
110	36	M	55/07/28	
51	69	M	58/07/28	DOD 58/09/01
2	14	F	55/08/04	
13	28	F	58/08/04	
67	31	M	55/05/04	
13	34	M	55/08/04	
69	60	F	55/08/01	
72	25	I	55/05/09	
55	23	F	55/08/10	
22	27	F	55/08/10	
27	25	M	55/08/10	
59	26	M	55/05/17	DOD 55/08/28 REA
24	25	F	55/08/15	REACTIVATED
122	31	F	55/08/15	
7	67	M	55/05/15	
28	20	M	55/08/20	
52	15	M	55/05/25	REACTIVATED
58	23	F	55/05/25	
40	30	M	55/08/25	
140	65	F	55/05/25	
61	23	F	55/09/01	
73	24	M	55/09/01	
126	26	M	55/09/01	
25	34	M	55/09/15	
9	39	M	55/09/15	REACTIVATED
15	17	M	55/09/22	
48	41	F	55/09/22	
81	26	F	55/09/23	
53	29	M	55/09/29	
67	37	F	55/09/29	
61	40	F	55/09/29	
43	55	F	55/09/29	
47	23	M	55/10/06	
178	34	M	55/10/06	
65	17	F	55/10/13	
64	37	F	55/10/20	
61	60	F	55/10/20	
55	24	M	55/10/27	
19	49	M	55/10/27	
82	50	F	55/11/03	REACTIVATED
86	19	M	55/11/10	
56	65	F	55/11/10	
160	34	M	55/11/14	
18	59	F	55/11/14	REACTIVATED
7	2	F	55/11/24	
6	1	M	55/11/25	
161	45	M	55/11/28	REACTIVATED
165	18	M	55/12/01	
14	52	M	55/12/01	REACTIVATED
76	46	M	55/12/01	
8	65	M	55/12/01	
50	22	F	55/12/15	
29	34	F	55/12/15	REACTIVATED
	56	M	55/12/20	REACTIVATED
97	2	M	55/12/22	
55	15	M	59/01/05	
54	24	M	59/01/05	
78	24	M	59/01/05	
173	35	M	59/01/05	

MACASSAR TB (PULMONARY) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
51	28	F	57/09/21	
35	37	M	57/09/24	
14	26	M	57/10/05	
15	41	F	57/10/11	
31	21	F	57/10/15	
	30	F	57/10/19	
13	24	M	57/10/21	
30	15	F	57/10/22	
21	20	F	57/10/22	
20	30	F	57/10/22	REACTIVATED
70	19	F	57/10/28	REACTIVATED
14	22	M	57/10/28	
10	27	M	57/10/29	
16	29	M	57/11/05	
19	37	M	57/11/05	
57	61	M	57/11/05	REACTIVATED
34	29	M	57/11/07	
	45	F	57/11/12	
20	51	F	57/11/12	
15	22	F	57/11/19	
12	33	M	57/11/19	
5	52	M	57/11/19	
	65	M	57/11/19	
70	20	M	57/11/24	
15	21	M	57/11/24	
24	30	F	57/11/27	
	19	M	57/12/09	
2	33	F	57/12/09	REACTIVATED
32	18	M	57/12/10	
21	15	F	57/12/14	
19	19	F	57/12/14	
24	33	M	58/01/04	
8	24	F	58/01/07	
36	24	F	58/01/12	
37	24	F	58/01/20	
54	38	M	58/01/21	DOD 58/03/12
41	41	M	58/01/21	
41	45	M	58/01/21	
34	60	F	58/01/21	
110	40	M	58/01/26	
	63	M	58/01/29	DOD 58/02/05 REACT
45	31	F	58/02/04	
70	15	M	58/02/11	
40	22	F	58/02/11	
23	39	M	58/02/11	
41	37	M	58/02/11	
44	52	F	58/02/11	
119	34	F	58/02/15	
45	45	M	58/02/25	
125	15	F	58/03/03	
18	21	F	58/03/03	REACTIVATED
134	39	F	58/03/03	REACTIVATED
	60	M	58/03/03	
61	56	F	58/03/10	
	33	M	58/03/17	
11	52	M	58/03/17	REACTIVATED
50	60	M	58/03/17	REACTIVATED
12	21	F	58/03/31	
71	65	M	58/03/31	
145	31	F	58/04/14	
91	49	M	58/04/14	
44	35	F	58/04/21	
59	19	M	58/04/25	
54	30	F	58/05/10	
56	45	M	58/05/10	
35	45	M	58/05/10	
29	30	M	58/05/15	
46	23	F	58/05/19	
60	26	M	58/05/19	
11	27	F	58/05/19	
63	33	F	58/05/19	
9	55	M	58/05/19	
62	59	M	58/05/19	
60	62	M	58/05/19	
32	19	M	58/06/02	
61	20	F	58/06/02	
55	21	M	58/06/02	
70	21	M	58/06/02	
35	32	M	58/06/09	
21	60	M	58/06/09	
17	21	F	58/06/16	
24	30	F	58/06/16	
10	57	M	58/06/16	REACTIVATED
38	65	M	58/06/22	
62	18	M	58/06/23	
40	25	F	58/06/23	
18	36	F	58/06/23	
68	36	M	58/06/23	
14	29	F	58/06/30	REACTIVATED
19	46	M	58/06/30	
37	19	F	58/07/14	
22	55	F	58/07/14	
112	22	M	58/07/21	
2	46	F	58/07/21	
1	20	M	58/07/21	REACTIVATED
21	18	F	58/07/28	REACTIVATED
36	22	M	58/07/28	
116	36	M	58/07/28	
51	69	M	58/07/28	DOD 58/09/01
13	28	F	58/08/04	
67	31	M	58/08/04	
43	34	M	58/08/04	
69	60	F	58/08/04	
72	28	F	58/08/09	
55	23	F	58/08/10	
22	27	F	58/08/10	
27	25	M	58/08/10	
59	26	M	58/08/17	DOD 58/08/28 REA
34	25	F	58/08/18	REACTIVATED
122	31	F	58/08/18	

MACASSAR TB (PULMONARY) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
7	67	M	58/08/18	
28	20	M	58/08/20	
52	18	M	58/08/25	REACTIVATED
55	25	F	58/08/25	
40	30	M	58/08/25	
110	65	F	58/08/25	
61	23	F	58/09/01	
73	24	M	58/09/01	
128	26	M	58/09/01	
25	34	M	58/09/15	
9	39	M	58/09/15	REACTIVATED
48	41	F	58/09/22	
51	26	F	58/09/23	
53	28	M	58/09/29	
67	37	F	58/09/29	
61	40	F	58/09/29	
43	58	F	58/09/29	
47	23	M	58/10/06	
178	34	M	58/10/06	
54	37	F	58/10/20	
51	60	F	58/10/20	
55	24	M	58/10/27	
19	49	M	58/10/27	
82	50	F	58/11/03	REACTIVATED
86	19	M	58/11/10	
58	65	F	58/11/10	
160	34	M	58/11/14	
18	59	F	58/11/14	REACTIVATED
161	43	M	58/11/28	REACTIVATED
165	19	M	58/12/01	
14	52	M	58/12/01	REACTIVATED
76	46	M	58/12/01	
8	65	M	58/12/01	
50	22	F	58/12/15	
29	34	F	58/12/15	REACTIVATED
	58	M	58/12/20	REACTIVATED
55	18	M	59/01/05	
54	24	M	59/01/05	
78	24	M	59/01/05	
173	35	M	59/01/05	
56	47	M	59/01/05	
58	22	M	59/01/12	
79	50	M	59/01/12	
13	61	F	59/01/12	REACTIVATED
58	25	M	59/01/23	
91	24	M	59/01/26	
35	54	F	59/01/26	
82	45	M	59/01/26	
22	45	M	59/01/31	
36	32	F	59/02/01	
29	27	M	59/02/07	
26	24	F	59/02/09	
42	47	M	59/02/10	
2	31	M	59/02/16	
5	36	F	59/02/16	
11	50	M	59/02/22	REACTIVATED
55	22	F	59/02/23	
56	24	F	59/02/23	
22	30	M	59/02/23	REACTIVATED
43	24	F	59/03/02	REACTIVATED
24	19	F	59/03/09	REACTIVATED
19	24	M	59/03/09	
12	36	F	59/03/09	
6	35	M	59/03/09	
110	41	M	59/03/09	REACTIVATED
198	62	M	59/03/09	
13	71	F	59/03/09	
	34	F	59/03/16	REACTIVATED
4	35	M	59/03,16	REACTIVATED?
65	51	M	59/03/16	REACTIVATED
28	54	F	59/03/16	
1	21	M	59/03/23	REACTIVATED
41	25	M	59/03/23	
35	31	M	59/03/23	
26	30	F	59/03/30	
157	32	M	59/03/30	
21	36	M	59/04/03	
21	33	F	59/04/10	REACTIVATED
22	24	F	59/04/11	
169	15	F	59/04/13	
100	19	M	59/04/13	
27	31	F	59/04/13	
21	20	F	59/04/18	DOD 90/01/13 E
3	67	M	59/04/21	
45	24	M	59/04/26	
5	26	M	59/04/27	
10	26	F	59/04/27	
	51	M	59/04/27	REACTIVATED
11	43	M	59/05/02	
24	26	M	59/05/11	
12	32	M	59/05/11	
9	44	M	59/05/11	REACTIVATED
13	19	F	59/05/18	
14	30	M	59/05/18	
11	15	F	59/05/22	
75	22	M	59/05/22	
6	22	M	59/05/22	
355	61	F	59/05/22	REACTIVATED
16	22	M	59/05/28	
44	60	M	59/06/01	
14	30	F	59/06/06	REACTIVATED
8	45	M	59/06/12	
30	23	M	59/06/14	
	39	M	59/06/15	
17	47	M	59/06/16	
104	22	F	59/06/22	
29	53	M	59/06/22	DOD 89/08/24
27	20	M	59/06/28	
56	45	M	59/06/29	
27	50	F	59/07/06	REACTIVATED

MACASSAR TB (ALL FORMS) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
56	47	M	59/01/05	
55	22	M	58/01/12	
29	50	M	59/01/12	
13	61	F	58/01/12	REACTIVATED
92	2	M	59/01/19	
20	17	M	59/01/19	
58	25	M	59/01/23	
96	6	M	59/01/26	
91	21	M	59/01/26	
35	34	F	59/01/26	
92	45	M	59/01/26	
22	45	M	59/01/31	
36	32	F	59/02/01	
28	27	M	59/02/07	
32	1	F	59/02/09	
26	24	F	59/02/09	
42	47	M	59/02/10	
2	31	M	59/02/16	
5	36	F	59/02/16	
14	50	M	59/02/22	REACTIVATED
55	22	F	59/02/23	
56	24	F	59/02/23	
22	30	M	59/02/23	REACTIVATED
	1	F	59/03/02	
9	2	M	59/03/02	
43	24	F	59/03/02	REACTIVATED
6	6	M	59/03/09	
24	19	F	59/03/09	REACTIVATED
19	24	M	59/03/09	
12	36	F	59/03/09	
6	38	M	59/03/09	
110	41	M	59/03/09	REACTIVATED
195	62	M	59/03/09	
13	71	F	59/03/09	
86	7	M	59/03/16	
	34	F	59/03/16	REACTIVATED
4	38	M	59/03/16	REACTIVATED?
65	51	M	59/03/16	REACTIVATED
29	54	F	59/03/16	
34	1	M	59/03/20	
152	1	M	59/03/23	REACTIVATED
1	21	M	59/03/23	REACTIVATED
41	25	M	59/03/23	
35	31	M	59/03/23	
26	30	F	59/03/30	
157	32	M	59/03/30	
21	36	M	59/04/03	
24	33	F	59/04/10	REACTIVATED
22	24	F	59/04/11	
169	15	F	59/04/13	
100	19	M	59/04/13	
25	31	F	59/04/13	
21	20	F	59/04/18	DOD 90/01/13 REACT
25	6	F	59/04/20	
16	16	M	59/01/20	
5	67	M	59/01/21	
45	24	M	59/04/26	
9	26	M	59/01/27	
10	26	F	59/04/27	
	51	M	59/04/27	REACTIVATED
11	43	M	59/05/02	
37	5	M	59/05/11	
24	26	M	59/05/11	
12	32	M	59/05/11	
9	44	M	59/05/11	REACTIVATED
13	19	F	59/05/18	
14	30	M	59/05/18	
14	4	M	59/05/22	
11	15	F	59/05/22	
75	22	M	59/05/22	
6	22	M	59/05/22	
355	61	F	59/05/22	REACTIVATED
16	22	M	59/05/29	
44	60	M	59/06/01	
14	30	F	59/06/06	REACTIVATED
6	45	M	59/06/12	
30	23	M	59/06/14	
	39	M	59/06/15	
17	47	M	59/06/16	
104	22	F	59/06/22	
29	53	M	59/06/22	DOD 59/08/24
27	20	M	59/06/29	
56	48	M	59/06/29	
27	56	F	59/07/06	REACTIVATED
111	21	F	59/07/13	
3	16	F	59/07/17	
30	33	M	59/07/20	
	41	F	59/07/20	DOD 59/08/10
26	51	M	59/07/20	DOD 90/06/17
92	39	M	59/08/03	
90	55	F	59/08/03	
27	1	M	59/08/08	
32	2	F	59/08/10	
20	55	M	59/08/10	
92	20	F	59/08/14	
69	23	M	59/08/17	
95	30	F	59/08/28	
32	44	F	59/08/28	REACTIVATED
17	30	F	59/08/29	
7	40	F	59/08/30	
	19	F	59/08/31	
37	19	F	59/09/05	
36	36	M	59/09/07	
102	52	M	59/09/07	
67	32	M	59/09/19	
96	28	F	59/09/21	REACTIVATED
68	27	M	59/09/21	
29	34	F	59/09/21	
36	36	M	59/09/21	
109	18	M	59/09/22	

MACASSAR TB (ALL FORMS) NOTIFICATIONS (1955 -1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
44	25	F	59/09/22	
52	20	M	59/09/27	
63	18	M	59/09/28	
103	42	M	59/09/28	
27	D34	F	59/10/05	
106	25	F	59/10/12	
67	45	F	59/10/12	
122	21	M	59/10/13	
133	30	M	59/10/19	
111	1	M	59/10/26	
11	6	M	59/10/31	
134	30	M	59/10/31	
24	35	M	59/11/02	
18	47	M	59/11/02	
115	32	M	59/11/09	
218	46	M	59/11/09	
46	47	M	59/11/09	
99	48	F	59/11/09	
38	56	M	59/11/09	
139	60	M	59/11/09	
4	57	M	59/11/16	
1	58	M	59/11/16	
29	38	M	59/11/21	
54	17	M	59/11/24	
11	24	M	59/11/30	
15	25	F	59/11/30	
103	21	F	59/12/08	
3	4	F	59/12/14	
22	25	F	59/12/14	
56	37	M	59/12/14	
170	38	F	59/12/14	
15	20	M	59/12/21	
12	27	M	59/12/21	

MACASSAR TB (PULMONARY) NOTIFICATIONS (1955 - 1959)

XRAY NO	AGE	SEX	NOTIFI DATE	OTHER COMMENTS
111	21	F	59/07/13	
30	33	M	59/07/20	
	41	F	59/07/20	DOD 59/08/10
26	51	M	59/07/20	DOD 90/06/17
92	39	M	59/08/03	
90	55	F	59/08/03	
20	55	M	59/08/10	
92	20	F	59/08/14	
69	23	M	59/08/17	
95	30	F	59/08/28	
32	44	F	59/08/28	REACTIVATED
17	30	F	59/08/29	
7	40	F	59/08/30	
	19	F	59/08/31	
37	19	F	59/09/05	
36	36	M	59/09/07	
102	52	M	59/09/07	
67	32	M	59/09/19	
96	28	F	59/09/21	REACTIVATED
68	27	M	59/09/21	
29	34	F	59/09/21	
36	36	M	59/09/21	
109	18	M	59/09/22	
44	25	F	59/09/22	
52	20	M	59/09/27	
63	18	M	59/09/28	
103	42	M	59/09/28	
27	D34	F	59/10/05	
106	25	F	59/10/12	
67	45	F	59/10/12	
122	21	M	59/10/13	
133	30	M	59/10/19	
134	30	M	59/10/31	
24	35	M	59/11/02	
18	47	M	59/11/02	
115	32	M	59/11/09	
218	46	M	59/11/09	
46	47	M	59/11/09	
99	48	F	59/11/09	
38	56	M	59/11/09	
139	60	M	59/11/09	
4	57	M	59/11/16	
1	58	M	59/11/16	
29	38	M	59/11/21	
11	24	M	59/11/30	
15	25	F	59/11/30	
103	21	F	59/12/08	
22	25	F	59/12/14	
56	37	M	59/12/14	
170	38	F	59/12/14	
15	20	M	59/12/21	
12	27	M	59/12/21	

APPENDIX E7

FORMULA FOR CALCULATION OF:

RELATIVE RISK (RR)

ATTRIBUTABLE RISK (AR) & ATTRIBUTABLE RISK % (AR%)

NOTE:

Relative risk was calculated instead of Odds Ratio (OR), since this was not a case control study

RELATIVE RISK (RR)

Relative Risk: the risk ratio in comparing in how much more likely is one group to become infected (Mantoux positive) than the uninfected group (Mantoux negative)

$$RR \cong \frac{(a/a+b)}{(c/c+d)}$$

ATTRIBUTABLE RISK (AR)

Attributable risk: measure increased risk due to exposure and hence quantifies the amount of disease (infected - Mantoux positive) in the exposed group that could be considered attributable to (due to) THE exposure.

AR is very important (and underused) measure of effect. It can be called a measure of Public Health importance of exposure: 'if we eliminate this exposure how much disease will we prevent in the people exposed?'.

$$AR = \frac{a}{a+b} - \frac{c}{c+d}$$

ATTRIBUTABLE RISK % (AR%)

Attributable Risk %: measures the proportion of (infected - Mantoux positives) among the exposed that is due to the exposure

$$AR\% = \frac{(RR - 1)}{RR} \times \frac{100}{1}$$

APPENDIX F1

FORMULA FOR CALCULATION OF KAPPA STATISTIC

Calculation of Kappa Statistic:

a1 = n1/n = 19/209 = 0,043

a2 = n2/n = 190/209 = 0,909

b1 = n3/n = 67/209 = 0,301

b2 = n4/n = 142/209 = 0,679

Pe = [a1 X b1] + [a2 X b2]

= [0,043 X 0,301] + [0,909 x 0,679]

= [0,013] = [0,617]

= 0,630

Po = (a+d)/n = (7+130)/209 = 0,656

K = [Po - Pe]/[1 - Pe]

= [0,656 - 0,630]/[1 - 0,630]

= [0,026]/[0,370]

= **0,070**

Guidelines for the Evaluation of Kappa Statistic

k > 0.75	-	Excellent reproducibility
0.4 <= k <= 0.75	-	Good reproducibility
0 <= k < 0.04	-	Marginal reproducibility

SOURCES:

Rosner, B. Fundamentals of Biostatistics, 3rd ed, PWS-Kent Publishing Co., Boston, Massachusetts, 1986; p455 - 458

Fleiss, JL. Statistical Methods for Rates and Proportions, A Wiley-Interscience Publication, John Wiley & Sons, New York, 1973; p 146

APPENDIX F2

FORMULA FOR CALCULATION OF PREVALENCE OF DISEASE DIAGNOSED, SENSITIVITY, SPECIFICITY, POSITIVE PREDICTIVE VALUE AS WELL AS THE PREDICTIVE VALUE OF THE SCREENING TEST

(a) FORMULA FOR % PREVALENCE:

$$\frac{\text{number of persons classified ill \& are ill}}{\text{total number of person in the population tested}}$$

(b) FORMULA FOR % SENSITIVITY

$$\frac{\text{number of ill classified ill}}{\text{total number of ill persons}}$$

(c) FORMULA FOR % SPECIFICITY

$$\frac{\text{number of healthy classified healthy}}{\text{total number of healthy persons}}$$

(d) FORMULA FOR POSITIVE PREDICTIVE VALUE (PPV) OF A POSITIVE TEST RESULT

$$\frac{\text{those truly ill and classified ill by test result}}{\text{those persons classified ill by test result}}$$

(e) FORMULA FOR PREDICTIVE VALUE

$$\text{PREDICTIVE VALUE} = \frac{P \times \text{SENSITIVITY}}{(P \times \text{SENSITIVITY}) + (1-P) \times (1-\text{SPECIFICITY})}$$

With P = Prevalence of Disease (PTB): 1 355 per 100 00

SOURCE:

Ahlbom, Anders and Norrel, Steffan; National Institute of Environmental Medicine, Stockholm, Sweden. Introduction to Modern Epidemiology. 1984; Ch4, p23-28.

EPIDEMIOLOGY OF TB IN MACASSAR CAMP

NUTRITIONAL STATUS OF CHILDREN AGED 14 YEARS OR LESS

IDNUM	AGE	SEX	HAZ	HAP	HAM	WAZ	WAP	WAM	WHZ	WHP	WHM
8604	2	F	-3.9	0	55.22	-3.4	0.03	66.12	-1.2	10.23	86.78
8004	2	F	-2.0	2.15	92.32	-2.38	0.86	76.3	-1.3	9.14	86.73
1507	2	M	-0.1	42.6	99.31	0.51	69.6	106.95	0.74	77.15	105.92
1406	2	M	0.51	69.3	101.69	0.37	63.06	104.52	91.1	67.73	102.25
808	3	F	2.21	95.6	105.72	-0.41	34.1	95.71	-1.7	4.23	84.74
7205	3	M	2.39	99.1	109.57	0.76	77.57	109.41	-0.5	25.08	94.95
1503	4	M	0.01	50.4	100.05	0.15	55.94	101.63	0.28	60.85	102.61
4906	4	F	0.64	73.7	102.53	0.9	81.73	113.41	0.9	81.66	109.61
5205	5	M	-1.7	4.23	92.79	0.13	44.86	98.54	1.36	91.35	112.95
7812	5	F	-1.6	4.74	93.19	-0.56	19.45	90.58	0.22	58.64	102.34
7705	5	F	-1.2	11.1	95.04	-0.08	19.45	90.55	-0.1	45.04	95.9
3409	5	M	-0.6	24.8	97.16	0.38	64.93	104.96	1.16	87.7	110.92
1404	5	F	0	50.1	100.02	0.98	83.71	116.48	1.4	91.89	114.98
6103	5	M	2.43	99.2	110.16	1.59	94.4	120.49	-0.0	47.12	99.41
5204	6	F	-2.1	1.78	90.99	0.62	73.28	110.63	2.96	99.79	130.58
203	6	M	-1.9	2.65	91.91	-0.04	48.54	99.59	1.78	96.27	116.78
3407	6	F	-1.7	4.13	92.56	0.95	82.91	116.27	3.09	99.8	133.07
7706	6	F	0.49	68.4	102.06	0.14	55.69	102.44	-0.3	36.02	96.93
6305	6	F	0.89	81.2	103.81	0.35	63.76	106.02	-0.3	34.86	96.67
5905	7	F	-1.5	5.57	92.89	4.59	99.8	187.72	9.98	99.8	217.21
204	7	M	-1.0	14.0	95.45	-0.63	26.5	92.77	0.21	58.16	102.12
1205	7	F	0.9	81.5	104.08	-1.03	15.1	86.99	-2.4	0.63	78.04
3605	7	M	1.24	89.2	105.18	-0.32	37.3	96.27	-1.7	3.82	85.5
2507	8	F	-0.3	37.7	98.51	-0.82	20.67	89.55	-0.5	18.73	92.24
4303	8	F	0.27	60.5	101.28	0.03	51.16	100.63	-0.2	38.7	97.41
3406	8	M	0.62	73.3	102.62	0.75	77.42	113.85	0.52	69.94	107
4910	8	M	1.41	92.0	105.93	-0.1	46.19	98.83	-1.6	4.61	85.3
1303	9	M	-5.6	.0	75.66	0.15	55.85	103.08	8.73	99.8	184.15
6504	9	M	-1.9	2.45	91.55	-0.83	20.43	88.56	0.95	82.97	110.65
8105	9	M	-1.5	6.33	93.45	-3.2	0.07	56.87	-3.9	0	67.67
1403	9	M	-1.0	13.5	95.34	0.13	55.19	102.73	1.35	91.19	116.68
6007	9	M	-0.9	18.1	96.09	-1.09	13.79	85.31	-0.6	27.29	95.08
5509	9	M	-0.3	35.1	98.36	-0.83	20.43	88.56	-0.7	21.42	93.44
9405	9	M	-0.0	43.8	99.88	1.16	87.8	124.41	1.86	96.85	125.69

EPIDEMIOLOGY OF TB IN MACASSAR CAMP

NUTRITIONAL STATUS OF CHILDREN AGED 14 YEARS OR LESS

IDNUM	AGE	SEX	HAZ	HAP	HAM	WAZ	WAP	WAM	WHZ	WHP	WHM
3105	9	M	0.15	55.8	100.63	-0.04	48.6	99.53	-0.1	43.17	98.53
6506	9	F	0.58	71.8	102.85	-0.79	21.35	87.84	-1.8	3.3	81.24
4603	9	F	0.81	79.0	103.95	-0.11	45.79	95.35	9.99	99.9	999.9
7005	10	F	-0.3	36.7	95.33	0.05	52.17	101.33	9.99	99.9	999.9
4913	10	F	0.98	83.6	104.84	-1.23	10.92	79.59	9.99	99.9	999.9
8509	11	F	-4.1	0	80.12	-0.15	43.94	97.42	9.9	99.9	999.9
6510	11	F	-3.2	0.05	84.26	-2.71	0.33	54.12	9.99	99.9	999.9
5904	11	F	-1.8	3.39	91.24	-1.75	3.97	70.36	9.99	99.9	999.9
8104	11	M	-1.2	10.8	94.23	-1.31	9.53	79.32	-0.6	26.76	94.54
2506	11	M	-0.0	48.3	99.81	-0.14	44.29	97.73	-0.2	41.86	97.89
6505	11	M	0.26	60.1	101.21	-0.68	24.78	88.23	-1.3	9.1	85.4
4306	11	M	1.45	92.6	106.79	0.37	64.32	109.06	9.99	99.9	999.9
5503	11	F	1.9	97.1	109.13	0.52	69.78	113.66	9.99	99.9	999.9
6607	11	M	2.2	98.6	110.29	1.67	94.2	138.81	9.99	99.9	999.9
6304	12	F	-1.5	6.16	93.06	-1.07	14.26	81.56	9.9	9.99	999.9
1506	12	F	-0.6	25.3	97.02	-0.57	28.36	90.29	9.99	99.9	999.9
1406	12	F	-0.6	25.8	97.08	0.22	58.6	105.7	9.99	99.9	999.9
4909	12	M	-0.4	31.3	97.56	0.02	50.88	100.56	9.99	99.9	999.9
4903	12	F	-0.4	31.3	97.81	0.97	83.4	125.45	9.99	99.9	999.9
9404	12	F	0.66	74.4	102.96	0.32	62.49	108.35	9.99	99.9	999.9
7108	13	F	-1.8	3.23	92.25	-1.78	3.77	70.51	9.99	99.9	999.9
1604	13	M	-0.9	18.3	95.23	-0.82	20.73	86.76	9.99	99.9	999.9
6905	13	M	-0.7	21.7	95.87	-0.95	17.04	84.54	9.99	99.9	999.9
7204	13	M	-0.7	21.7	95.87	-0.4	34.29	93.43	9.99	99.9	999.9
8306	13	F	-0.1	43.2	99.28	0.16	56.49	104.13	9.99	99.9	999.9
7307	13	M	1.76	96.0	109.29	-0.4	34.29	93.43	9.99	99.9	999.9
3604	14	M	-3.8	0.01	79.69	-0.16	43.63	97.5	9.99	99.9	999.9
1103	14	F	-1.7	4.46	92.92	-0.66	25.51	89.5	9.99	99.9	999.9
6004	14	M	-1.5	6.26	91.96	-2.12	1.72	66.97	9.99	99.9	999.9
1304	14	F	-1.2	10.5	94.79	-0.28	38.8	95.47	9.99	99.9	999.9
6303	14	M	-1.1	11.8	93.79	-0.22	41.16	96.51	9.99	99.9	999.9
4605	14	F	-0.9	17.0	96.03	0.89	81.25	121.32	9.99	99.9	999.9
403	14	F	-0.2	40.7	99.03	0.14	55.66	103.42	9.99	99.8	999.9

APPENDIX F3a

NUTRITIONAL STATUS OF CHILDREN AGED <= 14 YEARS IN MACASSAR CAMP, BASED ON Z-SCORES, MEDIAN & PERCENTILE OF HEIGHT FOR AGE. WEIGHT FOR AGE AND WEIGHT FOR HEIGHT

MOHAMMED,A.:Epidemiological study of tuberculosis
in Macassar Camp
M.Sc. Med.Sc. Stellenbosch Dec. 1995 10/10

NUTRITIONAL STATUS OF RESIDENTS AGED >= 15 YEARS IN MACASSAR CAMP, BASED ON BODY MASS INDEX (BMI).

EPIDEMIOLOGY OF TB IN MACASSAR CAMP

NUTRITIONAL STATUS OF RESIDENTS AGED 15 YEARS OR OLDER

IDNUM	AGE	SEX	HT (M)	WT (KG)	BMI
8103	15	F	1.56	46	18.9
4909	15	M	1.67	47	16.9
7505	15	M	1.558	43.4	17.9
8507	15	F	1.64	65	24.2
9403	15	M	1.58	47.5	19.0
1505	15	M	1.65	51.7	19.0
5903	15	F	1.32	33	18.9
6904	16	F	1.62	59	22.1
7501	16	F	1.471	52.4	24.2
7401	16	M	1.511	41.1	18.0
7607	16	F	1.575	39	15.3
7203	16	F	1.61	52	20.1
8403	16	M	1.69	69	23.8
9103	17	F	1.6	92	35.9
4305	17	F	1.63	51.5	19.4
8204	17	F	1.622	52.2	19.8
505	17	M	1.78	60	18.9
4911	17	M	1.779	49.9	15.8
2504	17	F	1.6	73	28.5
8305	17	F	1.56	41	16.1
1501	17	F	1.583	62.9	25.1
3104	17	M	1.599	67	26.2
5305	17	F	1.47	46.5	21.5
6003	17	M	1.72	55	18.6
7105	17	F	1.46	41	19.2
7003	18	F	1.57	47	19.1
505	18	F	1.371	39.4	21.0
1603	18	F	1.56	50.1	20.7
7709	18	M	1.65	61	22.4
303	18	M	1.64	60	22.3
6805	18	F	1.605	55	21.4
9506	19	F	1.59	45	17.8
7605	19	F	1.65	51	19.1
3701	19	F	1.61	60	23.1
1107	19	M	1.558	45.9	20.1
2201	19	F	1.638	50.1	18.7
7101	19	M	1.541	42.5	18.0
7506	19	F	1.37	40	21.3
2301	19	F	1.65	66	24.2
3203	20	F	1.61	70	27.0
8105	20	M	1.676	64	22.8
7507	20	M	1.9	81	17.7
807	20	M	1.75	62	19.6
8505	20	M	1.753	68.3	22.2
7504	21	F	1.437	39.2	19.0
7403	21	M	1.73	55.4	18.5
7601	21	M	1.67	55.6	19.9
6603	21	F	1.63	55.5	20.9
3704	21	M	1.674	60	21.4
5602	22	M	1.71	61	17.4
9504	22	F	1.676	56.7	20.2
504	22	M	1.65	57	20.9
9107	23	F	1.58	85	34.0
9104	23	F	1.606	83.9	32.5
7303	23	M	1.8	74.9	23.1
6206	23	F	1.59	49	19.4
7610	23	M	1.685	56.3	19.8
5503	23	M	1.8	62.5	19.3
4904	23	F	1.71	95	32.5
1705	24	F	1.474	42.4	19.5
6005	24	F	1.6	58.6	22.9
6006	24	F	1.65	62.9	23.1
7703	24	F	1.625	119	45.1
1408	24	M	1.192	57	40.1
7304	24	M	1.85	74.2	21.7
7103	24	F	1.523	53.4	23.0
7704	25	M	1.765	67.5	21.7
503	25	F	1.196	50.7	22.7
8601	25	F	1.44	66.3	32.0
4301	25	F	1.665	74.1	26.7
5403	25	M	1.74	55.5	18.3
7202	26	M	1.62	49.7	21.5
6009	26	F	1.521	51.3	22.2
7707	26	F	1.61	89	34.3
1704	26	F	1.78	63	19.9
5005	27	F	1.61	44	17.0
7710	27	M	1.58	55.2	22.1
5602	27	M	1.61	51	19.7
6502	27	M	1.75	73.5	24.0
3504	27	F	1.452	47.9	17.2
5001	27	M	1.651	68.5	25.1
6107	25	F	1.7	76.5	26.5
6202	25	F	1.63	66	20.6
5503	25	F	1.541	52.1	21.8
7901	25	F	1.61	70.7	27.3
4302	28	M	1.61	65	25.1
6501	29	F	1.59	53.9	21.3
5902	29	F	1.636	53.1	19.5
202	30	F	1.64	55	20.1
6102	30	F	1.622	85	33.4
9101	30	F	1.656	44.6	16.3
7904	30	F	1.48	49	22.4
1505	30	F	1.25	67	42.9
5303	31	F	1.63	93	35.0

EPIDEMIOLOGY OF TB IN MACASSAR CAMP

NUTRITIONAL STATUS OF RESIDENTS AGED 15 YEARS OR OLDER

IDNUM	AGE	SEX	HT (M)	WT (KG)	BMI
7201	31	F	1.705	75	25.8
1501	31	M	1.7	60.7	21.0
6201	31	M	1.72	62.5	21.1
6204	32	M	1.75	90	29.4
5107	32	M	1.69	67	23.5
5401	32	M	1.803	71	21.8
5902	32	F	1.54	67.5	28.5
402	32	F	1.623	60.4	22.9
6301	32	F	1.569	64	26.0
5901	32	F	1.626	45	19.3
5203	32	F	1.526	68	29.2
201	32	M	1.673	83.5	29.8
8102	33	M	1.71	58.5	20.0
1502	33	F	1.66	49.5	18.0
1703	33	F	1.612	57.2	22.0
3103	33	M	1.8	68.5	21.1
7701	33	M	1.701	70	24.2
1401	35	M	1.703	75.6	26.1
7501	35	F	1.56	84	34.5
6101	35	F	1.65	84.5	31.0
1801	35	M	1.78	77	24.3
5502	36	F	1.603	44.4	17.3
4907	36	F	1.617	66	25.2
1602	36	F	1.56	46	18.9
5106	37	M	1.76	59	19.0
6902	37	F	1.6	76.5	29.9
2502	37	F	1.62	108	41.2
5101	37	F	1.22	49	32.9
5303	37	M	1.854	71.1	20.7
302	38	M	1.65	67.2	24.7
6403	38	M	1.615	68.3	26.2
6302	38	M	1.61	55.7	21.5
9102	38	M	1.795	86.5	26.5
5201	38	M	1.715	70	23.8
5202	38	F	1.554	58.3	24.6
1102	10	F	1.68	85	30.1
5201	10	M	1.666	69.2	24.9
3701	40	F	1.495	56.7	25.3
4801	40	F	1.61	78.5	30.3
501	41	F	1.59	65.5	25.9
1301	12	M	1.4	75.7	38.6
7102	42	F	1.525	42.1	18.0
5102	42	M	1.65	67.6	24.8
7602	42	F	1.591	66.7	26.7
1509	42	F	1.549	60.5	25.2
2501	43	M	1.72	55	18.6
7401	44	M	1.66	77.5	28.1
6901	44	M	1.62	41	16.8
6601	44	F	1.64	117	43.5
6001	45	F	1.57	51	20.7
7302	45	F	1.676	55	20.6
8307	45	F	1.58	49	19.6
3401	46	M	1.704	77.2	26.6
2202	46	F	1.56	72	29.6
9701	47	F	1.714	67	22.9
7601	47	M	1.65	48.7	17.9
9108	47	F	1.65	70	25.7
6602	48	M	1.647	61.8	22.8
1201	49	M	1.661	80.6	29.2
3101	49	M	1.944	62.4	30.1
9407	49	M	1.616	52.9	20.2
502	49	F	1.602	67.2	26.2
3702	50	M	1.73	54.1	18.1
6205	50	F	1.595	42.7	16.8
7805	51	F	1.45	40	19.0
3602	51	M	1.736	67.1	22.4
7301	51	M	1.84	63.2	18.7
2201	52	M	1.661	69.5	25.2
9402	53	F	1.465	57.4	26.8
8501	53	M	1.72	60.5	20.5
9106	53	M	1.73	64.6	21.6
6501	53	F	1.65	63	23.1
501	53	M	1.737	54.2	27.9
1701	57	F	1.494	60.9	27.3
1901	57	M	1.702	57	30.0
5206	57	M	1.78	86.2	26.9
5501	57	F	1.549	52	21.7
7101	55	M	1.622	71.9	27.3
1702	58	M	1.702	46	15.9
5105	59	M	1.717	82.5	31.1
5104	60	M	1.684	63.5	22.1
7501	62	M	1.5	40.2	17.9
1101	63	F	1.463	41	19.2
9301	63	F	1.6	71	27.7
5101	63	M	1.683	89	31.1
7502	65	M	1.641	52.9	19.6
6502	70	M	1.676	77	27.4
101	72	M	1.85	115	33.6

RESULTS OF TB ELISA & MANTOUX TESTS OF MACASSAR CAMP POPULATION

IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE	IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE	IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE	IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE
7205	0.057	0	N	1	M	C	5504	0.112	9	N	22	F	C	8601	0.125	12.7	N	25	F	C	5306	0.07	15.5	Y	13	F	C
1503	0.05	0	N	4	M	C	7501	0.127	9	N	62	M	C	5002	0.139	12.7	Y	29	F	C	503	0.179	15.5	N	25	F	C
7512	0.013	0	N	5	F	C	7904	0.203	9.1	N	30	F	C	6205	0.045	12.7	N	50	I	C	1703	0.071	15.5	N	33	F	C
7706	0.013	0	N	6	F	C	1002	0.075	9.1	N	30	F	C	5501	0.048	12.7	N	53	M	C	9701	0.103	15.5	Y	47	F	C
3107	0.061	0	N	6	F	B	3703	0.171	9.2	N	19	F	C	1302	0.055	12.8	N	29	M	C	7503	0.031	15.9	N	20	M	C
5905	0.079	0	N	7	F	C	7301	0.356	9.2	Y	51	M	C	5901	0.172	12.8	N	32	F	C	501	0.109	16	Y	53	M	C
4910	0.085	0	N	9	M	C	7403	0.271	9.3	N	21	M	C	5101	0.432	12.8	Y	63	M	B	1601	0.045	16.3	Y	35	M	C
2507	0.026	0	N	9	F	C	402	0.1	9.3	N	32	F	C	6303	0.134	12.9	N	11	M	C	6302	0.322	16.3	Y	36	M	C
1303	0.107	0	N	9	F	C	1205	0.137	9.5	N	7	F	C	7602	0.266	12.9	N	42	F	C	6009	0.126	16.4	N	26	F	C
5105	0.069	0	N	9	M	C	5506	0.016	9.5	N	18	F	C	1702	1.002	12.9	N	56	M	C	1101	0.061	16.4	N	63	F	C
5509	0.154	0	N	9	M	C	5301	0.526	9.5	N	63	F	C	7404	0.13	13.1	N	16	M	C	1603	0.052	16.6	N	18	F	C
4603	0.045	0	N	9	F	B	202	0.11	9.6	N	30	F	C	6901	0.063	13.1	N	44	M	C	5510	0.056	16.7	N	11	F	C
6504	0.051	0	N	9	M	C	1509	0.062	9.6	N	42	F	C	7604	0.155	13.3	N	21	M	C	6902	0.105	16.7	N	37	F	C
1303	0.055	0	N	9	M	B	9103	0.243	9.5	N	17	F	B	8303	0.202	13.3	N	31	F	C	5501	0.125	16.7	N	57	F	C
3105	0.125	0	N	9	M	C	1502	0.025	9.5	N	33	F	C	7710	0.004	13.3	N	27	M	C	5902	0.263	16.5	N	32	F	C
7005	0.101	0	N	10	F	C	1301	0.146	9.8	N	42	M	B	505	0.145	13.4	N	17	M	C	5303	0.577	16.5	N	37	F	C
2506	0.19	0	N	11	M	C	9404	0.076	10	N	12	F	C	9102	0.077	13.4	N	38	M	B	9107	0.061	16.9	N	23	F	B
4306	0.126	0	N	11	M	C	7104	0.049	10	N	19	M	C	5105	0.233	13.4	N	59	M	B	5502	0.515	16.9	N	36	F	C
5104	0.1	0	N	11	M	C	5108	0.2	10	N	20	M	C	4605	0.124	13.5	N	14	F	C	1501	0.102	17.1	N	31	F	M
4509	0.064	0	N	12	M	C	7605	0.072	10.1	N	19	F	C	5507	0.128	13.5	N	16	F	C	6403	0.076	17.2	N	38	M	M
1506	0.027	0	N	12	F	C	6301	0.072	10.1	N	32	F	C	5602	0.089	13.5	N	27	M	C	3104	0.106	17.3	N	17	M	C
6905	0.061	0	N	13	M	C	7401	0.055	10.1	N	44	M	C	9407	0.259	13.5	Y	49	M	C	7707	0.153	17.5	N	26	F	C
1504	0.034	0	N	13	M	C	6107	0.062	10.2	N	28	F	B	507	0.207	13.6	N	20	M	C	5508	0.155	17.8	N	11	F	M
7204	0.059	0	N	13	M	C	4911	0.181	10.4	N	17	M	C	7303	0.166	13.6	N	23	M	C	303	0.072	17.5	N	18	M	F
6004	0.053	0	N	14	M	C	7708	0.049	10.5	N	5	F	C	7703	0.022	13.6	N	24	F	C	6602	0.12	17.8	N	45	M	F
7607	0.053	0	N	16	F	C	7302	0.221	10.5	N	45	F	C	7202	0.052	13.7	Y	26	M	C	5503	0.11	18	N	29	F	C
7105	0.07	0	N	17	F	C	8307	0.132	10.5	N	45	F	C	6001	0.02	13.7	Y	45	F	C	6102	0.044	18.1	N	30	F	M
7003	0.017	0	N	18	F	C	4908	0.078	10.6	N	15	M	C	5505	0.072	13.9	N	20	M	C	5107	0.04	18.1	N	32	M	M
2503	0.176	0	N	19	F	C	7103	0.066	10.6	N	24	F	C	7704	0.028	14	N	25	M	C	5102	0.133	18.1	Y	42	M	M
504	0.226	0	N	22	M	C	8403	0.099	10.7	Y	16	M	C	3101	0.125	14	N	49	M	C	3602	0.1	18.1	N	51	M	F
4904	0.225	0	N	23	F	C	6304	0.092	11	N	12	F	C	5201	0.223	14.2	N	38	M	C	6605	0.057	18.5	Y	18	F	C
8005	0.138	0	N	27	F	C	7709	0.027	11.1	N	18	M	C	5103	0.27	14.2	Y	50	M	B	3701	0.179	18.8	Y	40	F	C
6202	0.152	0	N	28	F	C	301	0.092	11.2	N	37	F	C	6007	0.144	14.5	N	9	M	C	6006	0.082	19.2	N	24	F	C
7201	0.054	0	N	31	F	C	4907	0.103	11.3	N	36	F	C	6605	0.056	14.5	Y	17	M	C	3401	0.073	19.5	N	46	M	F
6505	0.175	3.5	N	11	M	C	5904	0.053	11.4	N	11	F	C	6501	0.1	14.5	N	53	F	C	8103	0.065	20	N	15	F	C
7203	0.065	3.5	N	16	F	C	2504	0.124	11.4	N	17	F	C	5401	0.117	14.6	Y	32	M	B	8305	0.117	20.1	N	17	F	C
6502	0.147	3.5	N	27	M	C	3702	0.235	11.4	N	50	M	C	7601	0.057	14.6	N	47	M	C	9101	0.517	20.6	N	30	F	C
6501	0.13	3.6	N	29	F	C	7402	0.051	11.6	Y	42	F	C	7101	0.014	14.6	Y	55	M	C	6603	0.049	21	N	21	F	C
1103	0.223	3.6	N	14	F	C	1201	0.056	11.6	N	49	M	B	6005	0.093	14.7	N	24	F	C	1704	0.496	21.2	Y	26	F	M
7506	0.22	3.7	N	19	F	C	502	0.305	11.6	N	49	F	C	5106	0.111	14.7	N	37	M	B	5202	0.163	21.7	Y	65	M	F
7702	0.164	3.8	N	58	F	C	403	0.061	11.7	N	14	F	C	5101	0.196	15	N	37	F	C	1304	0.179	21.8	N	14	F	C
9405	0.097	3.9	N	9	M	C	7508	0.131	11.8	N	16	M	C	302	0.044	15	N	35	M	C	1203	0.153	22.6	N	28	F	C
2202	0.261	4	N	16	F	B	5602	0.104	11.9	N	22	M	C	7505	0.155	15	N	51	F	C	5504	0.323	22.9	Y	27	F	C
4913	0.065	4.5	N	10	F	C	5202	0.044	11.9	Y	38	F	C	3103	0.193	15.1	Y	33	M	C	7901	0.136	22.9	Y	26	F	C
6904	0.065	5	N	16	F	C	2201	0.019	12	N	52	M	B	7504	0.089	15.2	N	21	F	C							
4305	0.124	5.7	N	17	F	C	101	0.159	12	N	72	M	W	7610	0.233	15.2	Y	23	M	C							
1701	0.051	6.5	N	57	F	C	6001	0.112	12.2	N	27	M	C	1705	0.049	15.2	N	24	F	C							
6502	0.257	7.2	N	70	M	C	5903	0.192	12.3	N	15	F	C	1405	0.065	15.2	N	24	M	C							
7307	0.052	7.5	N	13	M	C	3402	0.114	12.4	N	40	F	B	6201	0.039	15.2	N	31	M	C							
5605	0.207	7.5	N	17	F	C	7304	0.065	12.5	N	24	M	C	6101	0.102	15.2	N	35	F	B							
6204	0.05	8	N	32	M	C	1505	0.089	12.5	N	30	F	C	2502	0.137	15.4	N	37	F	C							
7501	0.037	8.4	N	35	F	C	5201	0.116	12.5	N	40	M	B	4601	0.083	15.4	Y	40	F	B							
4301	0.151	8.7	N	25	F	C	3704	0.193	12.6	N	21	M	C	9108	0.101	15.4	N	47	F	B							
8503	0.1	8.8	N	23	M	C	7701	0.043	12.6	N	33	M	C	2501	0.197	15.5	N	43	M	C							
6206	0.121	8.9	N	23	F	C	5102	0.105	12.6	N	33	M	C	5104	0.103	15.5	Y	60	M	B							

RESULTS OF TB ELISA & MANTOUX TESTS OF MACASSAR CAMP POPULATION

IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE	IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE	IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE	IDNUM	ELISA OD	72HR MX	VES Y/N	AGE	SEX	RACE
8601	0.125	12.7	N	25	F	C	5306	0.07	15.5	Y	13	F	C													
5002	0.139	12.7	Y	29	F	C	503	0.179	15.5	N	25	F	C													
6205	0.045	12.7	N	50	I	C	1703	0.071	15.5	N	33	F	C													
5501	0.048	12.7	N	53	M	C	9701	0.103	15.5	Y	47	F	C													
1302	0.055	12.8	N	29	M	C	7503	0.031	15.9	N	20	M	C													
5901	0.172	12.8	N	32	F	C	501	0.109	16	Y	53	M	C													
5101	0.432	12.8	Y	63	M	B	1601	0.045	16.3	Y	35	M	C													
6303	0.134	12.9	N	11	M	C	6302	0.322	16.3	Y	36	M	C													
7602	0.266	12.9	N	42	F	C	6009	0.126	16.4	N	26	F	C													
1702	1.002	12.9	N	56	M	C	1101	0.061	16.4	N	63	F	C													
7404	0.13	13.1	N	16	M	C	1603	0.052	16.6	N	18	F	C													
6901	0.063	13.1	N	44	M	C	5510	0.056	16.7	N	11	F	C													
7604	0.155	13.3	N	21	M	C	6902	0.105	16.7	N	37	F	C													
8303	0.202	13.3	N	31	F	C	5501	0.125	16.7	N	57	F	C													
7710	0.004	13.3	N	27	M	C	5902	0.263	16.5	N	32	F	C													
505	0.145	13.4	N	17	M	C	5303	0.577	16.5	N	37	F	C													
9102	0.077	13.4	N	38	M	B	9107	0.061	16.9	N	23	F	B													
5105	0.233	13.4	N	59	M	B	5502	0.515	16.9	N	36	F	C													
4605	0.124	13.5	N	14	F	C	1501	0.102	17.1	N	31	F	M													
5507	0.128	13.5	N	16	F	C	6403	0.076	17.2	N	38	M	M													
5602	0.089	13.5	N	27	M	C	3104	0.106	17.3	N	17	M	C													
9407	0.259	13.5	Y	49	M	C	7707	0.153	17.5	N	26	F	C													
507	0.207	13.6	N	20	M	C	5508	0.155	17.8	N	11	F	M													
7303	0.166	13.6	N	23	M	C	303	0.072	17.5	N	18	M	F													
7703	0.022	13.6	N	24	F	C	6602	0.12	17.8	N	45	M	F													
7202	0.052	13.7	Y	26	M	C	5503	0.11	18	N	29	F	C													
6001	0.02	13.7	Y	45	F	C	6102	0.044	18.1	N	30	F	M													
5505	0.072	13.9	N	20	M	C	5107	0.04	18.1	N	32	M	M													
7704	0.028	14	N	25	M	C	5102	0.133	18.1	Y	42	M	M													
3101	0.125	14	N	49	M	C	3602	0.1	18.1	N	51	M	F													
5201	0.223	14.2	N	38	M	C	6605	0.057	18.5	Y	18	F	C													
5103	0.27	14.2	Y	50	M	B	3701	0.179	18.8	Y	40	F	C													
6007	0.144	14.5	N	9	M	C	6006	0.082	19.2	N	24	F	C													
6605	0.056	14.5	Y	17	M	C	3401	0.073	19.5	N	46	M	F													
6501	0.1	14.5	N	53	F	C	8103	0.065	20	N	15	F	C													
5401	0.117	14.6	Y	32	M	B	8305	0.117	20.1	N	17	F	C													
7601	0.057	14.6	N	47	M	C	9101	0.517	20.6	N	30	F	C													
7101	0.014	14.6	Y	55	M	C	6603	0.049	21	N	21	F	C													
6005	0.093	14.7	N	24	F	C	1704	0.496	21.2	Y	26	F	M													
5106	0.111	14.7	N	37	M	B	5202	0.163	21.7	Y	65	M	F													
5101	0.196	15	N	37	F	C	1304	0.179	21.8	N	14	F	C													
302	0.044	15	N	35	M	C	1203	0.153	22.6	N	28	F	C													
7505	0.155	15	N	51	F	C	5504	0.323	22.9	Y	27	F	C													
3103	0.193	15.1	Y	33	M	C	7901	0.136	22.9	Y	26	F	C													
7504	0.089	15.2	N	21	F	C																				
7610	0.233	15.2	Y	23	M	C																				
1705	0.049	15.2	N	24	F	C																				
1405	0.065	15.2	N	24	M	C																				
6201	0.039	15.2	N	31	M	C																				
6101	0.102	15.2	N	35	F	B																				
2502	0.137	15.4	N	37	F	C																				
4601	0.083	15.4	Y	40	F	B																				
9108	0.101	15.4	N	47	F	B																				
2501	0.197	15.5	N	43	M	C																				
5104	0.103	15.5	Y	60	M	B																				

IDNUM	PREG	MINI XRAY RSLT	AGE	SEX	NUTRI STAT MED	RACE	M/STAT	OCCUP STAT	CAMP YEARS	YRS @ COMPY	WORK CAT	NO.OF LIVE BIRTH	NO.OF MISCAR	NO.OF INFANT DEATH	HBP	ASMT ALLG	TB	DIAB	ABDOM OP	SMOKE	AGE @ TIME OF SMOKING	NO.OF CIGAR. DAILY	COUGH
6301		NAD	32	F	1	C	1	3	9	2	5	3	0	0	Y	N	N	N	N	Y	20	1	N
1701		NAD	57	F	-1	C	5	3	7	10	5	2	1	0	Y	N	N	N	N	N			Y
3703		NAD	19	F	1	C	6	2	15			0	0		N	N	N	N	N	N			N
403		NAD	14	F	1	C	6	2	5						N	N	N	N	N	N			N
8305		NAD	17	F	-2	C	6	2	10			0	0		N	N	N	N	N	N			N
505		NAD	17	M	-2	C	6	2	17						N	N	N	N	N	Y	16	1	N
6603		NAD	17	M	-2	C	6	2	11						N	N	N	N	N	N			N
7105		NAD	17	F	-2	C	6	2	17			0	0		N	N	N	N	N	Y	16	1	N
4903		NAD	15	M	-2	C	6	2	15						N	N	N	N	N	Y	14	1	N
4605		NAD	14	F	1	C	6	2	11						N	N	N	N	N	N			N
5903		NAD	15	F	-2	C	6	2	15			0	0		N	Y	N	N	N	N			N
6305		NAD	6	F	0	C	6	2	6						N	N	N	N	N	N			N
6904		NAD	16	F	0	C	6	2	16			0	0		N	N	N	N	N	N			N
2503		NAD	19	F	1	C	6	2	19			0	0		N	N	N	N	N	N			N
8102		NAD	33	M	-2	C	1	3	12	13	4				N	N	N	N	N	N			N
8104		NAD	11	M	-2	C	6	2	11						N	N	N	N	N	N			N
8105		NAD	9	M	-2	C	6	2	9						N	N	N	N	N	N			N
5503		RSPTB	28	F	2	C	1	3	25	9	5	2	0	0	Y	N	Y	N	Y	Y	19	2	N
1501		NAD	31	M	-1	C	1	3	18	15	4				N	N	N	N	N	Y	17	3	N
1502		OP	33	F	-2	C	1	3	18	5		3	0	0	N	N	N	N	N	Y	21	1	N
1503		NAD	4	M	0	C	6	0	4						N	N	N	N	N				N
8306		NAD	13	F	0	C	6	2	13						N	Y	N	N	N	N			N
8302			32	F		C	6	3	30	10	2	1	0	0	N	N	N	N	N	N			N
7002		NAD	38	F		C	1	3	24	3	5	5	0	0	N	N	N	N	N	N			N
6303		NAD	14	M	-1	C	6	2	9						N	N	N	N	N	N			N
7602		RSPTB	42	F	1	C	1	3	14	16	5	8	1	0	N	N	N	N	N	Y	15	2	Y
7601		NAD	47	M	-2	C	1	3	14	14		3N			N	N	N	N	N	Y	18	2	N
6403		NAD	38	M	-1	B	1	3	4	19	4				N	N	N	N	N	Y	16	2	N
5102		RSPTB	42	M	-1	B	1	3	10	15		3N			N	N	N	N	N	Y	20	2	N
9106		OP	50	M	-1	B	1	3	13	13	5				N	N	N	N	Y	Y	20	2	Y
301		NAD	37	F	1	C	1	3	10	18	2	1	0	0	N	N	N	N	N	N			N
303		NAD	18	M	-2	C	1	3	10	4	4				N	N	N	N	N	Y	16	1	N
7306		NAD	18	M		C	6	3	18	3	5				N	N	N	N	N	Y	15	2	N
6101		NAD	35	F	2	B	1	3	5	16		3M			N	N	N	N	N	N			N
7501		NAD	35	F	2	C	6	3	27	25	5	1	0	0	Y	N	N	N	N	N			N
7502			65	M	-2	C	4	5	27						Y	N	N	N	N	Y	18	3	N
4603		NAD	9	F	-1	B	6	2	9						N	N	N	N	N	N			N
6905		NAD	13	M	-2	C	6	2	13						N	N	N	N	N	Y	11	1	Y
402		NAD	32	F	-1	C	3	3	6	17	2	1	0	0	N	N	N	N	Y	N			N
7103		NAD	13	F	-2	C	6	2	13						N	N	N	N	N	Y	13	1	N
6902		NAD	37	F	2	C	1	3	25	12	5	5	1	0	Y	Y	N	N	N	Y	14	1	N
9102		NAD	38	M	1	B	1	3	10	20	4				N	N	N	N	N	Y	20	2	Y
9701		NAD	47	F	1	C	6	3	3	12	5	0	0	0	N	N	N	N	N	Y	16	1	N
7103		NAD	24	F	-1	C	6	3	24	4	5	1	0	0	N	N	N	N	N	Y	14	1	N
4601		NAD	40	F	2	B	1	3	7	7	5	4	0	1	N	N	N	N	N	Y	17	1	N
1201		NAD	49	M	1	B	1	3	30	30	4				N	N	N	N	N	N			N
6008			7	F		C	6	2	7						N	N	N	N	N	N			N
6003		RAPTB	20	F		C	6	3	20	6	5	0	0		N	N	N	N	N	Y	17	1	N
6604		NAD	18	F		C	6	4	18			1	0	0	N	N	N	N	N	Y	16	1	N
2505		RSPTB	42	F		C	1	3	19	9	5	5	1	0	N	N	Y	N	N	Y	15	1	Y
7003		NAD	18	F	-2	C	6	4	18			1	0	0	N	N	N	N	N	Y	16	1	N
6302		RSPTB	38	M	-2	C	1	3	16	16	4				N	N	Y	N	N	N			N
5904		NAD	11	F	-2	C	6	2	11						N	N	N	N	N	N			N
7608		NAD	13	F		C	6	2	10						N	N	N	N	N	Y	12	2	N
3105		NAD	9	M	1	C	6	2	9						N	N	N	N	N	N			N

IDNUM	OLD	TIRED	NIGHT SWEAT	WT LOSS	APPE TITE	BCG	BCG CONFM	SPORT /WEEK	SPORT YEARS	TB KNWLD SCORE	TOT VOL OF ABSALC INTAKE IN 2 WEEKS	ML TYPE DWELL	TYPE TOILET	CROWD INDEX	PFSL	TOT % FWAREA	SIGHN SYMPT	RPT XRAY	SPT	CULT	DISEA CATO	NOTIFI YEAR
6301		N	N	N	N	2		N		2	0	1	2	3	1.2	9.9						
1701	N	Y	N	Y	N	2		N		2	70	1	1	3	0.9	10.3						
3703		N	N	N	N	3		Y	3	2	40	1	1	1	1.5	3.3						
403		Y	N	N	N	1	1	N		2	60	1	1	1	2.1	3.4						
5305		N	N	N	N	1	1	N		2	0	1	1	1	1.0	4.3						
503		N	N	N	N	1	1	Y	2	2	35	1	1	4	2.7	4.5						
6005		N	N	Y	N	1	1	Y	3	2	35	1	2	4	0.9	5.7						
7105		N	N	N	N	1	1	Y	3	5	20	1	2	4	0.6	8.3						
4905		N	N	N	N	1	1	Y	3	2	35	1	1	4	0.7	9.9						
4605		N	N	N	N	1	1	Y	2	2	70	1	1	3	0.8	9.9						
5903		N	N	N	N	1	1	Y	2	2	35	1	2	4	0.6	5.7						
6305		N	N	N	Y	1	2	N			0	1	2	3	1.2	9.9						
6904		N	N	N	N	1	1	Y	4	3	20	1	2	4	0.9	5.7						
2503		N	N	N	N	1	1	Y	4	3	0	1	1	4	0.8	10.1						
8102		Y	N	N	Y	1	1	N		2	113	1	1	4	0.9	5.1						
8104		N	N	N	N	1	1	N			0	1	1	4	0.9	5.1						
8105	N	N	N	N	N	1	1	Y	1		0	1	1	4	0.9	5.1						
5503		N	N	N	N	1	1	N		2	0	1	1	4	0.3	8.1		POS	++	+	KCPTB	1984
1501		N	N	N	N	3		N		2	150	1	1	4	1.0	12.3						
1502		N	N	N	N	1	1	N		2	18	1	1	4	1.0	12.3	0/5	OP	-	-		
1503		N	N	N	N	1	2					1	1	4	1.0	12.3						
8306		N	N	N	N	1	1	N		2	0	1	1	1	1.0	4.3						
8302		N	N	N	N	1	1	N		2	18	1	1	1	1.0	4.3						
7002		N	N	Y	N	2		N		3	53	1	2	3	1.1	5.7						
6303		N	N	N	N	1	1	Y	2	4	0	1	2	3	1.2	9.9						
7602	N	N	N	N	N	3		N		5	10	1	2	4	1.2	5.8	1/5	NEG	-	-		
7601		Y	Y	Y	Y	3		N		2	53	1	2	4	1.2	5.8						
6403	N	N	N	Y	N	1	1	Y	2	2	300	1	2	1	2.2	9.9						
5102		Y	N	N	Y	3		N		4	338	2	3	3	1.4	4.2	2/5	NEG	-	-		
9106	N	N	Y	N	Y	3		N		3	145	1	1	4	0.5	3.3	3/5	OP	-	-		
301		N	N	N	N	3		Y	2	2	20	1	1	1	1.7	3.4						
303		N	N	N	N	1	1	Y	4	5	75	1	1	1	1.7	3.4						
7306		N	Y	Y	N	1	1	Y	2	3	900	1	2	4	3.0	5.8						
6101		N	Y	Y	N	3		Y	4	2	0	1	2	4	0.9	9.9						
7501		N	N	N	N	3		N		2	0	1	2	2	1.0	5.8						
7502		N	N	N	N	3		N		5	40	1	2	2	1.0	5.8						
4603		N	N	N	N	1	1	N			0	1	1	3	0.8	9.9						
6905		N	N	N	N	1	1	Y	2	5	0	1	2	4	0.9	5.7						
402		Y	N	N	N	1	1	Y	3	3	0	1	1	1	2.1	3.4						
7108		N	N	N	N	1	1	Y	2	4	0	1	2	4	0.6	8.3						
6902		N	N	Y	N	1	1	N		5	0	1	2	4	0.9	5.7						
9102	N	N	Y	N	N	3		N		4	317	1	1	4	0.5	3.3						
9701		N	N	N	N	1	1	N		2	0	3	3	1	2.4	24						
7103		N	N	N	N	1	1	Y	1	2	150	1	2	4	0.6	8.3						
4601		N	N	Y	Y	1	1	N		5	0	1	1	3	0.8	9.9						
1201		N	N	N	N	3		N		1	0	1	1	4	0.7	12.7						
6008		N	N	N	N	1	2	N			0	1	2	4	1.3	5.7						
6003		N	Y	N	Y	1	1	N		2	0	1	2	4	1.3	5.7						
6604		N	N	N	N	1	1	N		4	75	1	2	4	0.9	5.7						
2505	N	Y	Y	Y	Y	1	1	N		2	0	1	1	4	0.8	0.9		POS	+	+	KCPTB	1973
7003		N	N	N	N	1	1	N		4	53	1	2	3	1.1	5.7						
6302		N	N	N	N	3		N		2	75	1	2	3	1.2	9.9		POS	+++	+	KCPTB	1986*
5904		N	N	N	N	1	1	N			0	1	2	4	0.6	5.8						
7605		N	N	Y	Y	2		N		4	35	1	2	4	1.2	5.8						
3105		N	N	N	N	1	1	N			0	1	1	4	1.4	12.2						

EPIDEMIOLOGY OF TB I ACASSAR CAMP

IDNUM	PREG	MINI XRAY RSLT	AGE	SEX	NUTRI STAT MED	RACE	M/STAT	OCCUP STAT	CAMP YEARS	YRS @ COMPY	WORK CAT	NO.OF LIVE BIRTH	NO.OF MISCAR	NO. OF INFANT DEATH	HBP	ASMT ALLG	TB	DIAB	ABDOM OP	SMOKE	AGE @ TIME OF SMOKING	NO.OF CIGAR. DAILY	COUGH
505			22	F		C	6	3	12	1		0	0		N	N	N	N	N	N			N
5501		NAD	53	M	-2	C	1	3	25	25		3M			N	N	N	N	N	Y	15	2	N
7501		NAD	62	M	-2	C	1	3	34	34	4				N	N	N	N	N	Y	26	2	N
5504		RSPTB	22	F	-1	C	6	3	22	3	5	0	0		N	Y	Y	N	N	Y	17	1	N
6603	PREG		21	F	-2	C	6	3	17	2	5	1	0	0	N	N	N	N	N	Y	14	1	N
2201		NAD	52	M	-1	B	1	3	25	25	4				N	N	N	N	N	Y	20	4	N
6501		NAD	29	F	-1	C	1	3	3	12	2	2	0	0	N	N	N	N	N	N			N
4604			19	F		C	6	2	7			0	0		N	N	N	N	N	N			N
5403		NAD	25	M	-2	B	6	2	2						N	N	N	N	N	N			N
5906			2	F		C	6	0	2						N	N	N	N	N				N
5307		RSPTB	45	F	-2	C	4	4	3			4	0	2	N	Y	N	N	Y	Y	16	1	Y
5602		NAD	22	M	-2	C	1	3	18	4	4				N	Y	N	N	N	Y	18	1	N
5501		NAD	57	F	-1	C	4	1	15			8	1	2	N	N	N	N	N	Y	19	3	Y
5502		RSPTB	36	F	-2	C	6	3	26	18	5	2	0	0	N	N	N	N	N	Y	19	1	N
1402		NAD	33	F		I	1	1	5			3	0	0	Y	N	N	N	N	N			N
7709		NAD	18	M	-2	C	6	2	18						N	N	N	N	N	N			N
7513			0.	F		C	6	0	0.8						N	N	N	N	N				N
7812		NAD	5	F	-2	C	6	0	5						N	N	N	N	N				N
7806			19	F	-2	C	6	4	19			1	0	0	N	N	N	N	N	Y	12	2	N
8004			2	F	-2	C	6	0	2						N	Y	N	N	N				N
8003		NAD	5	M		C	6	0	1						N	N	N	N	N				Y
8202		NAD	39	F	-1	C	1	3	25	21	2	3	0	0	N	N	N	N	N	Y	20	1	N
1101		NAD	63	F	-2	C	1	3	20	16	5	7	0	0	Y	N	N	N	N	Y	17	1	N
7204		NAD	13	M	-1	C	6	2	6						N	N	N	N	N	N			N
7203		NAD	16	F	-1	C	6	2	7			0	0	0	N	N	N	N	N	N			N
7604		NAD	21	M	-2	C	6	3	21	3		3N			N	N	N	N	N	N			N
7201		NAD	31	F	1	C	1	3	5	1	5	3	0	0	N	N	N	N	N	Y	16	3	N
3104		NAD	17	M	0	C	6	4	17						N	N	N	N	N	Y	16	2	N
7104		RSPTB	19	M	-2	C	6	4	19						N	N	N	N	N	Y	15	1	N
5104		RSPTB	60	M	-1	B	1	3	12	12	4				N	N	N	N	N	Y	8	1	Y
4904		SP	23	F	2	C	1	3	23	0.3	5				Y	N	N	N	N	Y	20	3	N
3402		SP	40	F	2	B	1	3	6	2	5	9	0	2	N	Y	N	N	Y	Y	17	3	Y
1301		NAD	42	M	2	B	1	3	3	3		3N			N	N	N	N	N	N			N
5106		OP	37	M	-2	B	3	3	2	2		3M			N	N	N	N	N	N			N
1102		NAD	36	F	-1	C	1	3	3	0.3	5	1	0	0	N	N	N	N	N	Y	17	1	N
5901			32	F	-2	C	1	3	10	1	5				N	N	N	N	N	Y	17	1	N
1705		NAD	24	F	-2	C	6	3	6	3	5	1	1	0	N	N	N	N	N	Y	17	1	N
7707		NAD	26	F	2	C	6	3	26	0.3	5	1	0	0	N	N	N	N	N	N			N
6605		NAD	18	F	-1	C	6	3	15	0.5	5	1	0	0	N	N	N	N	N	Y	16	1	Y
1203		NAD	20	F	1	C	6	3	14	2	5	0	0		N	N	N	N	N	N			N
6009			26	F	-1	C	6	3	14	0.3	5	3	2	0	N	N	N	N	N	Y	14	2	N
202		NAD	30	F	1	C	1	3	5	9		0	0		N	N	N	N	N	Y	15	1	N
6802		NAD	27	M	-1	C	1	4	2						N	N	N	N	N	Y	14	2	N
1401		NAD	35	M	1	I	1	3	9	9	1				N	N	N	N	N	Y	8	2	N
6107		NAD	28	F	1	B	5	3	1	0.5	5	1	0	0	N	N	N	N	N	N			N
6202	PREG		28	F	-1	C	1	3	6	3		2	1	0	N	N	N	N	N	N			N
5301			37	M		B	3	3	14	14		3N			N	N	N	N	N	N			N
5202		NAD	55	M	-2	B	1	3	11	11		3N			N	N	N	N	N	N			N
5201		NAD	40	M	-1	B	3	3	2	2		3N			N	N	N	N	N	Y	13	3	N
1409		NAD	23	M		C	5	3	3	3	4				N	N	N	N	N	Y	14	3	N
5401		RSPTB	32	M	-1	B	1	3	5	5		3N			N	N	N	N	N	Y	17	2	N
8303		NAD	31	F	2	C	6	3	31	2		0	0		N	N	N	N	N	N			N
5402			33	M		B	6	3	10	16		3N			N	N	N	N	N	Y	25	2	N
101		NAD	72	M	2	W	4	5	6	3		3N			Y	N	N	N	N	Y	12	1	N
9103		RSPTB	17	F	2	B	6	4	0.5			0	0		N	N	N	N	N	N			N

IDNUM	BLD	TIRED	NIGHT SWEAT	WT LOSS	APPE TITE	BCG	BCG CONFM	SPORT /WEEK	SPORT YEARS	TB KNWLD SCORE	TOT VOL OF ABSALC INTAKE ML IN 2 WEEKS	TYPE DWELL	TYPE TOILET	CROWD INDEX	PFSL	TOT % FWAREA	SIGHN SYMPT	RPT XRAY	SPT	CULT	DISEA CATO	NOTIFI YEAR
506		N	N	N	N	1	1	N		4	0	1	1	4	2.7	4.5						
5501		N	N	N	N	2		N		2	188	1	1	1	2.7	11						
7401		N	Y	N	N	1		N		5	250	1	2	4	0.3	5.8						
5301		N	N	Y	N	1	1	N		4	50	1	1	1	2.7	11		POS	+++	+	KCPTB	1986
6603		N	N	N	N	1	1	N		1	188	1	2	4	0.9	6.7						
2201		N	Y	N	N	2		N		2	1056	1	1	1	3.0	10.1						
6501		N	N	N	N	3		N		2	50	1	2	1	2.0	6.3						
4604		N	N	N	N	1	1	N		4	0	1	1	3	0.8	9.9						
5403		N	N	Y	Y	3		N		2	0	2	3	3	1.3	3.1	0/5	NEG	-	-		
5906		N	N	N	N	1	2					1	2	4	0.6	5.7						
5307	N	N	N	N	N	1	2	N		3	675	1	1	1	1.0	4.3	1/5	NEG	-	-		
5602		N	N	N	N	1	1	N		4	560	1	1	4	0.6	8.1						
5501	N	Y	N	N	Y	3		N		5	1050	1	1	4	0.3	8.1						
5502		Y	N	N	Y	2		N		4	0	1	1	4	0.3	8.1	0/5	POS	++	+	PTB	1988
1402		N	N	N	N	2		N		4	0	1	2	1	0.8	25.6						
7709		N	N	N	N	1	1	Y	2	4	0	1	2	4	0.8	5.8						
7813		N	N	N	N	1	1					1	2	4	0.3	5.8						
7812		N	N	N	N	1	1					1	2	4	0.3	5.7						
7806		N	N	N	N	1	1	N		4	110	1	2	4	0.3	5.8						
8004		N	N	N	N	1	2					1	2	1	2.3	5.8						
8003	N	N	Y	N	N	1	2					1	2	1	2.3	5.8						
5202		Y	N	N	Y	1	1	N		2	25	1	1	1	1.9	5.1						
1101		N	N	N	N	2		N		4	0	1	1	3	1.3	12.3						
7204		N	N	N	N	1	1	Y	1	4	0	1	2	2	1.6	8.3						
7203		N	N	N	Y	1	1	N		2	53	1	2	2	1.6	8.3						
7604		Y	Y	Y	N	1	1	Y	1	3	525	1	2	4	1.2	5.8						
7201		N	N	N	N	1	1	N		5	0	1	2	1	1.6	8.3						
3104		N	N	N	N	1	1	N		4	75	1	1	4	1.4	12.2						
7104	N	N	N	Y	N	1	1	N		4	75	1	2	4	0.6	8.3	2/5	NEG	-	-		
5104		N	N	N	N	3		N		3	38	2	3	3	1.4	4.2	0/5	NEG	-	-		
4904	Y	Y	Y	Y	N	1	1	N		5	0	1	1	4	0.7	9.9	3/5	NEG	-	-		
3402		Y	N	Y	N	3		N		2	0	1	1	4	0.5	9.9	2/5	NEG	-	-		
1301		N	N	N	N	2		Y	4	5	0	1	1	1	2.3	4.3						
5106		N	N	N	N	3		Y	4	2	0	2	3	3	1.4	4.2	0/5	OP	-	-		
1102		N	N	N	N	1	1	N		2	0	1	1	3	1.3	12.3						
5901		N	Y	N	N	2		Y	4	5	35	1	2	4	0.6	5.7						
1705		N	N	N	N	1	1	N		5	150	1	1	3	0.9	10.3						
7707		N	N	N	N	1	1	Y	4	4	0	1	2	4	0.8	5.8						
6605		Y	N	N	N	1	1	N		4	0	1	2	4	0.9	5.7						
1203		N	N	N	N	1	1	N		4	0	1	1	4	0.7	12.7						
6009		N	N	N	N	1	1	N		4	150	1	2	4	1.3	5.7						
202		N	N	N	N	1	1	N		2	0	1	1	1	3.1	3.4						
6602		N	N	N	N	3		N		2	450	1	2	1	2.0	6.3						
1401		N	N	N	N	3		N		3	0	1	2	1	0.8	25.6						
6107		N	N	Y	N	1	1	N		1	38	1	2	4	0.9	9.9						
6202		N	N	N	N	1	1	N		2	0	1	2	4	2.0	9.9						
5301		N	N	N	N	3		N		2	0	2	3	3	1.3	3.1						
520_		N	N	N	N	2		N		2	0	2	3	4	2.1	2.9						
5201		N	N	N	N	2		N		2	18	2	3	4	2.1	2.9						
1409		N	N	Y	N	3		Y	2	4	375	1	2	1	0.8	25.6						
5401		N	N	N	N	1	1	Y	2	4	225	2	3	3	1.3	3.1						
5303		Y	N	N	N	1	1	N		2	0	1	1	1	1.0	4.3						
5402		N	N	N	N	1	1	N		4	0	2	3	3	1.3	3.1						
101		Y	N	N	N	3		N		3	677	1	1	1	5.4	1.3						
9103		Y	Y	N	Y	2		N		5	0	1	1	4	0.5	3.3	3/5	NEG	-	-		

IDNUM	PREG	MINI XRAY RSLT	AGE	SEX	NUTRI STAT MED	RACE	M. STAT	OCCUP STAT	CAMP YEARS	YRS @ COMPY	WORK CAT	NO.OF LIVE BIRTH	NO.OF MISCAR	NO. OF INFANT DEATH	HBP	ASMT ALLG	TB	DIAB	ABDOM OP	SMOKE	AGE @ TIME OF SMOKING	NO.OF CIGAR. DAILY	COUGH
8001		NAD	27	M	-1	C	1	3	1	1		3N			N	N	N	N	N	Y	12	2	N
7904			30	F	-2	C	6	3	10	3	5	1	0	0	N	N	N	N	N				Y
5005		NAD	27	F	-2	C	6	3	0.5	0.3	5	1	0	0	N	N	N	N	N	Y	18	1	N
4911		NAD	17	M	-2	C	6	2	17						N	N	N	N	N	Y	13	1	N
8301		NAD	63	F	1	C	4	1	29			12	0	5	Y	Y	N	N	Y	Y	28	3	Y
9101		RAPTB	30	F	-2	B	1	1	2			6	1	0	N	N	N	N	N	N			N
2203		NAD	19	F	-1	B	6	2	5			0	0		N	N	N	N	N	N			N
1405			2	M	0	I	6	0	2						N	N	N	N	N				N
2506		NAD	11	M	-1	C	6	2	11						N	N	N	N	N	N			N
2502		SP	37	F	2	C	1	3	15	10	5	4	1	1	N	Y	N	N	N	N			N
2202		NAD	46	F	2	B	1	3	26	20	5	0	0		N	N	N	Y	N	N			Y
3002		NAD	29	F	-1	C	1	3	1	14	2	2	0	0	N	Y	N	N	N	N			N
6901		NAD	44	M	-2	C	1	3	24	24	5				Y	N	N	N	N	Y	15	1	Y
2507		NAD	8	F	-2	C	6	2	6						N	N	N	N	N	N			N
2504		NAD	17	F	2	C	6	2	17			0	0		N	N	N	N	N	N			N
7403		NAD	21	M	-2	C	6	3	21	0.3		3M			N	N	N	N	N	Y	7	3	N
7101		NAD	58	M	1	C	1	3	28	28		3N			N	Y	N	N	N	Y	30	1	Y
4902			58	F		C	1	1	28			4	0	0	Y	N	N	N	N	Y	12	1	Y
6803			2	F		C	6	0	2						N	N	N	N	N				N
8403		NAD	16	M	0	C	6	2	5						N	N	N	N	N	Y	14	2	N
6205		NAD	50	F	-2	C	6	3	15	14	5	7	0	3	Y	N	N	N	N	Y	16	2	N
501		NAD	53	M	1	C	1	3	25	24		3N			N	N	N	N	N	Y	18	2	N
1509		NAD	42	F	1	C	4	3	0.5	0.3	5	5	0	0	N	N	N	N	N	Y	16	3	N
1403		NAD	24	M	1	C	6	3	8	0.5	4				N	N	N	N	N	Y	18	1	N
6601		SP	44	F	-2	C	1	1	10			8	2	1	N	N	N	N	N	N			Y
4002			28	F		C	1	3	3	1	5	2	0	0	N	N	N	N	N	Y	20	1	N
1702		RAPTB	58	M	-2	C	1	4	4						Y	N	Y	N	N	Y	25	1	N
7205		NAD	3	M	1	C	6	0	3						N	N	N	N	N				N
6002			39	M		C	1	3	14	4	4				N	N	N	N	N	Y	22	2	N
8304			22	F		C	6	3	22	3	5	0	0		N	N	N	N	N	N			N
8203		NAD	17	F	-1	C	6	2	17			0	0		N	N	N	N	N	Y	15	1	N
5507		NAD	19	M		C	6	4	18						N	N	N	N	N	Y	7	1	N
8801			27	M		C	1	3	4	2		3N			Y	N	N	N	N	Y	22	1	N
8505		NAD	20	M	-1	C	6	3	20	2	4				N	N	N	N	N	Y	13	2	N
5505		RSPTB	17	F	-2	C	6	3	17	0.3	5	0	0		N	N	N	N	N	Y	10	1	N
507		NAD	20	M	-2	C	6	2	20						N	N	N	N	N	Y	14	1	N
302		NAD	38	M	-1	C	1	3	6	6	4				N	N	N	N	N	Y	20	1	N
7503		NAD	20	M	-2	C	6	4	20						N	N	N	N	Y	Y	14	2	N
7309		NAD	23	M		C	6	3	18	0.5	1				N	N	N	N	N	Y	11	1	N
7603		NAD	23	F		C	6	4	15			2	0	0	N	N	N	N	N	Y	20	3	N
8101		NAD	37	F	2	C	1	3	6	0.5	2	4	0	0	N	N	N	N	N	N			Y
8503		NAD	23	M	-2	C	6	3	23	2	4				N	N	N	N	N	Y	12	4	N
6007		NAD	9	M	-2	C	6	2	5						N	N	N	N	N	N			Y
7404		NAD	16	M	-2	C	6	2	16						N	Y	N	N	N	Y	14	1	N
1605			19	F		C	6	3	12	2	5	0	0		N	N	N	N	N	Y	17	1	Y
7303		RSPTB	23	M	-1	C	6	3	23	2	5				N	N	Y	N	N	Y	9	1	N
7402		NAD	42	F	-2	C	1	3	25	25	2	4	0	1	N	N	N	Y	Y	Y	20	2	Y
7307		NAD	13	M	-1	C	6	2	13						N	N	N	N	N				N
7302		NAD	46	F	-1	C	1	1	25			5	0	0	N	N	N	N	Y	N			N
5101		RSPTB	63	M	2	B	1	3	10	20	4				N	N	N	N	N	N			N
3702		RSPTB	50	M	-2	C	1	3	19	2	4				Y	N	Y	N	N	Y	16	3	N
6501		NAD	53	F	-1	C	1	3	25	0.5	5	2	0	0	N	N	N	N	N	Y	16	1	N
6602		NAD	48	M	-1	C	1	3	19	4	5				N	N	N	N	N	Y	20	1	N
6201		NAD	31	M	-1	C	1	3	25	15	4				N	N	N	N	N	Y	15	2	N
8201		NAD	38	M	-1	C	1	3	30	26	4				N	N	N	N	N	Y	16	3	N

IDNUM	BLD	TIRED	NIGHT SWEAT	WT LOSS	APPE TITE	BCG	BCG CONFM	SPORT /WEEK	SPORT YEARS	TB KNWLD SCORE	TOT VOL OF ABSALC INTAKE ML IN 2 WEEKS	TYPE DWELL	TYPE TOILET	CROWD INDEX	PFSL	TOT % FWAREA	SIGHN SYMPT	RPT XRAY	SPT	CULT	DISEA CATO	NOTIFI YEAR
8001	N	N	N	N	N	1	1	N		5	150	1	2	1	2.3	5.8						
7804	N	N	N	N	N	1	1	N		2	0	1	2	1	1.0	5.8						
5005		N	N	Y	N	3		N		4	70	1	2	1	2.3	5.8						
4911		N	N	N	N	3		Y	2	2	0	1	1	4	0.7	9.9						
5301	N	Y	Y	N	N	2		N		5	0	1	1	1	1.0	4.3						
9101		N	N	Y	Y	2		N		1	0	1	1	4	0.5	3.3	2/5	POS	-	+	PTB	1988
2203		N	N	N	N	1	1	Y	4	2	0	1	1	2	3.0	10.1						
1405		N	N	N	N	2						1	2	1	0.8	25.6						
2506		N	N	N	N	1	1	N			0	1	1	4	0.8	10.1						
2502	N	N	N	N	N	1	1	N		3	0	1	1	4	0.8	10.1	1/5	NEG	-	-		
2202		N	N	N	N	3		N		2	0	1	1	1	3.0	10.1						
8002	N	N	N	N	N	2		N		2	0	1	2	1	2.3	5.8						
6901	N	Y	N	Y	N	3		N		2	0	1	2	4	0.9	5.7						
2507		N	N	N	N	1	1	Y	2		0	1	1	4	0.8	10.1						
2504		N	N	N	N	1	1	Y	4	1	18	1	1	4	0.8	10.1						
7403		N	N	N	N	2		Y	4	2	130	1	2	2	2.1	10						
7101	N	N	Y	Y	N	2		N		2	75	1	2	4	0.6	6.3						
4902	N	N	N	Y	N	2		N		5	0	1	1	4	0.7	9.9						
6803		N	N	N	N	1	1					1	2	1	2.0	6.3						
8403		N	N	Y	N	1	1	Y	4	2	135	1	1	3	1.9	9						
6205		N	N	N	N	2		N		2	0	1	2	4	2.0	9.9						
501		N	N	N	N	1	1	N		2	113	1	1	4	2.7	4.5						
1509		N	Y	N	N	3		N		1	225	1	1	4	1.0	12.3						
1408		N	N	N	N	1	1	N		1	105	1	2	1	0.8	25.6						
6601	N	Y	N	Y	Y	2		N		4	38	1	2	4	0.9	5.7	3/5	NEG	-	-		
4002		N	N	N	N	1	1	N		4	38	1	1	2	1.0	9.9						
1702		N	Y	N	Y	3		N		3	450	1	1	3	0.9	10.3	2/5	POS	+	+	PTB	1988*
7205		N	N	N	N	1	2					1	2	2	1.6	8.3						
6002		N	N	N	N	2	2	N		4	375	1	2	4	1.3	5.7						
8304		N	N	N	N	2		N		2	54	1	1	1	1.0	4.3						
8203		N	N	Y	Y	1	1	N		2	113	1	1	1	1.9	5.1						
5507		N	N	N	N	1	1	Y	3	2	653	1	1	4	0.3	9.1						
8801		N	N	N	N	1	1	N		5	38	1	1	2	1.6	3.3						
8505		N	N	N	N	1	1	Y	4	4	113	1	1	1	2.7	11						
5505		N	N	N	N	1	1	N		2	0	1	1	4	0.3	9.1	0/5	NEG	-	-		
507		N	N	N	N	1	1	Y	2	2	20	1	1	4	2.7	4.5						
302		N	N	Y	N	1	1	N		3	786	1	1	1	1.7	3.4						
7503		N	N	N	N	2		Y	1	4	1170	1	2	2	1.0	5.8						
7308		N	N	N	N	1	1	Y		4	324	1	2	4	3.0	5.8						
7603		N	N	Y	Y	1	1	N		2	150	1	2	4	1.2	5.8						
8101	N	N	N	N	N	1	1	N		2	0	1	1	4	0.9	5.1						
8503		Y	Y	Y	Y	1	1	N		1	450	1	1	1	2.7	11						
6007	N	Y	Y	N	Y	3		Y	1		0	1	2	4	1.3	5.7						
7404		N	Y	N	N	1	1	Y	3	2	100	1	2	2	2.1	10						
1605	N	N	N	Y	N	1	1	N		2	60	1	1	4	1.9	10.3						
7303		N	N	N	N	1	1	Y	4	4	100	1	2	4	3.0	5.8		POS	++	+	KCPTB	1985
7402	N	N	N	N	N	1	1	N		2	50	1	2	2	2.1	10						
7307		N	N	N	N	2		Y	4	2	0	1	2	4	3.0	5.8						
7302		N	N	N	N	1	1	N		2	35	1	2	4	3.0	5.8						
5101		N	N	N	N	3		N		5	113	2	3	3	1.4	4.2	0/5	POS	++	+	PTB	1988
3702		N	N	N	N	2		N		3	250	1	1	1	1.5	3.3		POS	+++	+	KCPTB	1986
6501		Y	N	Y	N	2		N		2		1	2	3	1.5	5.7						
6602		Y	N	N	N	1	1	N		4	0	1	2	4	0.9	5.7						
6201		N	N	N	N	1	1	Y	1	2	263	1	2	4	2.0	9.9						
8201		N	N	N	N	1	1	N		2	105	1	1	1	1.8	5.1						

EPIDEMIOLOGY OF TB IN MASSAR CAMP

IDNUM	PREG	MINI XRAY RSLT	AGE	SEX	NUTRI STAT MED	RACE	M/STAT	OCCUP STAT	CAMP YEARS	YRS @ COMPY	WORK CAT	NO.OF LIVE BIRTH	NO.OF MISCAR	NO OF INFANT DEATH	HBP	ASMT ALLG	TB	DIAB	ABDOM OP	SMOKE	AGE @ TIME OF SMOKING	NO.OF CIGAR. DAILY	COUGH
7606		NAD	17	F		C	6	4	10			0	0		N	N	N	N	N	Y	12	1	Y
4909		NAD	12	M	0	C	6	2	12						N	N	N	N	N	Y	12	1	N
4907		NAD	36	F	1	C	2	3	30	7	5	5	0	0	N	N	N	N	N	N			N
4910		NAD	8	M	-1	C	6	2	8						N	N	N	N	N	N			N
4306		NAD	11	M	-1	C	6	2	4						N	N	N	N	N	N			N
5905		NAD	7	F	2	C	6	2	7						N	N	N	N	N	N			N
1601		NAD	35	M	-1	C	5	3	15	15		3N			N	N	N	N	N	Y	15	2	N
7301		NAD	51	M	-2	C	6	3	30	30		3N			N	N	N	N	N	Y	16	2	N
5302		NAD	46	M		B	1	3	22	22		3N			N	N	N	N	N	N			N
1604		NAD	13	M	-2	C	6	2	13						N	N	N	N	N	N			N
4903		NAD	12	F	-1	C	6	2	12						N	N	N	N	N	N			N
4906		NAD	4	F	1	C	6	0	4						N	N	N	N	N				N
5604			20	F		C	6	4	20			2	1	0	N	N	N	N	N	Y	16	1	N
5601			58	M		C	4	3	29	4		3N			N	N	N	N	N	N			N
1504		NAD	17	F	0	C	6	4	14			1	0	0	Y	Y	N	N	N	N			N
1507			2	M	0	C	6	0	3						N	N	N	N	N				N
7810		NAD	9	F		C	6	2	5						N	N	N	N	N	N			N
1510		NAD	25	M		C	1	3	2	2	5				N	N	N	N	N	Y	15	2	N
7905		NAD	8	M		C	6	2	8						N	N	N	N	N	N			N
7901	PREG		28	F	1	C	1	1	5			4	0	0	N	N	N	N	N	N			N
7903			4	M		C	6	0	4						N	N	N	N	N				N
3308		RAPTB	51	M		C	5	4	20						N	N	Y	N	N	Y	14	2	N
5206		NAD	57	M	1	B	1	3	5	5		3N			N	Y	N	N	N	Y	16	3	Y
1205		NAD	7	F	-2	C	6	2	7						N	N	N	N	N	N			N
4901		SP	57	M	2	C	1	3	32	32	4				Y	N	N	N	N	Y	20	2	N
5509		NAD	4	F		C	6	0	4						N	N	N	N	N				N
7309		NAD	10	F		C	6	2	6						N	Y	N	N	N	N			N
6001		NAD	45	F	-1	C	2	3	26	6	5	4	4	0	N	N	N	N	Y	Y	16	2	N
7102			60	F		C	1	1	19			11	0	1	N	N	N	N	Y	Y	17	1	N
6004		NAD	14	M	-2	C	6	2	10						N	N	N	N	N	Y	12	1	N
6504		NAD	9	M	-2	C	6	2	4						N	N	N	N	N	N			N
6505		NAD	11	M	-1	C	6	2	4						N	N	N	N	N	N			N
1407		NAD	19	M	-2	C	5	3	19	2		3N			N	N	N	N	N	Y	16	1	N
7902		NAD	30	M		C	6	3	25	8		3M			N	N	N	N	N	Y	16	3	N
6903		NAD	15	F		C	6	2	15			0	0		N	N	N	N	N	N			N
9403		NAD	15	M	-2	C	6	2	2						N	N	N	N	N	Y	14	1	Y
9405		NAD	9	M	1	C	6	2	2						N	N	N	N	N	N			N
7802		NAD	27	F		C	6	3	27	7	5	5	1	0	N	N	N	N	Y	Y	16	2	N
5005			***	F		C	6	0	0.6						N	N	N	N	N				N
508		NAD	3	F	-1	C	6	0	3						N	N	N	N	N		-		N
7701		NAD	33	M	-1	C	6	3	16	18	4				N	N	N	N	N	Y	14	2	N
7803			34	M		C	6	3	4	2	5				N	N	N	N	N	Y	20	2	N
505		NAD	18	F	-2	C	6	4	15			1	0	0	N	N	N	N	N	Y	13	1	N
5205		NAD	5	M	-2	B	6	0	2						N	N	N	N	N				N
5204		NAD	6	F	1	B	6	0	2						N	N	N	N	N	N			N
4003		NAD	14	F		C	6	2	4						N	N	N	N	N	N			N
9105		NAD	6	F		B	6	0	1						N	N	N	N	N	N			N
6606		NAD	7	M		C	6	2	3						N	N	N	N	N	N			N
3404		NAD	16	M		B	6	2	6						N	N	N	N	N	N			N
1406		NAD	12	F	0	C	6	2	2						N	N	N	N	N	N			N
3407		NAD	6	M	1	B	6	2	5						N	N	N	N	N	N			N
203		NAD	6	M	-1	C	6	2	6						N	N	N	N	N	N			N
2501		NAD	43	M	-2	C	1	3	18	18	4				N	N	N	N	N	Y	18	2	N
3103		HSPTB	33	M	-1	C	1	3	12	5	4				N	N	Y	N	N	Y	10	2	Y

EPIDEMIOLOGY OF TB IN MACASSAR CAMP

IDNUM	BLD	TIRED	NIGHT SWEAT	WT LOSS	APPE TITE	BCG	BCG CONFM	SPORT /WEEK	SPORT YEARS	TB KNWLD SCORE	TOT VOL OF ABSALC INTAKE ML IN 2 WEEKS	TYPE DWELL	TYPE TOILET	CROWD INDEX	PFSL	TOT % FWAREA	SIGHN SYMPT	RPT XRAY	SPT	CULT	DISEA CATO	NOTIFI YEAR
7606	N	N	N	Y	N	1	1	N		4	225	1	2	4	1.2	5.8						
4909		N	N	N	N	1	1	Y	1		0	1	1	4	0.7	9.9						
4907		N	N	N	N	2		N		5	0	1	1	4	0.7	9.9						
4910		N	N	N	N	1	2	N			0	1	1	4	0.7	9.9						
4306		N	Y	N	N	3		Y	2		0	1	1	3	1.3	9.9						
5905		N	N	N	N	1	2	N			0	1	2	4	0.6	5.7						
1601		N	N	N	N	1	1	N		2	323	1	1	4	1.9	10.3						
7301		N	N	N	N	1	1	N		2	300	1	2	4	3.0	5.8						
5302		N	N	N	N	1	1	N		2	0	2	3	3	1.3	3.1						
1604		N	N	N	N	1	1	Y	4	4	0	1	1	4	1.9	10.3						
4903		N	N	N	N	1	1	Y	2		0	1	1	4	0.7	9.9						
4906		N	N	N	N	1	2					1	1	4	0.7	9.9						
5604		Y	N	N	Y	1	1	N		2	75	1	1	4	0.6	8.1						
5601		Y	N	N	Y	3		N		5	536	1	1	4	0.6	8.1						
1504		N	N	N	N	1	1	N		2	38	1	1	4	1.0	12.3						
1507		N	Y	N	N	1	2					1	1	4	1.0	12.3						
7810		N	N	N	N	1	1	Y	2		0	1	2	4	0.3	5.8						
1510		N	Y	Y	Y	1	1	N		4	490	1	1	4	1.0	12.3						
7905		N	N	N	N	1	1	N			0	1	2	1	1.0	5.8						
7901		N	N	N	N	1	1	N		2	0	1	2	1	1.0	5.8						
7903		N	N	N	N	1	2	N				1	2	1	1.0	5.8						
8308		Y	Y	N	N	1	1	N		2	1050	1	1	1	1.0	4.3	2/5	POS	++	+	PTB	198
5206	N	N	Y	N	N	3		N		4	300	2	3	4	2.1	2.9						
1205		N	N	N	N	1	2	N			0	1	1	4	0.7	12.7						
4901		Y	Y	N	N	3		N		3	113	1	1	4	0.7	9.9	2/5	NEG	-	-		
8509		N	N	N	N	1	2					1	1	1	2.7	11						
7809		N	N	N	N	1	2	Y	3		0	1	2	4	0.3	5.8						
6001		N	N	N	N	3		N		2	113	1	2	4	1.3	5.7						
1505		N	N	N	Y	1	1	Y	4	4	75	1	1	4	1.0	12.3						
7102		Y	N	N	N	3		N		3	70	1	2	4	0.6	8.3						
6004		N	Y	N	N	1	1	N		4	18	1	2	4	1.3	5.7						
6504		N	N	N	N	1	1	Y	1		0	1	2	3	1.5	5.7						
6505		N	N	N	N	1	1	Y	2		0	1	2	3	1.5	5.7						
1407		N	N	N	N	1	1	Y	2	1	70	1	2	1	0.8	8.3						
7902		N	N	N	N	1	1	Y	4	2	150	1	2	1	1.0	5.8						
6903		N	N	N	N	1	1	N		4	0	1	2	4	0.9	5.7						
9403		N	N	N	Y	1	1	Y	4	2	70	1	1	4	0.6	3.3						
9405		N	N	Y	Y	1	1	Y	2		0	1	1	4	0.6	3.3						
7802		N	N	N	Y	1	1	N		2	0	1	2	4	0.3	5.8						
8005		N	N	N	N	1	2					1	2	1	2.3	5.8						
803		N	N	N	N	1	2					1	2	4	1.2	12.3						
7701		N	N	N	N	1	1	N		1	150	1	2	4	0.8	5.8						
7803		N	N	N	N	1	1	Y	2	2	0	1	1	4	0.3	5.7						
805		N	N	N	N	1	1	N		1	75	1	2	4	1.2	12.3						
5205		N	N	N	N	1	1					2	3	4	2.1	2.9						
5204		N	N	N	N	1	1					2	3	4	2.1	2.9						
4003		N	N	N	N	1	1	Y	1	2	0	1	1	2	1.0	9.9						
9105		N	N	N	N	2					0	1	1	4	0.5	3.3						
6606		N	N	N	N	1	2	N			0	1	2	4	0.9	5.7						
3404		N	N	N	N	1	1	Y	1	4	0	1	1	4	0.5	9.9						
1406		N	N	N	N	1	1	Y	1		0	1	2	1	0.8	25.6						
3407		N	N	N	N	1	1	N			0	1	1	4	0.5	9.9						
203		N	N	N	N	1	2	N			0	1	1	1	3.1	3.4						
2501		N	N	N	N	1	1	N		1	53	1	1	4	0.8	10.1						
3103	N	N	N	Y	N	1	1	Y	4	4	225	1	1	4	1.4	12.2	2/3	POS	-	+	KCPTB	1987

IDNUM	PREG	MINI XRAY RSLT	AGE	SEX	NUTRI STAT MED	RACE	M/STAT	OCCUP STAT	CAMP YEARS	YRS @ COMPY	WORK CAT	NO.OF LIVE BIRTH	NO.OF MISCAR	NO. OF INFANT DEATH	HBP	ASMT ALLG	TB	DIAB	ABDOM OP	SMOKE	AGE @ TIME OF SMOKING	NO.OF CIGAR. DAILY	COUGH
6103		NAD	5	M	2	B																	
4913		NAD	10	F	2	C																	
7505		NAD	15	M		C																	
3101		NAD	49	M	1	C																	
4305		NAD	17	F	0	C																	
9107		NAD	23	F	2	B																	
8804		NAD	35	M		C																	
204		NAD	7	M	-1	C																	
5103		NAD	50	M	1	B																	
6506		NAD	9	F	-2	C																	
5203		NAD	32	F	1	B																	
7107		NAD	6	M		C																	
5401		NAD	32	F		C																	
3405		NAD	5	M	0	B																	
502		NAD	49	F	1	C																	
9407		RAPTB	49	M	-2	C																	
5105		RSPTB	59	M	2	B																	
7610		RSPTB	23	M	-2	C																	
3602		NAD	51	M	-1	C	1	3	32	32		3M			N	N	N	N	N	Y	21	3	N
3603		NAD	36	F		C	5	1	4			5	0	0	N	N	N	N	N	Y	15	2	N
4304			2	M		C	6	0	2						N	Y	N	N	N				Y
4303		NAD	8	F	0	C	6	2	3						N	N	N	N	N	N			N
4301		NAD	25	F	1	C	1	3	3	10		2	0	0	N	N	N	N	Y				
8502			45	F		C	1	1	25			11	0	0	N	N	N	N	N	Y	20	1	N
8507		NAD	15	F	1	C	6	2	15						N	N	N	N	N	N			N
4905			26	M		C	1	3	4	0.5		3N			N	N	N	N	N	N	18	2	Y
7807			33	F		C	5	4	29			1	0	0	N	Y	N	N	N	Y	18	1	N
7804		NAD	21	F	-2	C	6	3	21	0.3	5	2	0	0	N	N	N	N	N	Y	15	2	N
7805		RAPTB	51	F	-2	C	1	1	28			12	0	1	N	N	N	N	N	Y	12	2	N
7704		NAD	25	M	-1	C	1	3	16	0.7	5				N	N	N	N	N	Y	18	1	N
802			21	F		C	6	4	15			3	0	0	N	N	N	N	N	Y	15	2	N
8204		NAD	10	M		C	6	2	10						N	N	N	N	N	N			N
809			0.	F		C	6	0	0.2						N	N	N	N	N				N
807			1	F		C	6	0	1						N	N	N	N	N				N
801		NAD	41	F	1	C	6	3	15	16		4	0	1	N	N	N	N	N	Y	19	2	N
1603		NAD	18	F	-2	C	6	3	15	2	5	1	0	0	N	N	N	N	N	N			N
8601		NAD	25	F	1	C	1	3	10	0.3	5	4	0	1	Y	N	N	N	N				N
806			4	F		C	6	0	4						N	N	N	N	N				N
8604		NAD	2	F	-2	C	6	0	2						N	N	N	N	N				N
8603			6	F		C	6	2	6						N	N	N	N	N				N
7202		NAD	26	M	-2	C	1	3	6	10		3N			N	N	N	N	N	Y	16	2	N
1404		NAD	5	F	1	I	6	2	5						N	N	N	N	N				N
9402		NAD	53	F	-1	C	1	1	2						N	N	N	N	N				N
1602		NAD	38	F	-2	C	6	3	20	3	5	2	1	0	N	N	N	N	N	Y	16	3	N
1403		NAD	9	M	0	I	6	2	5						N	N	N	N	Y	Y	17	4	Y
8508		NAD	11	F	-1	C	6	2	11						N	N	N	N	N	N			N
8506		NAD	18	F	-2	C	6	2	18			0	0		N	N	N	N	N	N			N
6304		NAD	12	F	-2	C	6	2	9						N	N	N	N	N	N			N
6206		NAD	23	F	-2	C	6	3	3	3		0	0		N	N	N	N	N	Y	15	2	N
6502		NAD	70	M	1	C	1	5	33						N	N	N	N	N	Y	16	1	N
6005		NAD	24	F	-1	C	6	3	18	1	5	0	0		N	N	N	N	N	Y	16	3	N
7504		NAD	16	F	-1	C	6	2	16			0	0		N	N	N	N	N	N			N
1606			20	F		C	6	3	1	1	5	0	0		N	N	N	N	N	Y	16	3	N
3701		NAD	40	F	-1	C	1	3	11	2	5	1	1	0	N	N	N	N	N	Y	16	2	N
5902		RAPTB	32	F	1	C	1	3	10	1	5	3	0	0	N	N	Y	N	N	N			N

IDNUM	BLD	TIRED	NIGHT SWEAT	WT LOSS	APPE TITE	BCG	BCG CONFM	SPORT /WEEK	SPORT YEARS	T3 KNWLD SCORE	TOT VOL OF ABSALC INTAKE ML IN 2 WEEKS	TYPE DWELL	TYPE TOILET	CROWD INDEX	PESL	TOT % FWAREA	SIGNS SYMPT	RPT XRAY	SPT	CULT	DISEA CATO	NOTIFI YEAR
6103																						
4913												1	2	4	0.9	9.9						
7505												1	1	4	0.7	9.9						
3101												1	2	2	1.0	5.5						
4305												1	1	4	1.4	12.2						
9107												1	1	1	1.3	9.9						
8504												1	1	4	0.5	3.3						
204												1	1	2	1.6	3.3						
5103												1	1	1	3.1	5.1						
6506												2	3	3	1.4	4.2						
5203												1	2	3	1.5	5.7						
7107												2	3	4	2.1	2.9						
5401												1	2	4	0.6	8.3						
3405												1	1	3	1.9	5						
502												1	1	4	0.5	9.9						
9407												1	1	4	2.7	4.5						
5105												1	1	4	0.6	3.3		LEFT CAMP				
7610												2	3	3	1.4	4.2		NEG	-	-		
3602		N	N	N	N	2		Y	4	2		1	2	4	1.2	5.8		LEFT COMPANY				
3603		N	N	N	N	2		N		3	100	1	1	4	1.2	5.2						
4304	N	N	N	N	N	1	1				113	1	1	4	1.2	5.2						
4303		N	N	N	N	1	1	Y	1		0	1	1	3	1.3	9.9						
4301		N	N	N	N	2		Y	1	5	50	1	1	3	1.3	9.9						
8502		N	N	N	N	2		N		2	0	1	1	3	1.3	9.9						
8507		N	N	N	N	1	1	Y	4	2	0	1	1	1	2.7	11						
4905		N	N	N	N	3		Y	4	2	150	1	1	1	2.7	11						
7807	N	Y	N	N	Y	3	1	N		4	125	1	1	4	0.7	9.9						
7904		N	N	N	N	1	1	N		4	155	1	2	4	0.3	5.8						
7905		N	N	N	N	2		N		4	225	1	2	4	0.3	5.8						
7704		N	N	N	N	1	1	Y	4	5	110	1	2	4	0.3	5.8	0/5	NEG	-	-		
802		N	N	N	N	1	1	N		2	50	1	2	4	0.8	5.8						
8204		N	N	N	N	1	1	O	2		0	1	1	4	1.2	12.3						
809		N	N	N	N	1	2					1	2	1	1.9	5.1						
807		N	N	N	N	1	2					1	2	4	1.2	12.3						
501		N	N	N	N	1	1	N		2	0	1	2	4	1.2	12.3						
1603		N	N	N	N	1	1	N		4	60	1	2	4	1.2	12.3						
8601		N	N	N	N	1	1	N		2	0	1	1	4	1.9	10.3						
506		N	N	N	N	1	2					1	1	1	0.9	3.3						
8604		N	N	N	N	1	1					1	2	4	1.2	12.3						
8603		N	N	Y	N	1	2	Y	1		0	1	1	1	0.9	3.3						
7202		N	N	Y	N	1	1	N		2	150	1	1	1	0.9	3.3						
1404		N	N	Y	N	1	2	N				1	2	2	1.6	8.3						
9402		N	N	Y	N	2		N		4	230	1	2	1	0.8	25.6						
1602		N	N	Y	N	3		N		1	113	1	1	4	0.6	3.3						
1403		N	N	N	N	1	2	Y	2		0	1	1	4	1.8	10.3						
8505		N	N	N	N	1	1	Y	2		0	1	2	1	0.8	25.6						
8506		N	N	N	N	3		N		2	0	1	1	1	2.7	11						
6304		N	N	N	N	2		N			0	1	1	1	2.7	11						
6206		N	N	N	N	2		N		3	0	1	2	3	1.2	9.9						
6502		N	N	N	N	3		N		2	256	1	2	4	2.0	9.9						
6005		N	N	N	N	2		N		2	0	1	2	3	1.5	5.7						
7504	N	N	N	N	N	2		N		2	0	1	2	4	1.3	12.2						
1606		N	N	N	N	2		N	4	2	0	1	2	2	1.0	5.6						
3701		N	N	N	Y	2		N		2	383	1	1	4	1.9	10.3						
5902		N	N	N	N	2	1	N		2	0	1	1	1	1.5	3.3						
										2	0	1	2	4	0.6	5.7		POS	+	+	KCPTB	1982

IDNUM	PREG	MINI XRAY RSLT	AGE	SEX	NUTRI STAT MED	RACE	M/STAT	OCCUP STAT	CAMP YEARS	YRS @ COMPY	WORK CAT	NO.OF LIVE BIRTH	NO.OF MISCAR	NO. OF INFANT DEATH	HBP	ASMT ALLG	TB	DIAB	ABDOM OP	SMOKE	AGE @ TIME OF SMOKING	NO.OF CIGAR. DAILY	COUGH
1508		NAD	30	F	-2	C		3	5	4	5	2	0	0	N	N	N	N	N	Y	19	2	N
5509		NAD	9	M	-2	C	6	2	9						N	N	N	N	N	N			N
6102		NAD	30	F	2	B	1	3	4	0.5	4	3	0	0	N	N	N	N	N	N			N
5510		NAD	11	F	-2	C	6	2	11						N	N	N	N	N	N			N
6006		NAD	24	F	1	C	6	3	16	3	5	2	0	1	N	N	N	N	N	Y	17	1	N
9404		NAD	12	F	0	C	6	2	2						N	N	N	N	N	N			N
1506		NAD	12	F	-1	C	6	2	7						N	N	N	N	N	N			N
7703		NAD	24	F	2	C	6	3	22	.1	2	3	0	0	N	N	N	N	N	N			N
6204		NAD	32	M	1	C	1	3	20	13	4				N	N	N	N	N	Y	14	2	Y
5602		NAD	27	M	-2	C	1	3	26	4	4				N	N	N	N	N	Y	15	1	N
4302		NAD	29	M	-1	C	1	3	7	4	4				N	N	N	N	N	Y	15	1	Y
5107		NAD	32	M	-1	B	1	3	1	3	5				N	N	N	N	N	Y	6	1	N
1304			14	F	-1	B	6	2	2						N	N	N	N	N	N			N
1303		NAD	9	M	0	B	6	2	2						N	N	N	N	N	N			N
7710		NAD	27	M	-2	C	6	3	7	3	4				N	N	N	N	N	Y	15	2	N
504		NAD	22	M	-2	C	6	3	18	1	5				N	N	N	N	N	Y	16	1	N
1103			14	F	-1	C	6	2	6						N	N	N	N	N	N			N
7005		NAD	10	F	0	C	6	2	10						N	N	N	N	N	Y	14	1	N
7607		NAD	16	F	-2	C	6	2	16			0	0		N	N	N	N	N	Y	14	1	N
7708			5	F	-2	C	6	0	5						N	N	N	N	N				N
7702			58	F		C	6	1	20			3	0	0	N	N	N	Y	N	N			N
7706		NAD	6	F	0	C	6	2	6						N	N	N	N	N	N			N
7808		NAD	15	M	-2	C	6	2	15						N	N	N	N	Y	Y	14	1	Y
7304		NAD	24	M	-1	C	6	3	24	0.5		3N			N	N	N	N	N	Y	20	3	N
7401		NAD	44	M	1	C	1	3	26	26		3N			N	N	N	N	N	Y	20	1	Y
3704		RSPTB	21	M	-1	C	6	3	18	2		3M			N	N	N	N	N	Y	16	3	Y
5504		RAPTB	27	F	-2	C	6	4	27			1	0	0	N	N	N	N	N	Y	13	2	N
8108		RSPTB	20	M	-1	C	1	3	3	4	5				N	N	N	Y	N	Y			N
5508			11	F	0	C	6	2	11						N	N	N	N	N	N			N
8103		NAD	15	F	-1	C	6	2	15			0	0		N	N	N	N	N	N			N
503		NAD	25	F	-2	C	6	3	24	2	4	1	0	0	N	N	N	N	N	Y	20	1	N
3605		NAD	7	M	-1	C	6	2	5						N	N	N	N	N	N			N
7605		NAD	19	F	-1	C	6	3	19	1	5	1	0	0	N	N	N	N	N	Y	13	3	Y
401			37	M		C	1	4	5						N	N	N	N	N	N			N
8402			39	M		C	1	3	10	6		3M			N	N	N	N	N	Y	22	1	N
6203			6	F		C	6	2	6						N	N	N	N	N	N			N
3604		NAD	14	M	-1	C	6	2	5						N	N	N	N	N	Y	10	1	N
6607		NAD	11	M	2	C	6	2	11						N	N	N	N	N	N			N
7305			21	M		C	6	3	21	3	4				N	N	N	N	N	Y	16	3	N

IDNUM	BLD	TIRED	NIGHT SWEAT	WT LOSS	APPE TITE	BCG	BCG CONFM	SPORT /WEEK	SPORT YEARS	TB KNWLD SCORE	TOT VOL OF ABSALC INTAKE ML IN 2 WEEKS	TYPE DWELL	TYPE TOILET	CROWD INDEX	PFSL	TOT % FWAREA	SIGHN SYMPT	RPT XRAY	SPT	CULT	DISEA CATO	NOTIFI YEAR
1508		N	N	Y	N	1	1	N		1	215	1	1	4	1.0	12.3						
6609		N	N	N	N	3		N			0	1	1	4	0.3	8.1						
6102		N	N	N	N	3		N		2	0	1	2	3	0.9	9.9						
5510		N	N	N	N	1	1	N			0	1	1	4	0.3	8.1						
6006		N	N	N	N	1	1	N		2	0	1	2	4	1.3	5.7						
9404		N	N	N	N	1	2	N			0	1	1	4	0.6	3.3						
1506		N	N	N	N	1	1	Y	1		0	1	1	4	1.0	12.3						
7703		N	N	N	N	3		N		4	0	1	2	4	0.8	5.8						
6204	N	N	N	N	N	1	1	N		2	300	1	2	4	1.3	9.9						
9602		Y	N	Y	N	1	1	N		1	120	1	1	1	0.9	3.3						
4302	N	N	N	N	N	2		Y	4	2	75	1	1	3	1.3	9.9						
5107		N	N	N	N	2		N		4	0	2	3	3	1.4	4.2						
1304		N	N	N	N	1	1	N		1	0	1	1	1	2.3	4.3						
1303		N	N	N	N	2		N			0	1	1	1	2.3	4.3						
7710		N	N	N	N	1	1	Y	2	2	38	1	2	4	0.8	5.8						
504		N	N	N	N	2		N		4	112	1	1	4	2.7	4.5						
1103		N	N	N	N	1	1	Y	2	1	0	1	1	3	1.3	12.3						
7005		N	N	N	N	1	1	N			0	1	2	3	1.1	5.7						
7607		N	Y	N	Y	1	1	[illegible]	1	2	35	1	2	4	1.2	5.8						
7708		N	N	[illegible]	N	1	2					1	2	4	0.8	5.8						
7702		N	N	N	N	3		N		4	0	1	2	4	0.8	5.8						
7706		Y	N	N	N	1	2	N			0	1	2	4	0.8	5.8						
7808	N	N	N	N	N	1	1	Y	1	4	10	1	2	4	0.3	5.8						
7304		N	N	N	N	1	1	Y	4	2	20	1	2	4	3.0	5.8						
7401		Y	N	N	N	1	1	Y	1	2	0	1	2	2	2.1	10						
3704	N	Y	Y	Y	Y	1	1	N		2	185	1	1	1	1.5	3.3	0/5	LEFT CAMP - UNABLE TO CONTACT				
5504	Y	Y	Y	Y	Y	2		N		2	1050	1	1	4	0.3	8.1	5/5	POS ++	+	PTB	1988	
8108		Y	N	N	N	1	1	Y	2	2	350	1	1	4	0.9	5.1		POS -	+	KCPTB	1984	
5508		N	N	N	N	1	1	Y	2		0	1	1	4	0.3	8.1						
8103		N	N	N	N	1	1	N		1	0	1	1	4	0.9	5.1						
503		N	N	N	N	3		N		5	40	1	1	4	2.7	4.5						
3605		N	N	N	N	1	1	N			0	1	1	4	1.2	8.2						
7605	N	Y	Y	N	N	1	1	N		4	105	1	2	4	1.2	5.8						
401		Y	N	Y	N	1	1	Y	2	3	225	1	1	1	2.1	3.4						
8402		N	N	N	N	2		Y	2	1	113	1	1	3	1.9	8						
6203		N	N	N	N	1	2	N			0	1	2	4	2.0	9.9						
3604		N	N	N	N	1	1	Y	1	1	35	1	1	4	1.2	8.2						
6607		N	N	N	N	1	1	Y	1		0	1	2	4	0.9	5.7						
7305		N	N	N	N	1	1	Y	4	1	35	1	2	4	3.0	5.8						

EPIDEMIOLOGY OF TB IN MACASSAR CAMP

IDNUM	PREG	MINI XRAY RSLT	AGE	SEX	NUTRI STAT MED	RACE	M/STAT	OCCUP STAT	CAMP YEARS	YRS @ COMPY	WORK CAT	NO.OF LIVE BIRTH	NO.OF MISCAR	NO. OF INFANT DEATH	HBP	ASMT ALLG	TB	DIAB	ABDOM OP	SMOKE	AGE @ TIME OF SMOKING	NO.OF CIGAR. DAILY	COUGH
1206			2	M		C																	
5106			30	M		C																	
3601			15	F		C																	
7705			5	F		C																	
7511			2	F		C																	
5005			16	F		C																	
5511			3	F		C																	
506			17	F		C																	
6609			22	M		C																	
201			32	M	1	C																	
5506			26	M		C																	
3102			24	F		C																	
3409			1	M		B																	
503			24	F		C																	
1701			26	F	-1	C																	
5404			4	M		C																	
1204			13	M		C																	
6908			2	M		C																	
9401			31	M		C																	
4004			5	F		C																	
9406			2	M		C																	
7004			17	F		C																	
7609			3	F		C																	
1104			39	M		C																	
5303			37	M	-1	B																	
5606			3	M		C																	
4602			47	M		B																	
504			28	F		C																	
6503			31	F		C																	
5502	PREG		27	F		C																	
3607			3	F		C																	
5603			23	F		C																	
6104			5	M		B																	
5107			27	F		C																	
5207			39	F		B																	
3106			17	M		C																	
6106			26	M		B																	
6401			37	F		B																	
9105			47	F	1	B																	
7106			4	M		C																	
4001			28	M		C																	
5503			2	F		C																	
6105			28	M		B																	
6105		NAD	26	F		B																	
1207		NAD	30	M		C																	
4912		NAD	19	M		C																	
3406		NAD	8	M	1	B																	
3401		NAD	46	M	1	B																	
9104		NAD	23	F	2	B																	
1703		NAD	33	F	-1	C																	
7001		NAD	40	M		C																	
3405		NAD	10	M		B																	
1302		NAD	28	F		B																	
1202		NAD	37	F		B																	
3403		NAD	15	F		B																	

EPIDEMIOLOGY OF TB IN MACASSAR CAMP

IDNUM	BLD	TIRED	NIGHT SWEAT	WT LOSS	APPE TITE	BCG	BCG CONFM	SPORT /WEEK	SPORT YEARS	TB KNWLD SCORE	TOT VOL OF ALCHL INTAKE IN 2 WEEKS	ML	TYPE DWELL	TYPE TOILET	CROWD INDEX	PPSL	TOT % FWAREA	SIGHN SYMPT	RPT XRAY	SPT	CULT	DISEA CATO	NOTIFI YEAR
1206																							
5106													1	1	4	0.7	12.7						
3601													1	1	4	0.9	5.1						
7705													1	2	4	1.2	5.2						
7511													1	2	4	0.8	5.5						
5605													1	2	4	0.3	5.3						
5611													1	1	4	0.6	5.1						
506													1	1	4	0.3	5.1						
6609													1	1	4	2.7	4.5						
201													1	2	4	0.9	5.7						
5506													1	1	1	3.1	3.4						
3102													1	1	4	0.3	5.1						
3409													1	1	4	1.4	12.2						
503													1	1	4	0.5	9.9						
1704													1	2	4	1.2	12.3						
9401													1	1	3	0.9	10.3						
1204													1	1	3	1.9	8						
6906													1	1	4	0.7	12.7						
9401													1	2	4	0.9	5.7						
4004													1	1	4	0.6	3.3						
9406													1	1	2	1.0	9.9						
7004													1	1	4	0.6	3.3						
7609													1	2	3	1.1	5.7						
1104													1	2	4	1.2	5.8						
5303													1	1	3	1.3	12.3						
5606													2	3	3	1.3	3.1						
4602													1	1	4	0.6	8.1						
504													1	1	3	0.8	9.9						
6503													1	2	4	1.2	4.3						
8802													1	2	3	1.5	5.7						
3607													1	1	2	1.6	3.3						
5603													1	1	4	1.2	5.2						
6104													1	1	4	0.6	8.1						
8107,													1	2	4	0.9	9.9						
5207													1	1	4	0.9	5.1						
3106													2	3	4	2.1	2.9						
6106													1	1	4	1.4	12.2						
6401													1	2	4	0.9	9.9						
9105													1	2	1	2.2	9.9						
7106													1	1	4	0.5	3.3						
4001													1	2	4	0.6	5.3						
8803													1	1	2	1.0	9.9						
6105													1	1	2	1.6	3.3						
6105													1	2	4	0.9	9.9						
1207													1	2	4	0.9	9.9						
4912													1	1	4	0.7	12.7						
3406													1	1	4	0.7	9.9						
3401													1	1	4	0.5	9.9						
9104													1	1	4	0.5	9.9						
1703													1	1	4	0.5	3.3						
7001													1	1	3	0.9	10.3						
3405													1	2	3	1.1	5.7						
1302													1	1	4	0.5	9.9						
1202													1	1	1	2.3	4.3						
3403													1	1	4	0.7	12.7						
													1	1	4	0.5	9.9						

APPENDIX F5a

Calculation of Correlation Coefficient of Positive Mantoux & TB ELISA Tests of Macassar Camp Residents

Correlation coefficient: $r = -0,55$
$r^2 = 0,30$
95% confidence limits: $-0,90 < R < 0,25$

Source	df	Sum of Squares	Mean Square	F-statistic
Regression	1	0,1152	0,1152	2,63
Residuals	6	0,2625	0,0437	
Total	7	0,3776		

Coefficients

Variable	Mean	B coefficient	95% confidence Lower	Upper	Std Error	Partial F-test
MXREAD	18.0500	-0,0394532	-0,087114	0,008207	0,024316	2,632
Y-Intercept		1,2140058				

APPENDIX F5b

Calculation of Correlation Coefficient of Positive Mantoux & TB ELISA tests of Macassar Camp Residents which excluded the TB ELISA Outlier Reading

Correlation coefficient: $r = 0,01$
$r^2 = 0,00$
95% confidence limits: $-0,75 < R < 0,76$

Source	df	Sum of Squares	Mean Square	F-statistic
Regression	1	0,0067	0,0067	0,00
Residuals	5	43,6618	8,7324	
Total	6	43,6686		

B Coefficients

Variable	Mean	B coefficient	95% confidence Lower	Upper	Std Error	Partial F-test
ELISA	0,4304	0,2707382	-18,849623	19,391099	9,755286	0,0008
Y-Intercept		18,6691808				