Socio-economic Determinants of Tuberculosis and of Risk Taking Behaviour

Respiratory tuberculosis in the twentieth century and the decision to smoke

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November 2013

For Little Dot the duck

Abstract

This thesis is separated into two distinct parts. The first of these investigates the extent to which tuberculosis morbidity and mortality in the twentieth century were determined by the conditions in which people worked and lived and how institutional responses to the tuberculosis problem shaped individual chances of survival locally, nationally and internationally. We collate several new datasets to investigate socio-economic determinants of tuberculosis disease. Firstly, we use newly sourced data to investigate to what extent, why and to what effect different areas in interwar Britain pursued different policy agendas and how these affected tuberculosis outcomes, both through qualitative discussion and through the application of statistical methods. We argue that where local authorities identified tuberculosis as a socio-economic phenomenon, outcomes were favourable. Secondly, we identify a tripartite divide between developing, newly industrialising and developed countries after the introduction of antimicrobial drugs. We explain why peoples in some parts of the world did not benefit from the promises of prevention and cure enjoyed those in the West. Thirdly, we construct new a data set to investigate the cross country determinants of tuberculosis morbidity and mortality and to observe this tripartite divide, with special focus on the nutritional composition of diet, in the post war period. We find a tuberculosis Kuznets relationship and that adequate nutrition and living conditions most strongly predict tuberculosis outcomes. Finally, we ask whether, in the current era of drug resistant tuberculosis, nutrition science can be utilised to prevent the development of active symptoms if drugs cannot cure the disease. In the second part, individual level data sourced from the World Health Organisation is used to investigate whether background risks to health influence the decision to smoke. A modified double hurdle model of the decision to smoke reveals that background health risks may increase smoking, contrary to theoretical prediction, but that the effect is moderate.

Contents

Abstract 2

List of Tables 6

List of Figures 8

Acknowledgements 9

Author's Declaration 10

Introduction to thesis 11

1. Poverty, health and decentralised health decision policies: Tuberculosis mortality and fatality in interwar England and Wales 17

1.1 Introduction 17

1.2 Background 18

1.3 Sources and methodology 26

1.4 The Interwar Experience 27

1.5 Explaining differences in morbidity and mortality 30

1.5.1 Morbidity 30

1.5.2 Mortality 32

1.5.3 Fatality 34

1.6 Policy interventions: decentralised health decisions in the interwar period 35

1.6.1 Identifying the disease 36

1.6.2 'Curing' the disease 40

1.6.3 Non-residential care 42

1.6.4 Preventing the disease 46

1.6.5 Institutions and Personalities 50

1.7 Why did some areas do well, but others not so well? 54

1.8 Conclusion 67

1.9 Appendix 70

2. Health Expectations and Health Achievements: Respiratory Tuberculosis in the Global Economy between 1950 and 1980: A Developing Economy Perspective 94

2.1 Introduction 94

2.2 Data and Findings 95

2.2.1 Mortality 96

2.2.2 Morbidity 99

2.3 An emerging market economy problem? 100

2.4 The limitations of 'developed world' medical science and public health innovations in a developing economy perspective 106

2.4.1 Containing Transmission 107
2.4.2 Preventing infection via vaccination 109
2.4.3 Curing the Infected 112
2.5 Conclusion 115
2.6 Appendix 117
3. Exploring Reasons for the Variation in the Global Tuberculosis Problem 1950-1980 127
3.1 Introduction 127
3.2.1 Data sources 132
3.2.2 Data Issues 132
3.2.3 Descriptive statistics 135
3.3 Panel Data Methods 137
3.3.1 GMM estimation of the tuberculosis Kuznets curve 138
3.3.2 Random effects, fixed effects and GMM estimation of the unrestricted model 142
3.4 Empirical Results for GMM estimation of the tuberculosis Kuznets curve 143
3.4.1 Empirical Results for the unrestricted model 146
3.4.2 Empirical results for the unrestricted model including source of calories 150
3.5 Discussion 151
3.5.1 The tuberculosis Kuznets curve 151
3.5.2 Nutrition 152
3.5.3 Medical provision 156
3.5.4 Insolation 157
3.5.5 Urbanisation and industrialisation 158
3.6 Conclusions 160
3.6 Appendix 163
4. Nutrition, standards of living and the future of tuberculosis prevention 182
4.1 Introduction 182
4.2 MDR-TB and XDR-TB 183
4.3 Socio-economic strategies to control TB 188
4.4 Nutritional approaches to prevent latent TB from becoming active 191
4.4.1 TB pathogenesis and nutrition 191
4.4.2 In vivo studies 192
4.5 Conclusions 200
5. Background health-related risks and smoking choices in developing and emerging market countries 202

5.1 Introduction 202
5.2 Background 205
5.2.1 Theoretical Literature 205
5.2.2 Empirical Literature 206
5.3 Data 209
5.4 The risk factors of households surveyed in WHS 215
5.5 Empirical Methodology 216
5.6 Results 222
5.7 Conclusion 226
5.8 Appendix A 228
5.9 Appendix B 252
Conclusion to thesis 254
Bibliography and references 264
Primary Sources 264
National Archives 264
West Yorkshire Archives (Wakefield) 267
Official Publications 267
Secondary Literature 270

List of Tables

Table 1.1 Summary Statistics: Respiratory Tuberculosis Mortality in English and Welsh Counties (crude death rate per million living) 70

Table 1.2 Summary Statistics: Respiratory Tuberculosis Mortality in English and Welsh Towns (crude death rate per million living) 71

Table 1.3 Achievements in the Control of Respiratory Tuberculosis Mortality: Towns in England and Wales 72

Table 1.4 Male Unemployment in 1931: Summary Statistics 73

Table 1.5 Overcrowding in Housing England and Wales in 1921 and 1931: Summary Statistics 74

Table 1.6 Residential Policy for Patients with Tuberculosis in 1929 75

Table 1.7 Summary Statistics Community Based Activities in 1929 76

Table 1.8 Efficiency of Diagnosis in 1929: Summary Statistics 78

Table 1.9 The socio-economic and medical determinants of case rates (OLS) 79

Table 1.10 The socio-economic and medical determinants of death rates: robust OLS 80

Table 1.11 The socio-economic and medical determinants of death rates: Quantile Regressions 83

Table 1.12 The socio-economic and medical determinants of death rates: Conditional Quantile Regressions 85

Table 1.13 The socio-economic determinants of the case fatality ratio (OLS) .. 86

Table 1.14 Respiratory Tuberculosis: Crude death rate per mill pop at all ages: Summary Statistics 88

Table 1.15 Efficiency of Clinical Work 89

Table 1.16 Use of Sanatoria: Summary Statistics 90

Table 1.17 ‘Care in the Community’ 91

Table 2.1 Respiratory Tuberculosis Death Rates in 1965 117

Table 2.2 Incidence of Respiratory Tuberculosis in 1967 121

Table 2.3 Patients Treated in Various Hospitals and Dispensaries by Disease: All India 123

Table 2.4 India: Patients Treated in Hospitals and Dispensaries for Respiratory Tuberculosis 124

Table 3.1 Average calories consumed and annual rate of growth of urban

population for selected countries, 1967. 163
Table 3.2 GMM estimation of the tuberculosis Kuznets curve 165
Table 3.3 Random effects, fixed effects and Arellano-Bond GMM estimation of the unrestricted model for morbidity and mortality 167
Table 3.4 Arellano-Bond GMM estimation of the effect of individual food groups on tuberculosis morbidity and mortality 169
Table 3.5 ILO data availability 170
Table 3.6 Good policies and bad policies by country 175
Table 5.1 Variable name, type, definition, related survey question and notes 228
Table 5.2 Sample characteristics for total sample, selected tobacco sample and tobacco regression sample 234
Table 5.3 Probit, Truncated Regression and Tobit models 237
Table 5.4 Double Hurdle and Box Cox Double Hurdle models 241
Table 5.5 Box Cox Double Hurdle estimates for restricted models 245
Table 5.6 Countries Surveyed in the WHS in partnership with WHO, 2002 – 2004 252

List of Figures

Figure 1.1 Crude Death Rate per 1 million lving from respiratory tuberculosis in England and Wales 92

Figure 1.2 Residual verses fitted plot using data derived from the Registrar General's Annual Review 93

Figure 1.3 Residual verses fitted plot using data derived from Ministry of Health's notification register.....93

Figure 2.1 GNI per capita and respiratory tuberculosis mortality: Japan.....125

Figure 2.2 GNI per capita and respiratory tuberculosis mortality: Hong Kong 125

Figure 2.3 GNI per capita and respiratory tuberculosis morbidity: Japan.....126

Figure 2.4 GNI per capita and respiratory tuberculosis morbidity: Hong Kong126

Figure 3.1 Respiratory tuberculosis case rates in selected Developing Countries 178

Figure 3.2 Respiratory tuberculosis case rates in selected Newly Industrialised Countries.....178

Figure 3.3 Respiratory tuberculosis case rates in selected Developed Countries 179

Figure 3.4 Respiratory tuberculosis death rates in seleced Developing Countries 179

Figure 3.5 Respiratory tuberculosis death rates in selected Newly Industrialising Countries.....180

Figure 3.6 Respiratory tuberculosis death rates in selected Developed Countries 180

Figure 3.7 Tuberculosis Morbidity Kuznets curve 181

Figure 3.8 Tuberculosis Mortality Kuznets curve 181

Figure 5.1 Frequency Distribution of TOBACCO.....251

Figure 5.2 Frequency Distribution of TOBACCO > 0 251

Acknowledgements

First and foremost I thank my supervisor, Professor Sue Bowden, for her tireless support and collaboration and reminding me of the importance of seeing the wood for the trees. I also would like to extend my gratitude to Professor Peter Simmons and Dr. Martin Forster for their detailed consideration of my work, Dr. Matthias Morys for his enthusiastic and critical insights, Dr. David Clayton for his pastoral support that preceded and motivated my doctoral study, Dr. Judith Spicksley for her curiosity and thoughtful comment, Professor Paul Mosley and to the many others that have offered their attention and insights at the Centre for Historical Economics and Related Research at York. I am also grateful for the technical help of Dr. Joao Jalles and for the assistance of the administrative staff in the Economics Department, in particular Jo Hall.

I would like to thank my family and friends. I want to thank Nathalie Sinyard for her supportive companionship in all stages of the production of this work. I would like to thank my parents, Stuart and Angela for their financial and emotional support. I would like to thank Troy Stutely for his tireless interest in my well being during this process, as well as his help in checking the final document for errors in spelling and grammar. All remaining errors are my own. I would like to thank Neil and Lesley Sinyard for their advice, time and generous provision of respite. I would like to thank my brother, Lawrence. Finally, I must thank the ducks, Pipe, Peep, Duck Davies, Googie and Crowbar for providing duck based therapy.

Author's Declaration

I, Alex Sadler, declare that this thesis, and the work present within it, are my own unless otherwise stated and has been generated by me as the result of my own original research. I confirm that this work was done wholly during candidature for a PhD in Economics at the University of York. Where I have consulted the published work of others, this is always clearly attributed. Where I have quoted from the work of others, the source is always given. I have acknowledged all main sources of help. Where the thesis is based on work done by myself jointly with others, the name of the co-author is given. Chapter 1 is joint work for which my contribution is fifty per cent and Professor Sue Bowden's contribution is fifty per cent. Chapter 2 is joint work for which my contribution is fifty per cent and Professor Sue Bowden's contribution is fifty per cent. Chapter 3 is joint work for which my contribution is fifty per cent and Professor Sue Bowden's contribution is fifty per cent. Chapter 4 and Chapter 5 are my work only.

Introduction to thesis

Risks to health interact with economic circumstances, often in surprising ways. The risk of acquiring an infectious disease, frequently characterised as indifferent between striking the rich or poor, has, since at least the turn of the 20th century, been increasingly understood in terms of environment. Exhaustion, poverty and deprivation have complemented the epidemiological explanation of transmission and infection processes to explain why the burden of infectious disease varies so substantially for different peoples living and working in different communities. Historically, the risk of acquiring tuberculosis has been attributed to differences in living and working conditions, in war and during peacetime. Where living conditions were cramped and working conditions were poor and when food was scarce, especially during periods of war, respiratory tuberculosis surged throughout the populations of Europe.

In this thesis, the control of tuberculosis is divided into three distinct historical episodes. The first episode began in 1882 after Robert Koch published his discovery that *mycobacterium tuberculosis w*as the microbial agent responsible for the disease, eventually dispelling the older idea that tuberculosis was an inherited condition. During this period the means of identifying the infected became available, but crucially, a cure did not. The early 20th century approach to the tuberculosis problem was characterised by the use of the sanatoria, ostensibly to offer convalescence to the infected but more pragmatically to prevent the spread of disease throughout increasingly crowded towns and cities. In other words, eradication of this scourge was not a meaningful consideration of policy makers, doctors, patients or their families but control and management was. The discovery of streptomycin 1943 by Albert Schatz and the first use of streptomycin in clinical trials 1946 – 1947 promised to change this narrative. By 1948, tuberculosis patients were already being treated with this new antibiotic drug. From there, innovations in antimicrobial drugs arrived rapidly and soon new drugs that were more effective in curing the disease and had fewer side effects were in widespread clinical usage; at least in the West. The development of antimicrobial medicines, which brought with them the promise

of ending the centuries of suffering a death caused by tuberculosis, marks the end of the first episode.

This unique episode has important qualitative characteristics that chapter 1 exploits. Tuberculosis was for the first time accurately recognised as an infectious disease and this offered policy makers and individuals information not hitherto available; that tuberculosis could be managed with judicious practices. This approach seemed to be corroborated by two important observations. The first was since at least 1850 estimated mortality rates from tuberculosis had been falling in England and Wales, despite the absence of any curative medicine at all and little in the way of progressive public health effort. The second observation was that wartime environments and greater incidence of tuberculosis in the human often went in concert. For some contemporaries these observations motivated the idea that tuberculosis control could be undertaken at a local level, not with the use of a magic pill, but through careful examination of the socio-economic factors that determined the disease. We exploit these circumstances, in chapter 1, to study the success and the failure of English counties and towns to address the tuberculosis problem in the interwar period where the aetiology of the disease was known, but conventional medical intervention was not. We use both a quantitative and qualitative approach to develop the case that in interwar Britain, much of the variation in incidence and mortality rates between English counties and towns can be explained by socio-economic factors. We also argue that judicious contemporaries in local authorities and in healthcare were, using the available scientific knowledge, correct in their focus on improving living and working conditions, nutrition and, such as it was, medical provision. The reasons for this variation is probed in chapter 1 using newly constructed data derived from Ministry of Health Returns sourced from the National Archives at Kew, and supplemented by data from the Census of Population, 1931. The contribution of chapter 1 is not only to explore the correlations between socio-economic determinants of tuberculosis and the burden of the disease, but also to gain a qualitative understanding of why, during this episode, some towns and counties were able to achieve remarkable reductions in rates of tuberculosis death, whilst others were not.

The second episode is bounded on one side by the introduction and subsequent mass

usage of drugs that could kill *mycobacterium tuberculosis* in human hosts and on the other by mutations in the genome of the bacteria that later prevented its destruction and with the advent of HIV co-infection in the 1980s. This “Golden Age” began triumphantly in the late 1940s and early 1950s when those in the developed world, with access to these new medicines were able take seriously the idea that tuberculosis could be eliminated as a public health problem. Public health successes in tuberculosis control were not, however, paralleled in the developing world. Developed countries were sufficiently resourced to ensure that where the need arose, treatment could be produced, delivered and monitored, but developing economies were not. Chapter 2 focuses on the second episode in the control of tuberculosis on a global basis. This was a period in which the potential existed for the reach of the disease to be dramatically shortened and for human misery to be alleviated, as occurred in post-war Europe and North America. That the same pattern was not observed in developing countries is the motivation for chapter 2. Here, a broader data set than that which was previously available is used to understand why, despite the promise of scientific advance, tuberculosis remained such a burden in developing countries and for so long. Chapter 2 contributes by investigating, not only the practical reasons for a lack of success in developing countries, but also the telling experience of emerging market economies. Rapid urbanisation and overcrowding coincided not only with the availability of new drugs but also with unprecedented rates of tuberculosis mortality and morbidity, thus leading to the idea that development itself can provoke a worsening in the tuberculosis problem if urban health systems cannot keep pace.

As we did with interwar Britain we seek quantitative corroboration on a global basis for the observation of chapter 2 that rapid urbanisation might explain death rates. We also ask whether the global burden of tuberculosis is informed by the risks to the person or on the quality of medical institutions or both. Chapter 3 contributes by collating and analysing a global dataset comprising mortality, morbidity and measures of healthcare development derived from World Health Statistics Annual, nutrition data from the World Health Organisation (WHO) Global database on body mass index and urbanisation data from the United Nations Demographic Yearbook. Chapter 3 contributes by assessing the relative importance of each of these factors. In addition, this chapter investigates rich data on the nutritive components of diet

collated at the country level by the WHO. It also tests the hypothesis generated in chapter 2 that a tuberculosis 'Kuznets' curve characterises the relationship between tuberculosis mortality and morbidity and income.

Various hypotheses in the historical and contemporary literature have argued that low protein calories, low total calories or a lack of specific nutrients such as vitamin D increase the susceptibility of individuals to activation of the disease and death. Hence we use a qualitative approach to assess whether variation in dietary composition across countries is related to variation in tuberculosis morbidity and mortality rates. We also recognise that during this episode medical science had the ability to cure the disease. Medical data are more reliable and more complete for the European sub-sample investigated in our other work (Bowden, Jalles, Pereira and Sadler, 2014). Thus, in chapter 3 we compare our findings on a global basis with our findings from Europe in the post-war period.

The second episode in the control of tuberculosis came to a halt in some parts of the world at different times and continues in others. In the former Soviet Union for example, the collapse of public health infrastructure and services in the early 1990s led to a failure of the national tuberculosis program to regulate the administration of drugs. This led not only to an increase in the numbers of infected patients, but crucially to the dissemination of mycobacteria resistant to first line drugs. At the same time, growing HIV / AIDS problems in sub-Saharan Africa emerged from isolation in the basin of the river Congo and spread out among the populations of Africa. Kinshasa, Zaire is believed to have played host to the first HIV epidemic in the 1970s. Subsequent research has revealed that death rates from tuberculosis were higher among Kinshasa outpatients that were also infected with HIV (Williame, 1989). By the 1980s HIV was carried into Eastern Africa, Russia, India and beyond. Thus, the third episode is one in which the future of tuberculosis treatment is uncertain. In many areas what was uncertainty about the future reliability of antimicrobial treatments for tuberculosis has become certainty that existing methods are no longer effective. Where a patient is infected with a drug resistant strain of mycobacterium tuberculosis, or where a patient is infected with HIV the medical response is similar to that of the first episode. Again the emphasis is on the control of transmission as discussed in chapter 1. But alongside this is the recognition of the

consequences of allowing the features of the second episode to slip away. Prolonging the era of effective treatment of tuberculosis is the driving force behind global efforts, instigated by the WHO, to reduce the likelihood that drug resistant strains will proliferate more widely among populations. Thus the third episode is, at present, a local one with worrisome global potential.

Chapter 4 reviews the literature to deliver evidence that suggests that the trend is towards increasing antimicrobial resistance. In this context chapter 4 draws on the lessons learned by policy makers and healthcare professionals in the interwar period in chapter 1 to control the disease the last time effective treatments were not available. Chapter 4 also draws inspiration from findings in chapter 3 concerning the importance of adequate nutrition. It asks whether modern nutrition science offers any prophylactic measures that individuals and governments can take to reduce the risk that tuberculosis will become active, at least in patients that do not have HIV/AIDS, in the context of a world in which conventional antimicrobial medicines are increasingly ineffective. Thus chapter 4 reviews the recent nutrition science literature to discover whether or not there exists an effective intervention available that will reduce the chances that individuals will become infected. Thus the third episode is one in which the scholar is privileged to draw upon accumulated knowledge to find alternative tuberculosis control strategies not available to those under similar circumstances in the first. The contribution of chapter 4 is to ask whether this privilege of knowledge, with special focus on nutrition, offers any practical solutions to the tuberculosis problem in the context of the failure of antimicrobial medicines. Chapter 4 suggests that the outcome of the third episode in tuberculosis control is itself uncertain. It may lead to a return to the first because the curative power of medicine is outpaced by the indifferent mutation of pathogens such as the HIV virus or mycobacterium or it may lead to a fourth where, once more, the promise of deliverance is credible.

Chapter 5 departs from the focus on tuberculosis of the first four chapters but retains focus on health risks. Environmental risks may not only be risk factors that generate susceptibility to disease, they may also influence the individual in substantive ways to take fewer risks that are under their control. Early theoretical treatment regarded risks as one-dimensional and defined risk aversion to these risks. But later

developments recognised that decision making regarding risks takes places in the context of multiple independent risks. Infection by *mycobacterium tuberculosis* is, for all practical purposes, outside of the control of the decision maker. The social determinants of tuberculosis morbidity and mortality are, therefore, in effect, risk factors that the individual cannot avoid which influence the chance that an individual will have some associated illness. Individuals can however choose some risks to their health including whether or not to smoke. Thus chapter 5 turns the previous analysis on its head. Instead of asking what environmental risk factors are associated with disease, it asks whether risks to health themselves influence health related decision making in the context of the choice of whether or not to smoke. Theoretical justification for the phenomenon and empirical validation is presented in chapter 5, for the special case of risks to wealth analysed in the extant literature. Chapter 5 innovates by asking whether or not, when risks are related to health, the phenomenon persists. To do this, chapter 5 constructs a household-level dataset from the 2002 WHO World Health Survey that includes data on whether or not respondents smoked. It also includes a wealth of data on the environmental risks to which individuals are subject in their household and community environment. WHS data are supplemented by data from the Uppsala Conflict Data Program and WHO Statistical Information System to examine country-specific risk environments. Suitable econometric approaches are discussed and justified.

Risks to health are an important feature of disease aetiology whether in the form of unwanted infection by *mycobacterium tuberculosis* or by chosen actions such as smoking. In the case of the former, risks to health interact with economic factors to a degree such that disease rates can be considered pillar of the standard of living to the same extent as measuring income. It in this vein that focus now turns to the case of tuberculosis in interwar England and Wales.

1. Poverty, health and decentralised health decision policies: Tuberculosis mortality and fatality in interwar England and Wales

Sue Bowden and Alex Sadler

1.1 Introduction

For centuries, tuberculosis was one of the major causes of illness and death amongst peoples. It is a disease that has attracted a wide literature, notably from an economic history perspective in terms of its increase during times of urbanisation and industrialisation to its decrease at a time when medical intervention in terms of prevention and cure were limited but standards of living, at least in the developed world were beginning to improve. Insofar as the disease impeded the infected to live a long and productive working life, it has implications in human development parlance as a constraint on the abilities of peoples to acquire and use the capacity to live long and healthy lives. Equally in standard of living parlance, illness and death from the disease represent a new way of looking at the conditions in which peoples lived and worked.

This chapter considers the case of respiratory tuberculosis in interwar England and Wales for three reasons. Section 1.2 discusses recent suggestions in the literature that respiratory tuberculosis is an alternative measure of and complement to the more common measures of standards of living (Bowden et al., 2014). The conditions under which infection spread were largely related to poor (overcrowded) living and working conditions; amongst those infected those suffering from poor nutrition came to have active symptoms of the disease (see below). Section 1.3 discusses the sources from which data were derived, which data were selected and for what use. Section 1.4 examines how and why, in the face of the depressed conditions of this period, death and case rates of the disease were contained. But in so doing, we identify those parts of the country – differentiating between and amongst rural and urban areas and London metropolitan boroughs – that best succeeded in dealing with the illness. In section 1.5 we apply statistical testing to explore the explanatory

powers of a range of explanatory factors focusing on the long standing debate between the standard of living and the medical initiatives schools of thought.

In sections 1.6 and 1.7 we exploit this period as a case study in devolved health decision making. Modern day economic and political discourse favours, in theory at least, devolution of decision making in the allocation of health resources to the local level. Local rather than central decisions are, it is claimed, optimal in dealing with the specific health needs and the allocation of scarce resources of local communities. A study of respiratory tuberculosis in the interwar period in England and Wales, prior to the introduction of the National Health Service, allows us to examine this view. Prior to the National Health Service, health systems in this country were operated by public health services at a local level. Local authorities had considerable latitude in their choices but were, as ever, constrained both by the income at their disposal and the disease regimes with which they were faced. There were considerable differences in policy approaches and strategic choices in the prioritisation of policies to deal with the totality of the public health problems at this time. Our question is to what extent, why and to what effect different areas pursued different policy agendas and in what ways these agendas affected tuberculosis outcomes. Our motivation is based on the observation (see below) that there were significant variations between administrative areas in terms of death rates. Our concern is to identify what authorities could and did do in the face of prevailing knowledge and medical technology and then to discuss in detail what a selected sample of authorities did which could explain why they did particularly well – or particularly badly. At this time, prior to the National Health Service, local authorities had considerable freedom and power in defining and implementing policies. Given the onus today in reverting to a more local as opposed to a centralised system, we felt it timely to investigate how and why such differences occurred. In addition, we wanted to explore what authorities did and did not do– at a time when no reliable chemotherapeutic cure existed – with lessons for today's world of drug resistant tuberculosis.

1.2 Background

The decline in respiratory tuberculosis mortality in England and Wales pre-dated the introduction of chemotherapeutic interventions to cure the disease (Figure 1.1). From the middle of the nineteenth century to the beginning of the Great War the crude death rate had fallen by more than half from 2565.0 per million in the period 1858-60 to 1108.8 per million in 1908-10. The effect of the war increased the crude death rate approximately 35 per cent above its pre-war level (MH 55/139, Table 1, p.6). Thereafter, tuberculosis death rates continued their long-term decline in England and Wales. London saw a decline of 25 per cent in death rates, with some boroughs, including Hackney, experiencing a 50 per cent decline (MH 66/1081, p. 41). Nonetheless as we note below, people in some parts of the country were more at risk from contracting and dying from the disease than others.

The decline in respiratory tuberculosis mortality formed part of a general trend of a decline in death rates in this country – amongst babies, amongst children and amongst adults. There has long been a heated debate as to why this occurred. For some observers, this forms part of the wider debate on the reasons for the modern rise in the size of populations. Some argue that the decline in mortality in England and the USA from 1901 owed little to medical *intervention* (McKinlay et al., 1977; McKeown et al., 1972). Most famously McKeown in the 1970s set out his proposition that the modern rise in populations owed nothing to medical science (McKeown 1976a and b)).[1] The thesis is that health improvements derived less from medical interventions and public health initiatives than to improvements in living conditions and in particular to improvements in nutrition. Improvements in nutrition derived from changes in agricultural productivity. In fact, McKeown was adamant that medical science played little part in explaining the fall in mortality until well into the twentieth century. Mckeown was dismissive of the role of quarantine and vaccination.[2] Perhaps most controversial of all was his argument that sanitary reform played little part in explaining the decline in mortality.

[1] McKeown was concerned with the long run rise in the global population. His overall thesis was that the rise in the population was explained by the decline in mortality. The work of the Cambridge Group found that the rise in the population of England was determined by the rise in fertility (Wrigley and Schofield, 1981). A comprehensive review of the debate is provided by Colgrove (2002).

[2] Although he did acknowledge that vaccination was important in relation to smallpox.

Not surprisingly, his thesis provoked heated debate and controversy. McKeown was criticised for his over-reliance on changes in nutrition, for ignoring the role played by vaccination and for under-playing the role of public health initiatives as in the improvement in clean water. There was also debate as to how changed living conditions arising from industrialisation and urbanisation impacted on mortality. Szreter (1988) argued that the very process of industrialisation and urbanisation worsened the living and working conditions for the bulk of the population and that public health – with its political (or as Szreter defines it, 'practical politics') and institutional manifestations were crucial mechanisms for delivering improved living conditions (Szreter, 2002). Other authors widened the argument and definition of public health by pointing out the improvements in housing (the reduction of overcrowding and greater access to natural light), in education and the introduction from the early twentieth century of minimum social services which, it is argued, could have driven the decline in mortality (Martini et al., 1977; Auster et al., 1972). Link and Phelan (2002) have delivered a robust argument in favour of the importance of social conditions in determining disease and the role of public health in ameliorating these conditions.

The above informs the analysis undertaken in this chapter. Mature industrial economies achieved reductions in mortality prior to the introduction of drug regimes that could cure respiratory tuberculosis. To that extent, there is debate in the literature as to why this decline took place (Wilson, 1990). For many years it has been noted that overcrowding increases the risk of infection whilst nutrition increases the risk of developing active symptoms of respiratory tuberculosis. There is a large historical and contemporary scientific literature which argues that infection is the result of sustained exposure to the bacillus – in both working and living conditions. Hence as overcrowding takes place (as with industrialization and urbanisation), the risks of infection increase and, as living standards improve and overcrowding diminishes, so the risks decrease. The association between tuberculosis and overcrowding dates back to the increase in tuberculosis mortality in the sixteenth century amongst urban populations (Daniel, 1997, pp. 22-27; Johnston, 2003, p. 339). The association was furthered by analyses (notably confined to analyses of Western Europe and the USA) linking tuberculosis mortality with increased overcrowding conditions associated with industrialisation and urbanisation

in the West and in North America (Bates, 1992; Bryder, 1988; Burnet, 1932, pp. 502-510; Dormandy, 1999; Dubos R & J, 1952; Ott, 1996; Rothman, 1994; Smith, 1988; Teller, 1985). The observation that overcrowding facilitates the spread of disease was noted in both wartime England (McDougall, 1952, p. 116; Medical Research Council, 1942, pp. 8-9) and Europe (Biraud, 1943/4, p. 572 and 687; Doull 1945, p.786).

It has been argued that of those who become infected, only 10–12% will develop tuberculosis disease; the development of active symptoms can take any time between a few weeks and several decades (Borgdorff et al., 2002, p. 217). What makes conditions in which the disease spreads into the conditions in which active forms of the disease occur was suggested in a seminal piece by Besanco, who argued that under-nourishment alone brought about an increase of 50 per cent in the number of active cases (Besancon, 1945, p. 287). His work found support from retrospective analyses: surveys of pre-war England found that under-nourishment was a highly significant predisposing factor (McDougall, 1952, p. 116) and in work undertaken on the Second World War. McDougall, for example, demonstrated that nutritional deficiency during the Second World War was linked to tuberculosis mortality increases in Belgium, France and the Netherlands and also, but to a lesser extent, in Italy, Spain and Hungary (McDougall, 1950, p. 241). Fifty years later, Smallman-Raynor and Cliff argued that one of the specific factors that contributed to wartime increases in active tuberculosis was protein-calorie malnutrition (Smallman-Raynor and Cliff, 2003 and 2004, p. 647). These middle twentieth century reports and retrospective empirical analyses have found support in recent scientific work (Johnston, 2003, 337). Crucially, this research has demonstrated that while infection rates are not influenced by a person's weight; underweight individuals who are more likely to be suffering from nutritional inadequacies are more likely to experience subsequent tuberculosis disease (Comstock, 1980, p. 449).

Contemporary sources also noted the role played by what we may term public health initiatives. A telling overview report for the League of Nations (Burnett, 1932) focused on additional aspects of prevention and treatment and brought together the importance of the role of medical and state agencies. Success in both respects, it was argued, derived from the existence and effective operation of medical agencies. Here

the decline of mortality was traced to the work of two agencies. First, there were dispensaries which traced cases, assisted patients' families, provided disinfection and acted as public education agents (Porter, 1999, 282) and whose essential agent was the visiting nurse. Second, there were patient institutions: namely the sanatoria. The sanatoria provided fresh air and rest for the infected, but more crucially isolated sources of infection, and thus attempted to curtail infection while providing some form of treatment, if not cure, for the ill (Burnet, 1932, pp. 531-2; Wilson, 1990, pp. 366-396). It was only after chemotherapeutic intervention became effective, that the role of the isolation hospital was reduced (Bulla, 1977, 39). The League of Nations report also noted that such work was complemented and improved by a political, administrative and social framework that comprised the State, voluntary associations and social insurance which supported those agencies (Porter, 1999, p. 282).

In the UK a series of legislative changes underpinned the role of state interventions. In 1909, the Government required notification of Poor Law cases of pulmonary tuberculosis and then in 1911 the notification of all hospital cases; from 1 January 1912, every medical practitioner was required to notify all cases of Pulmonary; non-pulmonary became notifiable in 1913(WRD7/6/3/282, p. 151 and 154). On 15th July 1911, the Sanatorium Benefit of the National Insurance Act of 1911 came into operation. This provided for construction of sanatoria (Lane, 2001, p. 143). This was soon followed by a general scheme for the provision of treatment in both sanatoria and dispensaries for all members of the community; the Government contributed one half of the costs of the maintenance of the sanatoria and the dispensaries (Kearns, 1995 p. 56; WRD7/6/3/282, p. 154). Responsibility for running the schemes was placed in the hands of County Councils and County Borough Councils.

In 1921 the Public Health (Tuberculosis) Act provided treatment for *all* even if they were not insured under the National Insurance Act of 1911 (Cherry, 1996, p. 55; Lane, 2001, p. 43). The 1921 Act required local authorities make adequate provision for sanatoria treatment of tuberculosis within the county or county borough boundary regardless of whether the patient was insured or not. Previously, coverage for insured and uninsured persons had been administered by the Insurance Committees where the fees paid by the insured guaranteed sanatorium treatment for the uninsured. Insurance committees were relieved of their mandate to provide treatment to the

tuberculous and responsibility was transferred to the local authorities financed from both the Exchequer and local rates (MH 55/142, Public Health (Tuberculosis) Bill 1921). Rates paid by the insured were reduced by the magnitude of the burden transferred to the local authority (MH 55/142, miscellaneous correspondence). If local councils failed in their legal duties to patients, the Minister for Health was permitted to make his (*sic*) own arrangements for the treatment of tuberculosis at the expense of the council. In the UK, the agencies involved in tuberculosis work included the health departments of local authorities, voluntary organisations and the Poor Law Institutions. This work highlights and draws our attention to the role of institutions particularly at the devolved local level – a factor we explore in this chapter.

Work on health in the interwar period, not surprisingly given the depressed economic conditions of the time, has largely concentrated on how and if unemployment impacted on health. If, as we are to believe from the literature, tuberculosis is associated with poverty, then one might expect the poverty resulting from unemployment would have led to some increase in the disease problem. The official view of the period was that pre-Welfare state provision in the form of benefits and school medical services meant that unemployment was not associated with adverse health effects. This view has recently found support from Congdon and Southall's (2004) analysis of infant mortality which found a tailing off of the relationship between infant mortality and unemployment in the 1930s. Welshman (1997) meanwhile has argued convincingly that local Medical Officers of Health campaigned and pursued vigorous policies to deal with infectious diseases, malnutrition and the effects of unemployment on health in the interwar years and by implication that the work of such Officers reduced the risk of any adverse effect on health of the prevailing poverty of these years. The optimistic view, in essence, argues that the provision of unemployment benefits together with public health reforms and improvements in preventative and interventionist medicine reduced the risks of any increase in the disease burden.

The 'negative' view, which conforms to the more radical stances of the period, suggests that health was a function of where one lived. The 'where one lived' aspect

provides an essential research angle of this research. In terms of the literature, early research by M'Gonigle and Kirby (1936), Morris and Titmuss (1944), and Titmuss (1938) argued for a positive link between unemployment and illness. The 'negative' view both of contemporaries and later observers has been on the localized variations in and hence implications of unemployment induced poverty and of the availability of public health as well as medical services. To a large extent contemporary work has tried to address the question of whether local authorities 'failed' in delivering their remit in the delivery of public health. Thus Harris (1995, p. 113) has reported that school medical service was much better developed in county boroughs and large urban areas than in county areas, although there were exceptions. In line with the localized argument, Webster (1982, 1985, 1994) has suggested that the health of women and children were adversely affected by the depressed economic conditions of the interwar years and that variations in child survival were a function of where a child lived. A major recent contribution to the literature on the importance of localized variations is the work of Powell (2005) who found that the distribution of doctors in the period was a reflection of affluence rather than need. His work examined the availability of doctors but did not, given its focus, assess the outcome of that provision. Congdon and Southall (2004) meanwhile have reported that differences in housing conditions and female employment patterns played a major role in explaining area variations in the risks of death particularly amongst infants. As we discuss below, whilst our research finds that there were indeed differences, some of the most deprived areas were the most successful in driving down the tuberculosis problem. It is explaining the how and why for these differences at a local level that forms the basis of this research.

In the recent past, a critique of the negative view of the role of local authorities and public health officials has emerged (Gorsky, 2008). A critical aspect of the critique is that the negative view has overly concentrated on urban areas and a 'fixation' with expenditure as the key area of concern. As Gorsky (2011) has convincingly argued, much of the recent work has been somewhat misplaced in its emphasis on municipal health expenditure (as recorded in official publications) both as a proxy for quality and effort in service delivery and as an indicator of local discretion in health policy. Whilst we agree that the variation in performance requires analysis, we concur with Gorsky (2011) that financial expenditure is not the most appropriate indicator. We

use outcomes (morbidity and mortality) and look at the provision of different health care policies. Equally we concur with Gorsky that an over dependence on the situation in county boroughs is not too helpful – hence our explicit decision to explore the variety of experiences both between and amongst rural and urban areas. Finally, we have explicitly embraced Gorsky's (2011) recommendation that health outcomes be assessed and explained in terms of a range of socio economic factors including housing and curative services which as he argues, better reflects the range of activities undertaken by health departments, and captures all the policy levers available to local officials seeking to raise population health. To that extent this research embraces those services which Gorsky has identified as being crucial to health outcomes.

Whilst Gorsky (2011) has stressed local authorities, other scholars have outlined and stressed the crucial role played by the pioneering individual. A recent paper on this by Neville (2012) has identified the important role played by councillors and entrepreneurs. This is another theme pursued in our research. Here we stress the views and actions (or lack of them) by local health officials and the interplay between those officials, local general practitioners and indeed the audit officials reviewing their performance by central government.

In other words, there is a substantial literature which argues that 'standard of living' factors explain respiratory tuberculosis morbidity (illness) and mortality (deaths). As such, the literature has identified the importance of overcrowding and nutrition as being significant in explaining both the long run decline and the short run increase during the First World War. To that extent we would expect to find that variations in morbidity and mortality in the period that we examine can be largely explained by differences in socio economic conditions. There were however significant medical interventions which could also help explain variations in morbidity and mortality. Interventionist medicine before the days of chemotherapeutic intervention played little part. Whilst there was a ceiling on what public health could achieve in terms of *curing* the disease at this time, preventative medicine was important in the work pursued by health professionals in terms of controlling infection and providing palliative care for the affected. Our question, therefore, is what light these insights

can shed on our understanding of respiratory tuberculosis in the interwar period against a background of devolved health decision making at the local level.

1.3 Sources and methodology

The source materials for the interwar years are rich and comprehensive. First, the annual returns of the Registrar General provide the essential information on respiratory tuberculosis death rates in all geographical areas of England and Wales. This information was used to compile data on respiratory tuberculosis morbidity and mortality. Second, returns to the Ministry of Health which detailed the activities of local authorities in terms of use of sanatoria, efficiency of diagnosis, use of Tuberculosis Officers and Nurses and the extent to which local authorities used prevailing technology for diagnosis, provide the essential quantitative information for an assessment of local authority health initiatives.

In addition, interwar source materials has been largely enriched as a result of the information that is derived from and is held by the National Archives relating to the Local Government Act of 1929 – the first real audit of health service provision at the local level in this country. The Local Government Act of 1929 abolished the guardians of the poor and transferred their responsibilities for poor law and registration to county councils and county borough councils. It also recognised the system of grants in aid and created the general grant (Butler and Sloman, 1980, p. 398). It further empowered the Minister of Health to withhold monetary grants from local authorities if they were not providing efficient health services. Because of the Act, an audit was taken of the work of local health authorities in England and Wales. Surveys were undertaken in order to satisfy the minister that the services were efficient and the results reported. The returns that resulted from this audit provide a rich, varied and comprehensive collection of quantitative and qualitative information at a detailed local level that enable us to evaluate the nature of and policy responses to the tuberculosis problem in England and Wales. The files from that survey exist in full at the National Archives. 1,084 files covering the period 1930 to 1943 detail the prevailing disease regimes in every County Council, Borough and Urban District

Council, Rural District Council and Metropolitan Borough in England and Wales. These files contain the detailed survey reports, survey appendices and survey correspondence. From our perspective, the key files relate to Appendix L which contains the detailed reports filed by each local area on tuberculosis. In addition and crucially, these local authority files were scrutinised by central government officers. As a result, the files include detailed audit reports made by officers from the Department of Health on every area and correspondence between the Department and the local authorities on given issues. The former are particularly invaluable in containing critiques of local authority policies and recommendations for improvement. The latter give an insight into how local authorities responded to central government recommendations.

The Census of Population conducted in 1931 provides comprehensive data on a variety of 'standard of living' barometers that have been used in our statistical analysis: as in, for example, overcrowding, population density, family size and (from 1931) unemployment. It further provides proxies for the provision of medical care in this period in terms of the number of doctors and nurses per 1,000 population.

Finally, the interwar years have detailed Medical Officer of Health reports at a local level. These reports detail not only the various local tuberculosis initiatives but also assess the social and environmental conditions of their areas. To this extent, we have used both the local archives as well as the audit files to investigate what individual authorities did – and with what success.

1.4 The Interwar Experience

Tables 1.1 and 1.2 present summary statistics on respiratory tuberculosis mortality in English and Welsh counties and towns in the interwar years.[3] We note first that both

[3] County boroughs are the large towns – that is towns with a population of more than 50,000. Administrative counties are the sum of the urban and rural districts in given counties (an administrative unit) where each urban district has a population of less than 50,000. In these terms, the administrative counties are less populated than the heavily populated county boroughs. Throughout this chapter we refer to the administrative counties as 'counties' and the county boroughs as 'towns'

towns and counties recorded a decrease in mortality over the period. In standard of living parlance, this indicates that morbidity and mortality from the disease fell – indicating an improvement in standards of living. Second, we note that *in general* mortality was more severe in towns rather than counties. Finally, we note that whilst the variance in mortality tended to decrease, it remained much higher in towns than in counties (Table 1.3). Again, in standards of living parlance, this indicates that peoples in some areas were less likely than others to experience the negative effects the disease created. As such, our research question is why were the risks of contracting and dying from this disease so much higher in some parts of the country? Is this another example of the standard of living school of thought or is there a role for public health initiatives?

One explanation for the variation centres on differences in standards of living, as evidenced by unemployment (Table 1.4) and overcrowding (Table 1.5). There are both positive and negative interpretations of the effects of unemployment on health in this period. Early research (M'Gonigle and Kirby (1936), Morris and Titmuss (1944), Singer (1937), and Titmuss (1938) argued for a positive link between unemployment and mortality. Recent work has questioned that interpretation. Welshman (1997) has argued that local Medical Officers of Health campaigned and pursued vigorous policies to deal with infectious diseases, malnutrition and the effects of unemployment on health in the interwar years and by implication that the work of such Officers reduced the risk of any adverse effect on health of the prevailing poverty of these years.

In terms of standards of living as captured by overcrowding, three definitions of overcrowding (Table 1.5) have been used. Three points follow. First, for all definitions and in both counties and towns, overcrowding fell between 1921 and 1931. Second, overcrowding for all definitions was worse in 1921 and 1931 in towns than in counties. Finally, there was significant variation amongst both towns and counties in overcrowding by all definitional standards in both 1921 and 1931. We hypothesise, following the standard of living literature outlined above, that if medical intervention had no role to play then the variation in tuberculosis death rates would be captured mainly by differences in standards of living.

A second explanation centres on variations in the availability of medical care. We hypothesise, following the public health literature outlined above, that if medical intervention had a role to play then the variation in tuberculosis morbidity and death rates would be captured by variations in public health initiatives at the local level. Here we explore different definitions of such initiatives.

Care can be separated into three policy areas: The first area was residential care for those diagnosed with the illness. In 1911 there were 84 sanatoria in Britain providing about 8,000 beds; by 1930 there were 500 sanatoria providing 25,000 beds for patients (Lane, 2001, p. 143). We find that in the interwar years, there were significant differences both between and amongst county councils and county boroughs and metropolitan boroughs in their use of residential care as evidenced in the high levels of variation (standard deviation) between local authorities at this time in four barometers of residential care (Table 1.6).

The second area focused on community based activities, often at or in connection with the dispensaries. These activities included diagnosis and identification of those ill or in contact with the infected (and means of diagnosis), co-operation between tuberculosis officers and general practitioners and care offered to individuals at home. Again (Table 1.7) we find significant variation between local authorities in these terms.

The third area was concerned with the efficiency of notification of the disease. In the interwar period, as now, central government was concerned with measures of efficiency. The barometers used at this time, were the percentage of all persons on the dispensary register on 31st December whose diagnosis was not completed and the percentage of new cases and contacts whose diagnosis was completed within three months of first examination.

Again, (Table 1.8) we find variation between and amongst county councils, county boroughs and metropolitan boroughs. The variation for diagnoses within three months is however very small. What strikes the modern reader most forcibly however is how quickly, overall, diagnoses were made. Most, but not all, areas were making returns which demonstrated that over 90 per cent of patients were diagnosed

within three months of their first examination. The key rider here is of course whether, as in the West Riding of Yorkshire, haste in diagnosis led to incorrect diagnoses (MH66/292, p. 12).

Variation in efficiency of notification and in care outside the dispensaries has a further application routed in public perception of the disease being associated with poverty. Thus, it was noted in the case of the East Riding of Yorkshire that 'there appears to be more than the usual amount of local dislike to being labelled as tuberculous and possibly the practitioners are influenced by this feeling; similarly I was told that the tuberculosis nurses' visits are not popular as the house visited then becomes a marked one in the village' (MH66/278, p. 45).

A final explanation, nutrition, combines both living standards and medical intervention in terms of the interventions made by some authorities to provide nutritional assistance to those at risk and those already ill. Comprehensive and reliable data on nutrition over geographical areas are not available for the interwar years. Boyd Orr (1936, p. 21) argued that the poorest 10 per cent of the population consisted in the main of families in which there was a disproportionate number of children or other dependants per earner. The disproportionate number he classified as having four or more children or other dependants per earner; such families were defined as being 'Group 1' families who, he argued, were most likely to suffer from malnutrition. Average family size thus *could* be a proxy for nutrition.

To summarise, we find significant differences amongst and between areas in terms of their ability to deal with pulmonary tuberculosis. We also find significant variation for a range of explanatory variables. In the next section we subject the explanatory variables to statistical testing.

1.5 Explaining differences in morbidity and mortality

1.5.1 Morbidity

Our interest is in the regional variation in the burden of tuberculosis mortality in the context of general decline. For this purpose cross sectional data by metropolitan borough, town and county administrative regions are exploited to detect explanatory factors in the interwar period using selected available data from the period 1921 - 1931. Regressors for any particular region were categorised into three general (and partially overlapping) groups: living conditions and socio-economic indicators and medical provision. The null is that none of the above socio-economic factors had any impact on the burden of tuberculosis in England in the interwar period.

To estimate the effect of various socio-economic and medical variables on the case rate in cross section the model is first estimated by OLS as the standard linear model

$$Crude\ Case\ Rate\ = \boldsymbol{Xb} + \boldsymbol{e}$$

where X is the information matrix consisting of a constant (reporting of which is suppressed) and the regressors, $\boldsymbol{b}$ is a vector of parameters to be estimated and $\boldsymbol{e}$ is the disturbance. Contained in X are DENSITY (persons per acre), THREEPRM (no. of private families per 1000 living with more than 1.5 persons per room), FAMSIZE (average no. persons per family), MALEUNEM (per cent of male labour force 'out of work'), DOCTORS (doctors per 1000 population), SNURSES (sick nurses per 1000 population), MWIVES (midwives per 1000 population), XRAY (no. of x-ray exams per 100 'new cases' and 'contacts examined), SPUTUM (no, of sputum exams per 100 'new cases and 'contacts examined'), BEDS (average number of beds available per 100 tuberculosis deaths), TREATED (total no. patients treated (i.e. excluding observation) per 100 tuberculosis deaths), DIAG (per cent of new cases whose diagnosis was complete in under 3 months since first examination), NODIAG (per cent of all persons on the dispensary register whose diagnosis was not complete), COMP (no. of cases on the dispensary register per 100 on the notification register), CONSULT (no. of consultations at homes per 100 deaths from tuberculosis) and HOMEVISIT (no. of other home visits by tuberculosis officers per 100 deaths from tuberculosis). Crude case rates per million population (CCR) are constructed from both M. O. H. notification register and Registrar General's Statistical Review. The results of this analysis are given in Table 1.9.

Although the mean variance inflation factor (VIF) in specification 5 is only 1.98, the value for male unemployment is much higher at 3.86 in indicating multicollinearity. As with many economic variables pertaining to the standard of living, high correlation tends to suppress their significance. Ramsey's reset tests confirm that non-linear relationships between the existing parameters are insignificant for all specifications. In addition, variances are not confirmed as heteroskedastic. The values of the coefficients are as expected. The analysis suggests that the chances of developing respiratory tuberculosis increases with both overcrowding and family size and decrease sharply if the community is rural.

1.5.2 Mortality

Here we entertain the possibility that a better model of the relationship between the crude death rate and the explanatory variables contains continuous by continuous interactions of some of the dependent variables. We test the proposition that marginal effect of medical administration on death rates is greater when primary services are already in operation. We choose sick nurses per 1000 population as the conditional primary service variable. The model takes the following standard linear form as is estimated via ROLS (robust ordinary least squares) and QR (quantile regression).

$$Crude\ Death\ Rate\ = \boldsymbol{Xb} + \boldsymbol{e}$$

where the information matrix now contains SNURSESCOMP, SNURSESCONS and SNURSESHOME – interactions of the aforementioned variables. Specification ROLS 1 estimates the model using concurrent living conditions data. However, our preferred method is to use lagged values for population density and household overcrowding to accommodate for the observation that development of symptoms often follows exposure by several months and sometimes years.

Sample errors are heteroskedastic. Initially an OLS model was estimated using White's heteroskedastic consistent estimator for the standard error. Inspection of the residuals versus fitted regression values, however, revealed some outliers which may exert undue influence over the values of the coefficients. As a precaution against

misdiagnosis and against invalid standard errors arising from non-normality a Shapiro-Wilk W test for normal data was performed for the benchmark OLS specification (not reported). A p-value of 0.61 is not sufficient to reject the null hypothesis that the errors are normally distributed. Therefore, we proceed, by comparing existing estimates with techniques robust to outliers. Robust regressions ROLS re-weights observations according to their Cook's distance in order to accommodate observations with large residuals or high leverage. Our findings are reported in Table 1.10.

ROLS permits changes in the values of both the coefficients and standard errors in contrast to the Huber-White sandwich estimator for the standard error in OLS. The FAMSIZE coefficient is marginally significant in most specifications. In addition both SNURSES and the interaction with COMP are not significant when re-weighted using ROLS. An alternative to OLS estimation in the presence of outliers is the median regression. The least absolute deviation estimator is a special case of the quantile regression where the effects of the regressors are on the conditional median of the crude death rate from respiratory tuberculosis rather than the conditional mean. Since the objective function is no longer to minimise squared deviations from the mean, but absolute deviations from the median of the response variable, outliers cease to have disproportionate effects on the estimates (Table 1.11).

Quantile regression conditioned at the median of the response variable reveals little change in the fundamental results from ROLS. However, quantile regression offers the opportunity to condition on any level of the response variable and track effects of the regressors across the range. The effect of the independent variables may vary across the values of the dependent variable. A model is constructed to assess whether the values of the 'coefficients' change when the weighting Q changes between 0.25 and 0.75, where 0.5 coincides with the median of the crude death rate from respiratory tuberculosis. The following simultaneous quantile regression model uses bootstrapping to estimate standard errors so that confidence intervals may be constructed to compare coefficients across quartiles. Some hitherto insignificant variables have been omitted (Table 1.12).

The most notable result of this exercise is the changing coefficient on the number of doctors per 1000 population. One interpretation could be that when the death rate is high the primary factors determining the death rate are standard of living variables and whether a community is urban or rural. But when the crude death rate is lower, medical intervention is required at the margin to reduce death rates further. An alternative speculation is that doctors are present in greater numbers in areas that have lower death rates because these areas are higher income. Sick nurses per 1000 population is a similar variable but it remains insignificant across quartiles of the crude death rate.

The effect of family size is greatest when crude death rate is highest. Some authors have suggested that family size is a proxy for nutrition. If this is true and the proxy is effective it would suggest that larger families do indeed explain higher death rates in areas with substantial burden of disease. Otherwise, family size is not effective in explaining variation in death rates.

1.5.3 Fatality

Thus far no correlation between medical interventions and the burden of tuberculosis in interwar Britain has been observed. Though standards of living differences are important in explaining the likelihood of both contracting and dying from tuberculosis infection, medical interventions may affect the likelihood that once disease is contracted, that it will result in death. As such, the crucial and all important intervention by the health services may have been averting death.

Four variables previously expressed in terms of deaths are transformed here and expressed in terms of cases; BEDSCASE, (no. beds available per 100 cases of respiratory tuberculosis), TREATEDCASE, (no. patients treated per 100 tuberculosis cases), CONSULTCASE, (no. consultations at homes per 100 cases of tuberculosis) and HOMEVISITCASE (no. of other home visits by tuberculosis officers per 100 cases)

The following model was estimated;

$$Case\ fatality\ ratio\ = \boldsymbol{Xb} + \boldsymbol{e}$$

When estimated however, the errors were severely heteroskedastic. A residuals against fitted values plot of the above case fatality ratio model revealed a possible source (Figure 1.2).

The outlier in the top right is responsible for some of the bias in the estimates and for the reporting of heteroskedasticity in the variances. In addition to this particular observation a number of anomalies exist in the case rate data supplied from the Registrar General's Annual Review. As a result the calculation of the case fatality rate is also affected.

Therefore, we constructed a new fatality series. The new case fatality series uses crude death rate data as above. The crude death rate is now divided by case rate data based on the returns relating to cases of tuberculosis on the Ministry of Health's (M. O. H.) notification register rather than the Registrar General's Annual Review to give the case fatality ratio. This process produces a better behaved series. Figure 1.3 is the new residuals versus fitted plot for OLS (22) using the M. O. H. case series.

Some surprising results are obtained in table 1.13. The positive coefficient on BEDSCASE and TREATEDCASE suggests that medical interventions are chasing higher fatality rates rather than remedying them. The results suggest also that outcomes are consistent across social strata. Standard of living variables are not important in explaining differences in case fatality rates. The comprehensiveness of the tuberculosis program also had slight beneficial effects. Regions with a greater proportion of patients on the dispensary register fared better.

1.6 Policy interventions: decentralised health decisions in the interwar period

In this section we review variations in policy as implemented by local authorities. We draw heavily on the 1929 audit files to identify issues relating to the policies in different areas. We discuss policies in terms of identifying, 'curing' and preventing the disease. We first identified mortality rates over time differentiating between county boroughs, council councils and metropolitan boroughs. Table 1.14 presents the key summary statistics on respiratory tuberculosis mortality in English and Welsh counties and towns in the interwar years. County boroughs are the large towns – that is towns with a population of more than 50,000. Administrative counties are the sum of the urban and rural districts in given counties (an administrative unit) where each urban district has a population of less than 50,000. In these terms, the administrative counties are less populated than the heavily populated county boroughs.

We note first that both towns and counties recorded a decrease in mortality over the period. Second, we note that *in general* mortality was more severe in towns rather than counties. Finally, we note that whilst the variance (standard deviation) in mortality tended to decrease, it remained much higher in towns than in counties.

The results provided the motivation for this section. We wanted to find an answer to why there were such differences. Why then were the risks of contracting and dying from this disease so much higher in some parts of the country?

1.6.1 Identifying the disease

In terms of identifying the disease, two tools were available: sputum tests and the X-ray. Hence we begin by exploring variations among and problems within administrative areas in the identification of the disease as identified by the 1929 auditors.

Diagnoses via the sputum test varied enormously both within and between county boroughs, county councils and the metropolitan areas (Table 1.15). The evidence suggests that sputum tests were less comprehensively carried out in the county council areas. Prima facie, the evidence would suggest that county areas were less

likely to carry out sputum tests.

Diagnoses via sputum examination were significantly more popular among both doctors and patients. Sputum samples could be undertaken by tuberculosis dispensaries. This meant, for doctors and patients, the tests could be taken in a local dispensary. Nevertheless, although the sputum test was relatively easy and cheap to take, there were still problems. First, the tests had to be examined in order to determine whether the patient had tuberculosis or otherwise – and this needed laboratory facilities. Counties, especially around London, often lacked the necessary laboratories to determine tuberculosis infection, but they could avoid the cost of installing their own facilities by sending samples to London for examination. Second, sputum examinations were only effective in identifying those patients with active symptoms of respiratory tuberculosis. Tubercular lesions were required to have penetrated the bronchioles in order for a sputum test to return a positive result.

Dispensaries however were not without their problems. There were demand side issues in relation to how individuals viewed and accessed their services. One such disincentive was the cost of travel. This was a particular issue in rural areas. The costs of transportation meant that the number of cases treated was lower because rural workers were unwilling to leave their homes. A recurrent theme of the audit reports is the inaccessibility of dispensaries in the county areas. Thus, in Northumberland, it was noted that whilst the 'system of dealing with patients at the dispensaries is suitable for the south eastern area, where the bulk of the population resides, home visiting appears to be the only way of dealing effectively with the scattered population in the northern part of the county' (MH66/183, p. 6). It also, however, reflected another factor in the reluctance on the part of the public to travel either to dispensaries or to residential institutions: a reluctance born out of an acknowledgement and understanding on the part of the public that diagnosis did not at this time lead to cure.

Often patients would not return to dispensaries for three or six month check-ups once infection had been identified. Though the problem of achieving patient compliance with a prescribed health regimen persisted into the chemotherapeutic era, patients more acutely lacked incentives to pursue their own health before the 1950s. This was

identified by the auditors as a particular problem in the City of Southampton (MH66/219, p.5). Dr. Johnston of the Heatherwood Hospital, Ascot, reported that only about 45 per cent of cases applied for treatment at the hospital within one year of the appearance of the disease. Despite the problems with the sputum tests, many doctors preferred sputum examinations because of the poor resolution and quality of images produced by contemporary X-ray apparatus. But X-ray facilities could reveal tubercular lesions anywhere in the body, and persons with tubercular lesions within the lung in the early stages of the disease could be placed in sanatoria as a means of preventing the spread of infection. Hence the X-ray was not infallible but had the advantage of being able to identify the disease earlier than the sputum test.

As with sputum tests, so with X-ray; we find significant differences between and among administrative areas (Table 3.15). The problem with diagnosis with X-ray, as the audit and local files reveal, was that it was expensive, not always reliable and was not easily accessible to patients. To that extent, the experiences of interwar England and Wales, mirror the problems with the use of X-ray in the developing world between 1950 and 1980 (see chapter 2). In 1930, for example, the cost of X-ray apparatus in Pontefract in the West Riding cost £525 (WRD/6/61, p. 108). This was a problem in areas where financial resources were limited and there were constraints on capital expenditure – as the Ministry of Health noted in its report on tuberculosis in Wales in 1939 (Ministry of Health, 1939). West Hartlepool for example was criticised by the auditors for having no X-ray apparatus at all at its Tuberculosis Dispensary 'which militates against the efficiency of the Council's scheme' (MH66/1079, p. 2). In some areas, the problem was the age of the equipment which made it inefficient for the purposes of identification (WRD7/6/3/10, p. 114).

There were also demand constraints on the use of X-ray. We have already noted the reservations many doctors had as to its efficacy. There were also constraints however in terms of the abilities of peoples to access such facilities. In many rural areas, the cost of transport for the individual to the nearest X-ray facility was high. In Northumberland, provision for X-ray examination only existed at the 'inaccessible' Wooley Sanatorium (MH66/183, p.4). In the North Riding of Yorkshire, X-ray examinations were only available at Scarborough and Middlesbrough. In East

Sussex, patients had to travel up to thirty miles to Shoreham in order to be examined. It was not just the cost of transport, of course. In Nottinghamshire, the auditors described the relatively low number of X-ray examinations carried out as being 'due to the inaccessibility of the X-ray plant' (MH66/188, p. 4). It was also the cost in terms of lost income if the individual had to take time off work. Such was the extent of the problem that clinicians at Darvell Hall sanatorium pre-empted initiatives in the developing world from 1950 (see chapter 2), by suggesting that the difficulty might be overcome by the provision of a mobile X-ray unit, similar to those used in areas of the New World with sparse populations. Such units, however, required transportation – and the cost of fuel.

In addition to the cost and difficulty of transportation, the price required to use X-ray facilities in some counties was great. This constituted another and for some individuals an overriding and binding constraint. Here the audit files reveal variations in not just the price – but also who was responsible for the fee. In Oxfordshire, for example, patients were required to pay a fee of one guinea for an examination of the chest. As the audit files reveal, not surprisingly, X-ray uptake was, therefore, low with only three examinations per hundred cases against a county council average of twenty. A similar situation pertained in Yorkshire where patients were required to pay a fee that was, according to the auditors, 'somewhat expensive.' The majority of work was done at Scarborough by a private radiologist on a fee paying basis which made the scheme 'somewhat expensive' … 'the result is that the number of X-ray examinations per hundred new cases and contacts is so low as to be almost negligible' (MH66/282, 27). A solution could have been to use local hospitals: however the audit found that 'the local hospitals refuse to make arrangements for the examination of county cases in their X-ray Departments' (MH66/282, p. 28).

Finally, as with HIV/AIDS, where there was no guaranteed cure for the disease, individuals were reluctant to be diagnosed as having tuberculosis. The absence of a cure is one facet explaining reluctance for diagnosis. The other is the social stigma the disease carried. A further observation, as noted by the Medical Officer of Health for the North Riding of Yorkshire, was an attitude prevalent amongst adolescent female and the young married that ailments were seen as 'in the natural order of

things' such that 'the young married woman appears disinclined to seek early advice, it is only when she is unable to carry on her work in the house that she is persuaded to submit herself for examination' (MH66/286, p. 32).

1.6.2 'Curing' the disease

In the decades before the introduction of chemotherapeutic interventions in the 1950s, treatment of tuberculous patients in Britain comprised stays in regional sanatoria. Some sanatoria offered 'light treatment', but the majority of centres were used to facilitate natural convalescence rather than offer curative treatment. They were also, and crucially, seen as a way of isolating an infected person from close family members – notably in homes where overcrowding was such that the infected did not have use of a separate bedroom. Again, we find significant differences in the extent to which different authorities used the sanatoria (Table 1.16).

The problems with the sanatoria, as identified by the auditors, were several-fold. In some parts of the country sanatorium accommodation was described as being woefully lacking. West Hartlepool came in for particular criticism from the auditors in this respect. The residential accommodation provided by the authority was deemed to be inadequate because the only beds the authority provided were for cases of intermediate and advanced pulmonary tuberculosis needing hospital treatment and these were on the balconies and not wards (MH66/1079, p.2). A second revealed problem was that in some parts of the country, only those patients who were in the advanced stages of the disease were admitted. The high fatality rates in the West Riding of Yorkshire were attributed to the policy of admitting only advanced stage tuberculosis patients to sanatoria 'among whom no reasonable possibility of cure could be expected' (MH66/292, p. 8). The problem was not just one of policy decisions: in some parts of the country, the admission of late stage cases to the sanatoria reflected a lack of beds. Derbyshire was singled out for criticism because, even intermediate and advanced cases patients had to wait a long time for admission given the shortage of beds – this a result of the 'great scarcity of beds' (Interview

with Area Tuberculosis Officer, 19th May 1937, MH66/55, p.1).

In marked contrast, Liverpool was described as being ‘probably unique among English cities, in the number of beds available for tuberculosis. 'It is true that there is a high incidence of tuberculosis but the rate (one bed per 780 of the population) is hardly sufficient to justify this lavish supply of beds’ (MH66/489, p. 79). As such, the auditors requested that the Council considered ‘the possibility of reducing the number of beds occupied, particularly in view of the depressed financial state of Liverpool’ (MH66/489, p. 79). The suggestion of reducing the number of admissions was not welcomed by the three dispensary tuberculosis officers who ‘were obviously a little diffident to change what had for years been the accepted policy of the department namely to send away every patient who wanted to go, not only once but as many times as he wanted to go’ (MH66/489, p. 80).

The above might indicate that the problem was a lack of beds and/or a mistaken priority in only admitting those with advanced stage tuberculosis. From a demand perspective, people had to be persuaded to enter a sanatorium. For some households, that meant a lack of income (assuming that the infected was able to work). In Manchester, a scheme was introduced to overcome this constraint ‘the power of the purse gives a useful hold over some patients as, for example, by encouraging “a doubter” to enter the Sanatorium by making a grant to his family’ (MH66/742, p. 75). Manchester, however, was unique in this respect.

The other side of the problem was the growing doubts as to the efficacy and financial return on sanatoria as a mode of treatment. In the interwar years there was much debate as to the wisdom of residential care expressed both by central government and by local officials. In 1930, the Ministry of Health beseeched tuberculosis authorities to ‘consider carefully whether the results in their area by residential treatment offer the best possible return for the expenditure incurred’ (Ministry of Health, 1930, MH55/131, p.1). Then, as now, the emphasis was the best allocation of scarce resources to achieve the optimal outcomes. Part of the concern related to cure rates. Increasingly there was a feeling in some quarters that residential care was best provided to those cases which were most likely to respond to treatment – that is those classed as Group 1 (early stages).

1.6.3 Non-residential care

> 'It can be definitely asserted that if the tuberculosis problem is to be tackled at the root, then a much greater amount of attention must be paid to the environment of the individual. Where in fact there is a known focus of infection – in a house, or in a small community – it is only by constant and careful supervision of the contacts that real preventative work will be obtained and so enable a case to be diagnosed and receive sanatorium treatment early' (MH66/292, p. 12).

Thus wrote the Ministry of Health in its report on West Yorkshire: it is a claim which sums up the role of the dispensaries in delivering the all-important 'care in the community'. Kent was held up as a county which had an excellent care scheme – and was used by the Ministry of Health as a reference point for how to get things right for other counties (MH66/137, p. 8).

A key role of the dispensaries was not only to identify the ill but also to organise home visits to the infected and/or in contact with the infected by Tuberculosis Officers and nurses. Again, we find considerable variation between and amongst county councils, county boroughs and metropolitan boroughs (Table 1.17). To some extent, the emphasis on the home visit reflected increasing doubt as to the efficacy of residential care. In rural areas, where the population lived some way away from the dispensaries, it was the only real way to contact the ill and/or potentially infected. The reach of the 'care in the community' activities, it should be noted was extended in the 1930s given the progress of motor transport which facilitated home visiting even to the most remote area (MH66/292, p. 13).

For many areas non-residential care was criticised by auditors on three main grounds. Firstly, a common criticism of counties was the reach of their programmes. Thus in Essex, the quality of work done in the more remote and rural parts of the County were described as 'distinctly poor' (Report by D J Williamson, 11th March,

1931, MH 66/85, p.3). The criticism, we note, was not accompanied by any advice: 'it is difficult to see how this can be rectified' (Report by D J Williamson, 11th March, 1931, MH 66/85, p.3). Nottinghamshire (MH 66/188, p. 5) and the East Riding (MH 66/280, p. 1) attracted similar criticisms. It was noted that in Nottinghamshire 'the Tuberculosis Officers may not have time to develop contact examination, consultations and perhaps home visits as fully as may be desirable' (MH66/188, p. 4). The auditors noted in relation to the East Riding that the two dispensaries 'cannot adequately serve the patients in this sparsely populated district'. It was suggested that the number of tuberculous people living in areas remote from dispensaries were not dealt with adequately and the number of medical staff were insufficient to carry out the necessary work of visiting patients in remote areas and of getting in touch with their doctors; this led to a 'subnormal' amount of home visiting' the result being the contact with the infected was 'particularly poor' (MH66/280, p. 2 and 4).

Criticisms were also levelled at policy direction. In Essex, the scheme was criticised on 'the score of frequent chops and changes, and the lack of a far sighted settled policy' (Report by D J Williamson, 11th March, 1931, MH66/85, p.4). Leicestershire County Council meanwhile was criticised both for the low number of tuberculosis visits and the fact that 'tuberculosis visiting was not altogether adequately controlled by the T.O.' (MH66/137, p. 2). Home visiting by Tuberculosis Officers was also identified by the Ministry of Health to be a 'weak feature' of Middlesex's tuberculosis scheme (MH66/169, p. 6).

The problem however was not just the quality of the after care service – it was also whether it existed at all. Worcestershire was singled out because 'there are no systematic arrangements for the after-care of persons who have suffered from tuberculosis'. As the Ministry of Health robustly told the Council, it 'will appreciate that an efficient system of after-care is one of the most valuable features of the tuberculosis service as a means of preventing the spread of infection and consolidating the effects of treatment in individual patients' (letter to Town Clerk, Worcester, 9 November 1931, MH66/1079, p. 2). The East Riding of Yorkshire was criticised for a 'subnormal' amount of home visiting' the result being the contact with the infected was 'particularly poor' (MH66/280, p. 2). This was a particular

problem in rural areas in that county: ‘tuberculous people living remote from dispensaries are inadequately dealt with (MH66/280, p. 4). Nottinghamshire thus attracted similar comments to the effect that ‘the Tuberculosis Officers may not have time to develop contact examination, consultations and perhaps home visits as fully as may be desirable’ (MH66/188, p. 4).

In reply to the above criticisms, however, medical officials, doctors and members of voluntary organisations argued that the problem of late diagnosis was one of education rather than one of incentives. But here the audit files reveal a woeful absence of educational activities which might have persuaded members of the public to use these facilities. Typically, we find that individual counties provided very little in the way of propaganda concerning tuberculosis for their constituents. The National Association for the Prevention of Tuberculosis (NAPT) argued that in spite of many years of propaganda and working with communities, patients were often only seeking medical help when the disease was in an advanced stage. The evidence from the county reports is clear; few educational sessions were being held, in most counties, if any. However, even in cases where dispensaries offered free public lectures, attendance was poor. County boroughs did not always fare much better. Liverpool’s Medical Officer of Health, for example, had ‘for some unknown reason … set his face firmly against anything in the nature of health propaganda. Even posters in the dispensaries were forbidden’ (MH66/489, p. 67).

It was also argued that the problem was not one of education but rather the ‘cavalier’ attitudes of the patients. A major part of the propaganda campaign involved reminding the ill that treatment was long course, strict exercise and diet regimens had to be followed and obtaining permanent arrest of the disease was uncertain (MH55/139, p.28). In addition, those in receipt of sanatorium benefit were duly sent to workshops when their condition permitted and the case was made that patients ought to be subsidised to make their employment economically feasible (MH 55/130, pp. 38-39). In many cases, patients were not forthcoming because the probability of cure was low and the process to achieve it difficult. Health officials repeatedly berated the public for not coming forward at the earliest signs of tuberculosis. More than half of patients only sought professional attention once the disease was in an advanced stage.

In the West Riding, the problem was also the result of perceived failures in the non-residential care in the County. Several issues were identified: the small proportion of contacts in families of infectious cases who were examined, the small proportion of sputum examinations to total notified cases and the low number of visits to homes by the Tuberculosis Officers (MH66/292, p. 12). Here, a comparison made by the Ministry of Health's inspector between Lancashire and the West Riding is revealing. It was noted, inter alia, that 'The Lancashire Tuberculosis Officers examine a larger proportion of new cases than the West Riding Tuberculosis Officers. They also hold more consultations and pay more home visits. The homes of patients are kept under closer supervision in Lancashire than in the West Riding' (MH66/292, p. 13). The Inspector further noted a greater competence to diagnose in Lancashire which he attributed to 'the increased facilities, greater number of officers, and more co-ordinated scheme which Lancashire has at their disposal' (MH66/292, p. 13). By contrast, Leicestershire was singled out for praise in its high number of visits made to the home of patients by the Tuberculosis Officer (MH66/137, p.3).

The importance of the link between residential and non-residential care is best described by the cases of Sheffield and the West Riding of Yorkshire. We have already noted that the West Riding failed to admit sputum positive individuals until any chance of cure was very limited. The question of why relates to the non-residential activities of this authority. 'The main conclusion arrived at is that the present staff is inadequate numerically to cope efficiently with the amount of work, and the direct results of this inadequacy are reflected in hasty diagnosis, injudicious admission of patients to sanatorium, inability to visit homes frequently enough, and consequent omission of the essential duty of examination and supervision of contacts' (MH66/292, p. 12). Sheffield was praised for its success in driving down mortality rates. The success was attributed to its work in five areas. There was 'complete co-operation' on the part of the general practitioners in the Tuberculosis Scheme. The City provided adequate sanatorium and hospital accommodation for all citizens suffering from Tuberculosis or even suspected to be suffering from the disease. The City prioritised control of infection as in the treatment of a large number of early cases of tuberculosis discovered by means of the examination of contacts of notified cases and intensive work with regard to the isolation of infectious cases of

tuberculosis either in Hospital or in their homes. The City also provided a housing scheme for infectious cases of tuberculosis living under such conditions that isolation was impossible (MH66/872, p. 18). In other words, in the views of the auditors, institutional liaison and co-operation were crucial as in the totality of various measures for dealing with the 'tuberculosis problem'.

1.6.4 Preventing the disease

Modern day thinking argues that respiratory tuberculosis spreads in environments where peoples live and work closely together. This work confirms the work of those historians who have detailed how tuberculosis spread amongst the growing urban populations at a time of industrialisation – and those who noted how in both the first and second world wars, infection and death rates soared as peoples worked closely in factories (Barnes, 1995; Bates, 1992; Bryder, 1988; Burnet, 1932, pp. 502-510; Daniel, 1997, pp. 22-27; Dormandy, 1999; Dubos R & J, 1952; Johnston, 2003, p. 339 Ott, 1996; Rothman, 1994; Smith, 1988; Teller, 1985).

Contemporaries were well aware of the association. It was noted at the time by many clinicians that tuberculosis infection often occurred concurrently in patients whose standard of living was poor. In 1924, the Tuberculosis Officer for York argued that the returns on tuberculosis initiatives 'give but a poor return for the amount of money expended' given the 'unhygienic environments in which so considerable a portion of the community live' (WRD7/6/3/361, p. 6). Similar sentiments were echoed on July 1st 1926 in a conference of the National Association for the Prevention of Tuberculosis (NAPT). Institutions in Glasgow, it was claimed, required greater resources to reduce waiting list times. This provoked a surgeon of the Glasgow corporation hospital and sanatoria to argue that it would be better to '[improve] housing conditions' in general with 'provision of plenty of air and sunshine', in order to reduce the costs of the institution.

The call for improvements in housing conditions reflected the common view that overcrowding created the conditions in which the disease spread. Hence many Medical Officers of Health throughout the interwar years stressed the importance of

relieving overcrowding. Overcrowding in their view related not only to the number of rooms and the space available but also when the accommodation did not permit 'of the occupation of a separate room by a member of the family suffering from Tuberculosis' (Ministry of Health, 1939, p. 35). In some areas, preference was given to tuberculosis patients in the allotment of council houses – as in Liverpool (MH66/489, p. 67). In Sheffield, an important facet of the tuberculosis work was the rehousing scheme for infectious cases of tuberculosis 'living under such conditions that isolation is impossible' (MH66/872, p. 18). In Leicester, the Ministry of Health noted that the City had 'developed housing estates in the county area where it is understood the City rehouse a fair number of tuberculous patients' (MH66/137, p. 4). The downside in the case of Leicester was that the tuberculous patients then became chargeable to the County of Leicestershire … 'the general effect of this action of the City upon the county arrangements may be appreciable' (MH66/137, p.4).

There were however significant and important exceptions. In Manchester, a totally different approach was pursued. In Manchester an overt negative class approach dominated. The assumption was that this was a disease only of the poor: 'the houses of the well-to-do are, of course not visited' (MH66/742, p. 77). The senior Tuberculosis Officer in Manchester took the view that 'he does not see why they (people with tuberculosis) should have preference, for example in such matters as obtaining houses in competition with the healthy and, therefore, biologically more worthy persons, and he has frankly advised his Committee so' (MH66/742, p. 86). But then the reports do suggest a disturbingly negative view of those infected with tuberculosis in Manchester by this high ranking official. The auditors noted his view that 'the mass of persons with chronic pulmonary tuberculosis he regards as a reservoir of infection to the healthy, a charge upon them, and biologically of less value' (MH66/748, p. 86).

Dr. Coles of Oxford General Infirmary was prescient: he was 'sceptical of the value of administrative measures, and believes that the decline in tuberculosis can be attributed more to an increase in wages and improved social conditions than to active preventive measures.' It is evident from reports to the Ministry of Health that local medical professionals frequently suggested a better use of resources could be attained by treating tuberculous patients at home or in isolated sanatoria. To that

extent, the role of the sanatoria was in part a way of isolating the infected from other family members (Ministry of Health, 1939, pp. 35 and 40-1).

Modern day thinking is that amongst those infected, only 10 to 15 per cent of peoples show active symptoms of the disease (Borgdorff et al., 2002, p. 217; Comstock, 1980, p. 449; Johnston, 2003, 337). The argument is that malnutrition does not explain infection, but it does explain why some individuals go on to display active symptoms of the disease (Besanco, 1945, p. 287; Comstock, 1980, p. 449; Smallman-Raynor and Cliff, 2004, p. 647). Qualitative information suggests that Tuberculosis Officers and Medical Officers of Health in England and Wales pre-empted modern day thinking in their views on nutrition. For many such health professionals, the key to combating the disease lay in this area and in the reallocation of resources away from sanatoria to the delivery of food support to the infected and those at risk of infection. Thus the Medical Officer of Health for the North Riding argued that 'An adequate dietary, by that is meant a nourishing one, not judged by the cost but by the character of the food provided, is the best safeguard against the disease' (MH66/286, p. 33).

As early as 1920, some local authorities were providing nutritional support – in the form of eggs and milk – to insured persons, but there was no general consensus that local authorities should be provided with Exchequer assistance. At this time if local authorities wished to provide assistance to uninsured persons they were to provide this in liaison with their relevant Board of Guardians under the New Poor Law. By 1921, discussion was under way as to the best way of providing extra nourishment in necessitous cases in major urban centres in Britain. The debate concerned the appropriate direction of the resources of Insurance Committees centred on the issue of whether tuberculous persons in a situation of want and who were already receiving Poor Law assistance would also be eligible for food assistance.

The solution was sought experimentally. In Bristol, for example, food was given in necessitous cases only for those who were in a state of poverty but who were not, for whatever reason, receiving assistance from the guardians. Manchester's system made nourishment available in kind rather than in money, but only for a maximum period of a month. The goods supplied included vegetables, meat, eggs, milk and butter

(MH66/742, p. 75). In Liverpool, New Liberal ideas had permeated such that the Insurance Committee was willing to provide assistance irrespective of the applicant's circumstances or physical condition. The Liverpool Insurance Committee found within its remit a duty to those in need, even if the assistant nourishment ran the risk of being disseminated within the family or sold on at a profit by the recipient. By the reasoning that tuberculosis was a disease that resulted from poverty it was not necessary in any case for the patient to actually consume the additional food, since any improvement in the standard of living of the individual, whether through consumption of the food itself or by consumption of articles derived from the sale of food assistance, was beneficial (MH55/136/, p.1).

Radically different approaches were tested across different urban centres. In smaller towns such as Middlesbrough, voluntary charity (the Care Committee), insurance firms and the Poor Law Authority were more easily able to work jointly to provide relief based on individual informal appraisals of a patient's situation. Anybody with necessitous circumstances was eligible to receive food. By contrast, in Birmingham, patients who were not expected to recover from the disease and were unlikely to return to health were not considered for benefit at all by the local Insurance Committee. For those unfit for regular work, grants of food were regarded as an 'uneconomical expenditure of funds,' and were thus referred to seek assistance from the Poor Law Authorities (MH55/136.pp 2-4). By 1930, food assistance had become an important part of the process of recovery in many areas. Exceptions included Islington where 'extra nourishment is not provided' (MH66/356, p. 3). In some instances, failing to admit a patient to a sanatorium, owing most often to the expense or lack of availability was justified on the ground that the social cost of having an infected patient in domiciliary care was offset by the provision of food relief.

The desire of public officials to address the public health problem posed by the spread of tuberculosis was ostensibly supported by the NAPT. The tuberculosis problem was recognised by the voluntary organisation as a public health problem, but both government and the NAPT were aware of the need to 'educate' the public concerning the perceived need for State involvement.[4] Since many councils had

[4] MH 55/140, National Association for the Prevention of Tuberculosis, correspondence 13th

already taken on the role of insurance committees before the 1921 Public Health Act, the opinions of the NAPT correspond to those of other voluntary bodies in the matter of relinquishing their operations to the State. The public, on the other hand, required more persuasion. In a letter to the Minister of Health, the Chair of the NAPT described how '[the] ordinary citizens should be taught to realise the need for, and the significance of, the action of the State which involves costly expenditure. ...He will become keen to see that in his area the tuberculosis scheme is maintained and perfected'. The perception of sanatoria as a '"costly failure"', however, reveal far less sanguine public attitudes toward public health.[5]

1.6.5 Institutions and Personalities

The decisions of individual health care professionals, local institutions and local government had significant effects on the quality and the type of health care offered in the decentralised interwar medical system. Their decisions on provision were not often evidence-based. Rather, a particular doctor's preference or a particular institution's policy influenced access to, and quality of, health services. As a result patients in different areas relied on the quality and opinions of local health professionals and local government to ascertain best practice based on local circumstance. In some areas, (including in Cambridgeshire, Rotherham, Wakefield detailed below) individuals, institutions and local authorities made generally good choices with regard to tuberculosis control and treatment. In other cases individual decision making was poor (including in Salford, Herefordshire and Middlesbrough), the ramifications of which can be traced through to outcome performance. Aware of this state of affairs, auditors were concerned with assessing the degree of cooperation between tuberculosis officers, medical practitioners and voluntary organisations, the quality of individual health care professionals and the ability of local authorities to perform sufficient capital investment.

A recurrent theme through the audit reports is the importance of co-operation between the different agencies and people involved in tuberculosis care. It mattered

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October, 1924.

[5] Ibid.

because early diagnosis meant a greater likelihood of treatment. However, where local general practitioners and local officials did not work together, early diagnosis could be compromised. The audit files reveal that co-operation between Tuberculosis Officers and general practitioners varied enormously both between and amongst county councils, county boroughs and the metropolitan boroughs. Cambridgeshire, The North Riding of Yorkshire, Leicestershire and Middlesex County Councils were singled out for praise as local authority areas where co-operation between Tuberculosis Officers and general practitioners was good (MH66/21; MH282, p. 24 and MH66/286, p. 27; MH66/137, p.3; MH66/169, p.6). Derbyshire however came under criticism for the fact that general practitioners were rather slow in notification and in sending cases – often after observing them for 4 – 6 months they send them in advanced stages' (Interview with Area Tuberculosis Officer, 19th May 1937, MH66/55, p.1). Meanwhile in Bristol, co-operation between the Tuberculosis Officer and general practitioners was described as being 'not altogether satisfactory' (MH66/489, p. 3), an explanation for this being that 'possibly the dispensary service does not appeal sufficiently to the practitioners of Bristol' (MH66/489, p. 3).

A second recurrent theme was the number, quality and terms of service of health professionals involved in tuberculosis work. In its report on Bristol, the auditors argued that 'one tuberculosis officer can deal fairly adequately with the amount of tuberculosis represented by 160 deaths from tuberculosis' (MH66/489, p. 3). To that extent, Bristol was criticised insofar as the Tuberculosis Officers have 'insufficient time to deal with all contacts' (there were in that City about 380-400 deaths per annum (MH66/489, p. 3). In Birkenhead, again the insufficiency of staff at the tuberculosis dispensary was identified as a problem, as it meant the existing staff were unable to devote adequate time to their work (letter to the Town Clerk, Birkenhead, 30th January, 1934, pp. 1-2). East Riding's medical staff was criticised as being 'insufficient to carry out the necessary work of visiting tuberculous people' (MH66/280, p. 4).

Length of stay was an additional issue and was noted in the auditors' report on Manchester. Manchester, it was noted, had four whole-time Assistant Tuberculosis Officers – who 'usually proceed to senior appointments elsewhere' (MH66/748, p. 72). Implicitly this might suggest problems in terms of continuity of service.

Derbyshire meanwhile was criticised for its provision of Tuberculosis nurses: 'Nurses' visits are insufficient: there have been many changes of nurses, and much sickness amongst the nurses whom, moreover have too much to do' (Interview with Area Tuberculosis Officer, 19th May 1937, MH66/55, p.1).

Some county councils were reluctant to appoint more health professionals, despite pleas for such appointments by the Ministry of Health. Northumberland council was criticised in this respect: 'Protracted correspondence has taken place and interviews have been arranged with the Council on the question of additional medical staff and the best method of utilising such staff, but despite pressure from the Department the Council have evaded a definite decision in the matter. Finally in September, 1931, we were informed by the Council that the question of an additional appointment was again deferred…' (MH66/183, p. 3). The Ministry of Health's Report suggests that the Council's reluctance may have related to its relationship with the Clinical Tuberculosis Officer whose 'work has not been impressive in the past, but has improved during recent years. A section of his Council is still dissatisfied' (MH66/183, p. 2).

A further problem was whether the health professionals were part time or whole time. Worcestershire County Council was heavily criticised in this respect: 'as regards the efficiency of the dispensary side of the scheme, the Ministry have never been very satisfied, I think, with the arrangements.' The majority of the medical staff concerned in the work only give part-time to tuberculosis, and as Dr Coutts was very doubtful as to their qualifications they were only approved in the first place as Assistant T. O.s under the general supervision of Dr. Gordon Smith … Dr Gordon Smith himself only gives part time to the County Council and as he has a large sanatorium to look after it will be understood that he cannot give very much time to the supervision of the work of the Assistant T. O.s especially in view of the fact that to do so involves a great deal of time in travelling' (D. Williamson, 20th November, 1930, MH66/275, p.3). In 1939, it its report on Tuberculosis care in Wales, the Ministry of Health noted the problem caused when health professionals were part-time and unable, therefore to dedicate all their time to their tuberculosis work Ministry of Health, 1939, p. 234). An explanation for the preference for part-time officials according to the Ministry of Health was 'possibly they are also influenced

by the fear that whole-time officials would be much more strict, and his recommendations might involve them in further expenditure of money' (Ministry of Health, 1939, p. 239). . Part-time status could also lead to a conflict of interest (Ministry of Health, 1939, p. 238). A final problem was the perceived quality of the officials. Manchester was criticised for the age and 'class' of its 13 women tuberculosis officers. Although the audit report noted that all were fully trained, it continued 'some (were) ageing, perhaps 3 or 4 showing signs of wear and tear, the others pretty good but mostly of the servant or mill hand class' (MH66/742, p-. 73). In marked contrast, the part-time consulting Tuberculosis Officer in Essex was singled out by the Ministry as 'one of the most able T. O.s in England' (MH66/85, p. 3). The quality of work carried out at the Dispensaries and in residential institutions had improved significantly in the country 'as a direct consequence' of his activities (MH66/85, p. 3).

The implications of an inadequate staff were spelt out in the report on West Yorkshire. 'A careful enquiry has been made to ascertain why these environmental and other functions are not being prosecuted to their fullest extent in the County, and the main conclusion arrived at is that the present staff is inadequate numerically to cope with the amount of work, and the direct results of this inadequacy are reflected in hasty diagnosis, injudicious admission of patients to sanatorium, inability to visit homes frequently enough, and consequent omission of the essential duty of examination and supervision of contacts' (MH66/292, p. 12).

The final theme, not surprisingly, was one of cost efficiency and value for money in terms of the expenditure on tuberculosis, not least in the straightened circumstances of the interwar period. In its letter to Barrow Borough Council in 1932, the Ministry of Health acknowledged that the 'industrial and financial position' of the City made it difficult for the Council to develop services 'to the extent that they would desire' but suggested that a review of the organisation and administration of the services could secure improvements in many directions' (letter to Town Clerk, Barrow-in-Furness, 13th June 1932, MH66/1066, p. 2). The theme of the problem of undertaking large scale expenditure in the prevailing economic environment was also recognised in the Ministry of Health's letter to Bath City Council; again the recommendation was an improvement in organisation and administration' (letter to

Town Clerk, Bath, 14th December, 1931, MH66/1066, p. 1). Liverpool attracted comment by the auditors for its perceived lack of economy - the distribution of milk and eggs to those with tuberculosis was noted as being 'one of the few really economical services in Liverpool' (MH66/489, p. 65). One such item of expenditure was the practice of referring about 700 non-insured patients a year for treatment by their own doctors. Whilst this relieved the work load of the dispensary staff, the system was criticised for being 'an expensive luxury and for putting 'a great deal of money into the pockets of private practitioners' (MH66/489, p. 66). The cost to Liverpool was about £7,000 to £8,000 a year, of which £6,000 was for medical attendance at the rate of 3 shillings a visit (MH66/489, p. 65). The auditors recommended that the practice be discontinued in order to release money for the appointment of an additional tuberculosis officers, but noted that Liverpool's past history indicated that it would not give up such a system 'until forced by dire necessity' (MH66/489, p. 66).

1.7 Why did some areas do well, but others not so well?

We took a sample of authorities for more detailed scrutiny – we wished to explore why some areas managed better than others to drive down the tuberculosis problem. Our selected sample was based on the tuberculosis death records from the population of all counties and towns between 1920 and 1938. We identified those areas which recorded particularly high, particularly low and representative reductions in mortality. Amongst counties, those counties achieving the greatest decline between 1920 and 1938 were Northumberland (56%), North Riding (56%), Oxfordshire (55%), Herefordshire (55%), Devon (54%) and Dorset (54%). Those recording the smallest declines were Cumberland (12%), Staffordshire (28%), Warwickshire (28%) and Wiltshire (28%). The towns which achieved the greatest rates of returns were Wakefield (63%), Rotherham (60%), Dewsbury (58%), Rochdale (59%) and Southend (58%). Those which recorded the smallest were Eastbourne (16%) and Middlesbrough (13%). Cambridgeshire is representative with a recorded decline of 46%.

A possible explanation for this difference is that the counties and towns in the pre-

chemotherapeutic era which failed to make substantial progress had already reached a natural limit in their ability to prevent and treat cases of tuberculosis by 1920. Additional reductions in mortality were necessarily marginal. Staffordshire exemplifies the problem. Public health arrangements in the county were well administered and extensive in general, and in the processing of tuberculosis cases in particular. Public health workers and officials worked in close liaison, (MH 66/223 p. 12), after care provision was extensive, (MH 66/223 p. 42), health education was prioritised (MH 66/223 p. 45) and sanatorium accommodation was available for men, women and children at various specialised centres throughout the county (MH 66/223 p. 48). Extant problems, such as the partial failure of the village settlement scheme and the lack of oversight of Public Assistance institutions by the tuberculosis officer, were of negligible importance, and related to a lack of willingness of the population to participate in the case of the former and an administrative failure without any effect on patients in the latter. In general, coordination in the county was good. The maternity and child welfare service and the school medical service both made regular use of the tuberculosis officer (MH 66/223 p. 126).

Cumberland and Staffordshire suffered similarly from tuberculosis mortality in 1920 reporting death rates of 672 and 687 per million respectively. These rates compare to a mean of 735 deaths per million for all counties in 1920. Hence both counties began the immediate post war period in relatively favourable circumstances. Whereas Staffordshire and other counties such as Cambridge and Warwickshire converged to between 300 and 500 deaths per million, Cumberland saw no change in rates of tuberculosis mortality outside statistical noise. The explanation for this divergence in performance is twofold. Firstly, as a public health entity, Cumberland was fractured. The relatively well connected areas in and around Carlisle and Penrith en route to Scotland and in the agricultural Eden Valley permitted sophisticated health systems to develop and wealth to be generated. The Cumbrian Mountains separate the east of the county from the small industrial towns of Arlecdon and Frizlington, Egremont, Millom, Whitehaven and Workington in the west. These towns were crowded with unskilled migrant workers from Ireland and suffered a significant tuberculosis burden. A high proportion of the population were working class, often living in overcrowded slums. According to the audit files, the relative isolation of western seaboard towns also meant necessarily lesser health service coverage. For example

the district Medical Officer of Health often only visited sessions in towns other than Whitehaven twice each month and reported poor attendance (MH 66/42 p. 46). Without the inclusion of these towns, tuberculosis mortality rates fell in the county as elsewhere (MH 66/42 p. 44). Secondly, the 1933 audit of the tuberculosis scheme revealed systematic failures throughout in terms of the county's tuberculosis provision: too few patients on the dispensary register compared to the notification register; too few consultations; neglect of adult contacts; failure to advise contacts of tuberculous persons to seek medical examination; lack of provision of home visits to the working class urban districts in West Cumberland; too few sputum and X-ray examinations and provision of outdated and ineffective treatments (MH 66/41 pp. 47 - 48). Cumberland is an example of an authority of 'tuberculosis failure'.

Dissimilar to Cumberland and many other rural counties in 1930, Cambridgeshire made extensive use of X-ray facilities in the process of identifying potential cases. Four weekly sessions were held with patients conveniently at the dispensary rather than requiring additional travel or additional fees for out of county services. In 1932 a total of 1458 patients attended X-ray sessions at the dispensary (MH 66/23 pp. 1-3). Centralisation of the dispensary in a county with few natural impediments to travel and a population based primarily in Cambridge, Ely and Huntingdon facilitated relatively easy access. The drive for X-ray examinations and the identification of cases early was not a matter of local policy. Rather, as was the case for many areas before the advent of centralised medicine, the efficacy of public health rested on the motivations of local Health Officers and General Practitioners. Dr Philips, a local physician working in the Regent Street dispensary in Cambridge was passionate about the use of X-ray facilities in the county and as a result Cambridgeshire had the highest ratio of X-rays to cases of any English county. Early detection of cases in this way meant that more patients would benefit from sanatorium treatment, both in terms of their own convalescence and also in terms of the prevention of the spread of infection to their contacts. Areas which focused on advanced cases could only offer patients a place to die either in Poor Law Institutions or their own homes.

Other public health drives in Cambridge were equally impressive. Aftercare work was identified as being substantial and effective in the county. Assistance was given

to patients who had received sanatorium treatment by supplementing their incomes or obtaining suitable employment for them whilst they were recovering (MH66/23 p. 3) Financial and nutritional assistance was often long term and substantial, with some patients receiving up to thirty months of support after leaving an institution. Patients were supported financially with grants varying from 2/6d to 10/- per week and others were offered nutritional support in the form of milk and eggs and assistance with housework. These measures received careful oversight from the Public Assistance Committee on a case by case basis. (MH66/23 p. 4). It is of significance that much of this generosity for the tuberculosis afflicted was sustained by community involvement. The Cambridgeshire Association's appeal for funds for tuberculosis aftercare generated significant financial support locally. Additionally, greater numbers of patients could be supported by existing sanatorium facilities because of efficiency drives delivering a drop in the cost of sanatorium treatment of £3000. (MH 66/23 Appendix L.1)

In many respects Warwickshire mirrors the experience of Cambridgeshire with initially low but slowly falling mortality rates and excellent public health practices. Firstly, cooperation between different public agencies was extensive. Because of the geographical proximity of Warwickshire to major industrial centres in the West Midlands, the tuberculosis scheme was administered jointly by the County Council and Coventry County Borough by a Joint Tuberculosis Committee. Examination of contacts and cases was, if anything, conducted more extensively than in Cambridgeshire, across a network of seven dispensaries. As was the case across the country, little in the way of direct treatment was offered by dispensaries, but the dispensary in Coventry and subsidiaries in Warwickshire offered additional supplies of disinfectants, bed pans and bed rests to patients and their families. (MH 66/258 p. 74) Patients who suspected infection were screened for tuberculosis via a process of repeated sputum examination. Often upwards of five specimens would be taken to avoid error. The practices at the Coventry dispensary with regard to the examination of contacts were superior to those of the Cambridge dispensary. Coventry employed a unique approach to the examination of contacts. Only if an X-ray showed tubercular lesions or if a patient was displaying symptoms was a clinical examination performed. All contacts of every new case of tuberculosis were examined, not just the elderly or children. Examinations were conducted deliberately outside of

working hours, in contrast to the Regent Street sessions, in order to minimise disruption of contacts' working week. (MH 66/258 pp.76 – 78) As in Cambridgeshire, aftercare measures were extensive and probably more focused on those in need. Extra nourishment was granted to patients attending the dispensaries by the Joint Committee on the recommendations of the Tuberculosis Officer. Extra nourishment was not given to families whose income, after deducting rent, exceeded 10/- per week.

Perhaps an explanation for Cambridgeshire's and Warwickshire's early arrival at low but slowly falling mortality rates is not only as a result of exemplary public health and the role played by the University of Cambridge in generating and maintaining local health expertise, but also the nature of work in the country outside their provincial towns. Both counties were predominantly agricultural in the early twentieth century. A large proportion of the population was engaged in agriculture. Cambridgeshire had some light industries which provided male employment including cement making, brick making, paper making, jam making and printing. Similarly many men in Warwickshire, if they were employed in industry, worked in the north of the county in metal working and coal mining, or in the motor car industry in Coventry. Analysis of the returns pertaining to employment by industry reveal that relatively few men were employed outside of agriculture in both counties and so avoided much of the risk of contracting tuberculosis traditionally associated with industrial and domiciliary overcrowding and exhaustion. (MH 66/258 p. 8, MH 66/23 p. Appendix A) In terms of public health practice Cambridgeshire, Warwickshire and Staffordshire succeeded where Cumberland failed. Counties which were able to utilise existing medical technologies and resources effectively were able to approach a lower bound, curtailing avoidable mortality. In addition those counties which suffered the health implications of an industrial heritage fared worse than those that did not.

A similar pattern can also be found in the case of towns which already had low mortality rates throughout the 1920s. Eastbourne is the prime example of a town which experienced the familiar pattern of low initial rates of tuberculosis, slowly falling thereafter. Although health services in the town were adequate, little attempt was made to diagnose patients as early as possible. Rather the Town Council,

economical and conservative in character, preferred a policy of strict segregation of those known to be infected. There was little use of modern diagnostic and treatment methods with patients only referred to dispensaries at later stages of the disease. The Minister of Health for the town was rigid in his approach, favouring later diagnosis and admission of advanced cases to the sanatoria and a lack of interest in preventive measures. (MH 66/593 pp. 77-78) Eastbourne's early success in dealing with high levels of tuberculosis more likely stems from another source.

Before the arrival of the railway in Eastbourne in 1949, large landowners planned the development of the town, which was little more than a village. The town's population grew rapidly from 3433 in 1851 to 22014 in 1881. Much of the housing stock was good relative to other towns, fit for habitation and built on wide streets. Overcrowding was below that for other county boroughs in the 1930s. Healthy layout of the streets and good sewerage provided the town with some robustness against infectious disease. (MH 66/593 p. 3) This is because Eastbourne never developed as an industrial area. Rather occupations were mainly found in tourism, health and leisure, and in education and commanded far higher incomes. These high incomes persisted into the 1930s and Eastbourne was generally considered to be without any significant slums. In this light, Eastbourne's success occurred because of the wealth of its inhabitants rather than any particular strengths of public health. Early town planning and the absence of industrial occupations likely contributed to the early decline in tuberculosis mortality and convergence on a lower bound thereafter.

By contrast Middlesbrough had high levels of tuberculosis mortality in 1920 which remained high as late as 1938 mirroring the experience of western industrial townships in Cumberland. Growth of the town in the nineteenth century was based around the exploitation of ironstone in the Cleveland Hills. Coal from Durham could be used to smelt this iron and so ironworks in Middlesbrough developed because of its convenient location. The failure of Middlesbrough to deal with the problem of tuberculosis is reflective of its failure to deal with infectious diseases more generally. For example, outbreaks of smallpox forced the closure of the only sanatorium in the area for use as a smallpox hospital. (MH 66/754 p. 22) Inadequate accommodation for tuberculosis patients became a problem after the closure of the local Hemlington

Hospital to tuberculosis patients. Without a sanatorium and with the closure of the main local hospital, Middlesbrough only provided a third of the beds of the average county borough. (MH 66/754 p. 40) Advanced cases were either left to die in their homes or left for the Guardians to deal with. As a result of a lack of facilities, those which did receive treatment, only did so for vastly shortened periods of time. At the West Lane Hospital no X-ray facilities were available to tuberculosis patients and no specialist treatment was offered. In addition at the Holgate Institution for men, wooden verandas blocked sunlight into the tuberculosis ward. Women in Middlesbrough fared even worse. They were confined to a ground floor general ward with provision of a mere nine beds. (MH 66/754 p. 43) So dire was the situation in in Middlesbrough that correspondence between the Borough Council and the Ministry of Health reveals how the authority sought central support for its lack of resources (MH 66/754 miscellaneous correspondence). Despite pleas for additional support in 1932, the high mortality rates had not receded by 1940.

Arguably many unnecessary deaths from tuberculosis occurred in Middlesbrough. In light of the effective dispensary, it does not seem that there was a general administrative failure in the town. In terms of the dispensary, provision was not lacking. As in well performing areas, emphasis was placed on use of X-ray, examination of contacts, multiple sputum examinations, home visits by health visitors with access to motor cars and follow up of suspected cases. But Middlesbrough, as other industrial areas, suffered from the dual curse of high employment in traditional industries and lack of resources generally for infectious disease provision.

There were, however, examples of industrial areas which were able to catch up with the leading counties. Both Rotherham and Wakefield are examples of industrial towns which experienced the most rapid and sustained declines in tuberculosis mortality after 1920. Wakefield for example suffered 1243 deaths per million in 1920 but only 582 per million in 1938, less than half of the immediately post-war level. (See M.O.H. returns) The latter figure is comparable to the level experienced in many counties only five years earlier. Catch-up in these towns was well under way. In many important ways these towns were similar to Middlesbrough and contrasted with Eastbourne. In Rotherham metal workers formed the largest proportion of the

male working population and in Wakefield industry was dominated by iron industries, quarries and chemical works. The reasons for the relative decline in tuberculosis in these towns owes as much to misfortunes in Middlesbrough as it does to health initiatives in the towns themselves.

In Rotherham the main strength of the tuberculosis scheme was the willingness of general practitioners and the Tuberculosis Officer to recognise and attempt to remedy problems. In Rotherham, success was undoubtedly the result of a sustained effort to identify cases as quickly as possible. Two aspects of the work were significant: the bacteriological work and a concerted campaign to detect cases through an integrated visiting system and an enlightened move to eliminate some of the stigma associated with the disease which prevented people from seeking diagnosis. In Rotherham procedures for identification of infectious diseases were already sophisticated. Three agencies carried out laboratory work; the Medical Officer of Health investigated simple microscopical examinations at the Public Health Laboratory for both public and voluntary hospitals; the University of Sheffield undertook more advanced work including the Wassermann tests as well as examinations of bacteriological contaminants in the milk supply and finally analytical work for the City Corporation was undertaken by a public analyst (MH 66/847 p. 84).

This was complemented by an emphasis on home visiting where one visitor was responsible for all aspects of health. At the turn of the decade, the Borough of Rotherham was criticised because too few health visits were made (MH 66/847 p. 6, p. 63). The criticism was quickly addressed and a major overhaul of the system introduced. In 1933, home visiting arrangements were re-organised. From 1933, health visitors were given responsibility for all domiciliary visiting and each visitor was given responsibility for a specific are. Plans for routine work were drawn up so that the health visitor and medical officers knew which parts of the area were to be surveyed in a given week. The new scheme meant one visitor had knowledge of given areas and was in a better position to develop linkages between tuberculosis and maternity and child welfare sections (WRD7/6/3/216, p. 88). This all meant that more frequent visits were paid, and the initial home visit to a new case was made within a few days of notification (WRD7/7/6/3/216, p. 61). In addition, the Borough

put special emphasis on extending the work of the dispensary to include relatives of cases who had died; such cases were kept under close observation for up to a year (WRD7/6/3/217, p.64).

Effective liaison between agents is a strong theme of the Rotherham initiatives. This included the arrangement whereby the Tuberculosis Dispensary and the Sanatorium were under the control of one medical officer so that 'the officer acquires an intimate knowledge of the patient, the social and home conditions and can correlate these with the clinical condition and sanatorium treatment required to the best advantage of the patient' (WRD7/6/3/216, p. 61). Second, the Borough had a Tuberculosis Care Committee whose 'work is so intimately interlaced with the official tuberculosis work of the borough' (WRD7/6/3/218, p. 14). The emphasis here was on providing nutrition to the needy. This Committee was formed in 1929; by 1936, 6,100 gallons of milk, 32,500 eggs and 4,100 lbs. of meat were issued by the Committee in grants (WRD7/6/3/219, p. 13).

Finally, Rotherham was aware that it needed to persuade people to be diagnosed – as early as possible - and one of the key deterrents was the fact that 'the word "tuberculosis" to a large proportion of the lay population conjures visions of a terrible and fatal disease. The patients, who may be very slight cases, often postpone their visit to the dispensary until they definitely find themselves deteriorating in health. By this time the disease may have taken root and become difficult or impossible to eradicate' (WRD7/6/3/217, p. 74). The stigma had real meaning for the people of Rotherham in terms of their employability and work relations. 'Some of this stigma is still related to the disease "tuberculosis" and some of this stigma may attach itself to those who attend the dispensary. The unfortunate patient who returns to work may find himself the object of some coolness and suspicion from his fellow workers and employers, or may even find his job given to another. Difficulties and unpleasantness have been known to ensue to patients in whom no tuberculosis was found, simply because they had visited the tuberculosis dispensary'' (WRD7/6/3/217, p. 74). The Borough devised a way of overcoming the stigma attached to tuberculosis by re-naming the Tuberculosis Dispensary the 'Chest Diseases Clinic' (WRD7/6/3/217, p.74).

The narrative in Rotherham and Wakefield was also one of continued improvement in public health facilities overall: and the political will to do so. In Wakefield, there was a sustained drive to improve living conditions going back into the late nineteenth century through the appointment in 1890 of a Sanitary inspector who pursued a 'zealous and successful' campaign to abolish privies and substitute them with water closets and was the first Cleansing Superintendent in the country to 'introduce the system of controlled tipping in the disposal of domestic refuse' (WRD7/6/3/282, p. 127). A further major improvement was the provision of a new water supply from the Pennine Hills from 1888 which led to a 'pure and plentiful water supply', (WRD7/6/3/282, p. 129). Meanwhile, Wakefield appointed its first Part time Medical Officer of Health in 1866; this was changed into a full time position in 1903 (WRD7/6/3/282, p. 127). The full time Medical Officer of Health was a vehement and passionate campaigner for housing improvements and devoted much time both in the pre-war and interwar years to persuading the Council to improve its housing stock (WRD7/6/3/282, pp. 172-5). Between 1903, when he began his campaign and 1935, 903 houses were deemed to be unfit for occupation; between 1920 and 1935, the Corporation built 3,653 houses (WRD7/6/3/282, pp 175 and 177). The new houses built after 1925 had to abide by a byelaw which set standards for a healthy dwelling for the working class (MH 66/947. Survey Brief)

In Wakefield, a thirty three year retrospective assessment of the explanation for the decline in the tuberculosis problem in the City noted that the decrease in mortality was declining before special measures to combat the disease in the City were instituted, but argued that the special measures undertaken 'must have played a part in bringing about the very marked reduction in the disease' WRD7/6/3/282, p. 151). The measures highlighted as being important included the establishment of a dispensary in 1913, of sanatoria accommodation in 1912, the building of six special houses for ex-Sanatorium patients in 1926, the opening of a fever hospital with a tuberculosis block in 1934 which dealt with those patients requiring hospital treatment, and the existence of a bacteriological laboratory from 1912, based in Wakefield itself. WRD7/6/3/282, pp. 147-162). The report makes it clear that at least initially, persuading the Council to take action was not easy – a special report written in 1905 arguing the case for compulsory notification, provision of sanatoria accommodation, provision of hospital accommodation for advanced cases, periodical

veterinary inspection of dairy cows, with a view of reducing milk infection and the demolition of insanitary houses and rehousing met with no response: 'the only outcome was that the Corporation passed a resolution urging the Local Government Board to make pulmonary tuberculosis a compulsorily notifiable disease' WRD7/6/3/282, p. 152). A year later, however, the situation was to change – largely due to the then Chairman of the Board of Guardians who took a leading role in the propaganda work against the disease and to the unrelenting campaign by the then Medical Officer of Health to institute dispensaries and a sanatorium (WRD7/6/3/282, p. 153). Probably the greatest spur to action by the Council was on the 15th July 1912 when the Sanatorium Benefit of the National Insurance Act of 1911 came into operation which imposed responsibility for preparing and running schemes for sanatoria and dispensaries on County Councils and County boroughs – 'and to the maintenance of which the Government contributed one half of the cost'; when Wakefield became a County Borough in 1915 it took over full responsibility for the Tuberculosis scheme – and the Medical Officer of Health 'was appointed Clinical as well as administrative Tuberculosis Officer' (WRD7/6/3/282, p. 154).

Doubt, however, is cast on the thesis that public health initiatives were the primary driver of the decline of tuberculosis in the pre-chemotherapeutic era by the experience of Herefordshire. Herefordshire is a story of the total failure of public health, public transport and local government during the 1920s and 1930s and yet in this context, tuberculosis mortality rates had declined by 1938 to around half their 1920 level. In almost every stage of the tuberculosis scheme there was failure. Bureaucracy obstructed the identification of the infected. Patients had to wait for the approval of both the local general practitioner, who gives first line approval and then Sanatorium and Benefits committee, to which the case was referred by the general practitioner. (MH 66/93 p. 43) Once referred patients had to wait for the committee to hold its monthly session. In addition patients often had to be taken by car to X-ray facilities about forty miles from the centre of the county because of the absence of public transport in rural areas. (MH 66/93 pp. 41 – 43, p.6) This resulted in a total of only five X-rays taken during the year 1930.

Serious defects in the county sanatorium and isolation hospital hindered the prevention of the spread of infection and regression of the disease in those infected.

The principal criticisms were the poor standard of clinical work, the number of cases admitted which are not tuberculous, lack of facilities for X-ray work, pollution in the water supply, the lack of functioning central heating and a lack of storage space for patients' belongings. (MH 66/93 pp. 45-46) If patients did receive treatment, aftercare was unavailable. The sources of the problems experienced in Herefordshire were political. Outside Hereford, most of the county comprised poor agricultural communities, mostly of working class. A politically inactive voting population meant that the County Council lacked incentives to improve overall public health provision. (MH 66/93 p. 9) Operationally the tuberculosis scheme was hindered by the absence of cooperation between the Tuberculosis Officer, the Public Assistance Committee and voluntary organisations, a hallmark of successful County and County Borough councils.

Herefordshire is an outlier. The marriage of disastrous public health provision with rapidly falling mortality rates is an odd one. It suggests that, as is argued here, that a situation existed in English counties and towns whereby sustained improvements in living conditions, post 1918, could lead to declines in the rate of tuberculosis mortality, independent of public health effort.

Often failure to improve living conditions resulted in failure to reduce tuberculosis mortality. Public health officials maintained that the root of the tuberculosis problem in London boroughs and northern industrial towns such as Salford and Middlesbrough lay in poor living conditions and overcrowding. Success resulted where these issues were remedied and failure where they were not. In the London Boroughs of Bermondsey and Holborn overcrowding was emphasised by the Minister of Health. In many ways these boroughs were analogous. Both were primarily working class areas which suffered from overcrowding. In these areas improvement in housing was under way during the 1920s but many poorly constructed and unsanitary dwellings remained. (MH 66/351 pp. 9 –10). Although there was a lack of cooperation between the minister of health and housing officials in Bermondsey, (MH 66/307 pp. 6-7) Bermondsey was able to reduce rates of tuberculosis mortality more successfully than its analogue. Bermondsey instituted frequent health visits to patients residing in publicly maintained shelters for purposes of both patient and family education. Patients and families were informed that it was

essential for separation of the patient from the rest of the family and that patients should 'not run into the house at the first shower of rain.' (MH 66/307 p. 65). It can be speculated that Bermondsey's success lay in one of the few examples of effective health education of the period which procured patient isolation. Procedures in Holborn failed. Patients were discharged to overcrowded homes because of the unsuitability of general hospitals. (MH 66/351 p.45) This suggests that public health had the opportunity to be complementary to improvements in living conditions.

Where an authority had the political will, energetic and committed medical and sanitary officers and the financial resources, there was co-operation between various agencies significant steps were taken to improve living conditions in general, to isolate the infected from close family members and to provide via the dispensaries effective methods of identifying the disease and providing support in the Community. Authorities in areas of small and poverty stricken populations lacked the resources to provide the necessary health initiatives, and could not afford the fully qualified, full time medical officers of health who could campaign for better conditions (Ministry of Health, 1939, pp. 228-9). In some areas, local resistance precluded integration of resources and amalgamations of district resources – because local ‘jealousies and indignation’ objected to any such amalgamations (Ministry of Health, 1939, p. 235). Where agencies worked together for a common purpose, even when the area was relatively poor, significant progress was made. Here the case of Barnsley is instructive: the emphasis in the Medical Officer of Health Reports is not only on what the various agencies did, but also on the close co-operation between them. General practitioners co-operated with the Tuberculosis Officer whilst Maternity and Child Welfare and School Clinics worked closely with the Tuberculosis Officer (WRD7/6/3/10, pp. 128-129). The Public Assistance Committee and the Unemployment Board provided special nourishment for needy cases, the Housing Committee worked to move tuberculous families from bad home conditions, the Education Committee provided free milk to children, whilst home visits by doctors and nurses advised on precautions for limiting infection and educated families on warning symptoms (WRD7/6/3/10, pp. 112-118).

1.8 Conclusion

Given the available econometric evidence, policy makers may have underestimated the importance of overcrowding in working and living conditions in contributing to mortality rates. In general, towns suffered to a greater extent than counties, and towns with a heritage of traditional or heavy industries fared worse than those without. There is however a critical rider in insofar as many local Medical Officers of Health recognised that tuberculosis was a problem not best remedied by a conventional clinical approach and viewed poor housing and overcrowding as a, if not the, major constraint on improving tuberculosis outcomes. To that extent, the individual and local Medical Officers of Health were correct. In all specifications except OLS (5), our measure of overcrowding in the home was significant and positively associated with both tuberculosis morbidity and mortality. In addition, the results suggest that rural lifestyles were protective independent of living and population densities. In 1924, the Tuberculosis Officer for York argued that the returns on tuberculosis initiatives 'give but a poor return for the amount of money expended' given the 'unhygienic environments in which so considerable a portion of the community live' (WRD7/6/3/361, p. 6).

Overcrowding in the view of health officers related not only to the number of rooms and the space available but also from a health perspective when a lack of rooms in the household meant that the member of the family suffering from tuberculosis could not be isolated. We have identified local initiatives in Liverpool, Sheffield and Leicester which recognised the problem and undertook proactive policies. Statistical evidence provided in section 1.5 offers evidence that overcrowding within the home was an independently correlated with higher tuberculosis morbidity and mortality when accounting for both urban setting and population per acre. In other words, policy makers were on the right track.

There was preoccupation among policy makers with the quality and quantity of X-rays and sputum tests. As the regression results reveal, simply identifying patients as infected was not enough. The best method to prevent the spread of infection was to isolate the infected in shelters or sanatoria after an accurate diagnosis was made. The

results reveal that the interwar tuberculosis scheme failed to reduce the case fatality ratio which supports the hypothesis that the best available public health policy was to isolate the infected and examine contacts. Interventions such as home visits or consultations, though important from the viewpoint of health education, did little to affect outcomes for tuberculosis in particular. However, policy makers did recognise that prevention was preferable to treatment. Areas which admitted early stage cases into residential treatment, whilst isolating later stage cases, often saw faster declines in mortality. Areas, such as Derbyshire and the West Riding of Yorkshire which did not do this were criticised heavily for failing to admit patients in the early stages of disease.

One metric does offer an important insight into aspects of the tuberculosis scheme that were important. The ability of local authorities to get patients onto the dispensary register after notification alludes to the importance of the dispensary as a hub for both diagnosis of tuberculosis, education and examination of contacts. Areas such as Kent and Rotherham can attribute some of their success to the activities of the dispensaries including care schemes, frequent domiciliary work and monitoring of contacts. We note however that whether in reality this intervention improved survival probabilities or whether this variable is acting as a proxy for administrative efficacy is unclear.

Morbidity and mortality in relation to pulmonary tuberculosis can be seen as a complement to standard measures of the standard of living insofar as the risks of infection related to sustained exposure in overcrowded living and working conditions whilst the risks of developing active symptoms were related to malnutrition. To that extent, the revealed variation in respiratory tuberculosis adds another dimension to our understanding of living standards in England and Wales in the interwar years. Adherents and critics of the McKeown thesis will alike find support in our findings. Living standards did matter – but so did medical intervention.

Some policy makers recognised tuberculosis as a disease that has its roots in poor standards of living. Contemporaries' list of associations included overexertion, malnutrition, alcohol use, insanity, syphilis, diabetes, influenza, measles, whooping cough, overcrowding, insufficient ventilation in the home or work place, the nature

of certain occupations and the sanitary conditions of the dwelling. Many of these factors are recognised to be associated with tuberculosis activation in the present (MH55/139, p. 5). Thus interventions which would improve quality of life conducive to arresting the development of active symptoms were sought. Given that the disease was in continuous decline, it is argued here that the key to successful reduction of the burden of the disease were improvements in the standard of living. Where families were smaller and living and working conditions less cramped, tuberculosis was less burdensome. If family size can act as a proxy for nutrition, our results suggest that nutrition was marginally significant in explaining active cases. Innovative contemporary policy makers asked whether aspects of economic growth important to health could be accelerated and thus emphasised prevention rather than cure.

There was no single explanation for why some authorities were better able than others to drive down tuberculosis mortality. The 1929 audit revealed individual and particular failings but what is noteworthy is how few areas attracted criticism in relation to their tuberculosis schemes. In the total scheme of things, the number of authorities where failings were identified were few, and amongst those authorities the criticisms overall related to specific rather than overall issues, What we find from our analysis of the success stories, is that success derived not from one but from a total package of policies, sufficient staff to carry out those policies and effective liaison between different health professionals.

Chapter 1 has identified the conditions under which local authorities in England and Wales operated to control the transmission and arrest symptoms of tuberculosis and provide convalescence to sufferers. That healthcare in interwar Britain was not centralised has provided a unique opportunity to investigate how different jurisdictions experimented with tackling (or not tackling) the tuberculosis problem in the absence of curative drugs that became available after the Second World War. The next chapter investigates how lessons learned from Britain's, and other developed countries', success in reducing tuberculosis mortality post-war were not necessarily applicable in a developing world setting. Despite ongoing improvements in diagnosis, prevention and treatment in the West, Chapter 2 investigates why, in a period during which effective cures were available, tuberculosis morbidity and mortality remained significant burdens on populations in the developing setting.

Chapter 3 uses insights gained from the approaches of policy makers, institutions, doctors and voluntary organisations in interwar Britain to investigate the relative importance of socio-economic and medical factors in explaining respiratory tuberculosis incidence and mortality across a global sample of countries. Chapter 3 will examine the extent to which socio-economic approaches advocated in the pre-chemotherapeutic era complemented the medical interventions available in the era of reliable curative medicine. Directly measuring the nutritional content of diets for sampled countries becomes possible in the post-war era. Therefore, it is investigated whether diet is important in explaining differences in case (and death) rates across countries – a relationship that is only alluded to by those trying to address the tuberculosis problem in England and Wales.

1.9 Appendix

Table 1.1 **Summary Statistics: Respiratory Tuberculosis Mortality in English and Welsh Counties (crude death rate per million living)**

Year	Mean	Max	Min	Std. Dev.
1920	735	1,072	554	110
1921	750	1,081	567	122
1922	756	1,078	594	105
1923	693	971	326	119
1924	697	1,007	272	118
1925	691	948	439	100
1926	626	883	428	89
1927	654	912	388	106
1928	608	894	385	96
1929	635	957	364	99
1930	601	872	369	104
1931	595	895	223	118
1932	556	818	403	82
1933	538	821	325	101
1934	502	763	281	97
1935	480	680	243	87
1936	468	688	352	77
1937	455	705	284	86
1938	417	637	280	72
1939	416	701	303	82

Source: Registrar General's statistical review of England and Wales.

Table 1.2 **Summary Statistics: Respiratory Tuberculosis Mortality in English and Welsh Towns (crude death rate per million living)**

Year	Mean	Max	Min	Std. Dev
1919	1,212	1,447	553	117
1920	974	1,399	597	198
1921	967	1,530	527	200
1922	972	1,364	453	192
1923	923	1,357	439	189
1924	946	1,431	506	181
1925	945	1,398	513	197
1926	866	1,261	436	176
1927	892	1,401	162	197
1928	837	1,634	179	213
1929	893	1,413	512	194
1930	830	1,364	364	195
1931	848	1,464	483	215
1932	788	1,447	355	191
1933	786	1,376	466	193
1934	721	1,268	370	167
1935	689	1,150	391	170
1936	668	1,145	343	151
1937	672	1,029	359	158
1938	621	974	303	156
1939	617	1,031	336	158

Source: Registrar General's statistical review of England and Wales.

Table 1.3 **Achievements in the Control of Respiratory Tuberculosis Mortality: Towns in England and Wales**

	Highest				Lowest		
	1920	**1930**	**1938**		**1920**	**1930**	**1938**
Liverpool	1,399	1,233	810	Canterbury	597	377	308
Tynemouth	1,395	945	827	Eastbourne	599	598	620
Newcastle	1,379	1,054	862	Blackburn	661	827	664
Salford	1,360	1,256	963	Southport	669	715	318
Gateshead	1,337	1,062	974	Carlisle	684	739	498
Cardiff	1,336	952	856	Bath	689	364	586
S Shields	1,326	1,245	833	Dudley	694	907	649
Bootle	1,250	1,146	931	Dewsbury	713	809	303
Wakefield	1,243	481	458	Halifax	758	642	452
Manchester	1,218	1,185	856	Chester	762	925	564

Source: Table 10 Annual Statistical Review of the Registrar General.
Notes: The data refer to the death rate (all ages) per 1 million living persons. The table refers to the ten towns with the highest and lowest mortality rates in 1920.

Table 1.4 **Male Unemployment in 1931: Summary Statistics**

Towns	
Mean	16.39
Std. Dev.	7.01
Median	15.57
Max	36.63
Min	5.43
Counties	
Mean	9.38
Std. Dev.	3.50
Median	8.63
Max	22.80
Min	5.23

Source: Census of Population, 1931, Occupational Tables, Table 16.

Table 1.5 **Overcrowding in Housing England and Wales in 1921 and 1931: Summary Statistics**

Overcrowding: Counties in England and Wales					
	Mean	**Std. Dev.**	**Median**	**Max**	**Min**
Definition: the number per 1,000 private families living at a density of more than 3 persons per room					
1921	5.98	9.69	3	53	1
1931	4.6	5.83	2	27	0
Definition: the number per 1,000 private families living at a density of more than 2 but not more than 3 persons per room					
1921	35.71	28.03	27	146	11
1931	24.18	18.16	16	86	7
Definition: the number per 1,000 private families living at a density of more than 1.5 but not more than 2 persons per room					
1921	90.8	36.92	80	197	49
1931	67.07	30.19	56	160	32

Source: 1931 Census Housing Volume Table 13

Overcrowding: Towns in England and Wales					
	mean	stdev	median	max	min
Definition: the number per 1,000 private families living at a density of more than 3 persons per room					
1921	12.65	20.72	6	99	0
1931	10.63	15.12	5	75	1
Definition: the number per 1,000 private families living at a density of more than 2 but not more than 3 persons per room					
1921	51.05	35.5	43	162	9
1931	34.83	25.56	27	123	7
Definition: the number per 1,000 private families living at a density of more than 1.5 but not more than 2 persons per room					
1921	110.07	39.44	112	209	45
1931	79.82	33.24	75	158	32

Source: 1931 Census Housing Volume Table 12

Table 1.6 **Residential Policy for Patients with Tuberculosis in 1929**

Summary statistics for County Boroughs	**Beds available per 100 deaths**	**Patients treated per 100 deaths**	**Per cent staying more than 3 months**	**Per cent staying more than 6 months**
Mean	60.17	121.35	58.31	24.19
Median	58	114	59.2	16.7
Std. Dev.	23.44	70.67	13.60	25.50
Max	122	605	94.4	100
Min	16	5	16.8	0
Summary statistics for County Councils				
Mean	60.10	109.61	65.94	24.78
Median	57	104	67	25
Std. Dev	24.34	45.09	14.46	17.39
Max	176	349	100	75
Min	26	38	35.4	0

Source: Ministry of Health Memo 131/cT p.1, November 1930: National Archives MH55/131.

Notes: 'Beds available per 100 deaths' refers to the average number of beds available per 100 tuberculosis deaths (all forms of tuberculosis). 'Patients treated per 00 deaths' refers to the total number of patients treated (all forms of tuberculosis) excluding observation cases per 100 tuberculosis deaths. 'Per cent staying more than 3 months' refers to the percentage of adult pulmonary tuberculosis patients who stayed over three months. 'Per cent staying over 6 months' refers to the percentage of those classified on admission as in Class TB + Group 1 who stayed over six months.

Table 1.7 **Summary Statistics Community Based Activities in 1929**

County Councils						
	A	***B***	***C***	***D***	***E***	***F***
Mean	62.24	57.06	16.04	298.92	489.90	328.59
Median	59	52.5	7	275	204	331
Std. Dev.	17.58	28.64	33.42	185.76	725.59	159.06
Max	97	161	203	1148	3760	710
Min	24	13	0	36	6	38

Metropolitan Boroughs						
	A	***B***	***C***	***D***	***E***	***F***
Mean	56.41	75.41	17.30	219.52	134.48	504.55
Median	57	66	11	189	93	485
Std. Dev.	16.99	31.82	16.72	121.31	106.18	281.24
Max	89	149	74	628	414	1593
Min	32	28	0.6	67	0	206

County Boroughs						
	A	***B***	***C***	***D***	***E***	***F***
Mean	65.56	80.08	27.36	239.49	93.68	385.40
Median	*68*	*71*	*9*	*224*	*38*	*345*
Std. Dev.	*18.03*	*39.40*	*37.73*	*174.73*	*197.94*	*206.05*
Max	*97*	*197*	*184*	*798*	*1620*	*1052*
Min	*25*	*20*	*0*	*0*	*0*	*23*

Source: Ministry of Health Memo 131/cT p.1, November 1930: National Archives, MH55/131.

Notes:

Column A serves as an index of the comprehensiveness of the Authority's scheme. It shows what proportion of the total known tuberculous population in each area, on a given date, was included on the dispensary register. (MH55/131, Ministry of Health Memo 131/cT p.1, November 1930). It is defined as the number of cases of tuberculosis on the dispensary register on 31st December per 100 on notification register.

Columns B and C throw some light on the efficiency of the clinical work by indicating the use made of bacteriological and X-ray examinations as aids to diagnosis and treatment. . (MH55/131, Ministry of Health Memo 131/cT p.1, November 1930). Column B is defined as the number of sputum exams per 100 new cases and contacts examined. Column C is defined as the number of X-ray exams per 100 new cases and contacts examined.

Column D gives a measure of the co-operation between Tuberculosis Officers and general practitioners. Defined as the number of consultations at homes or otherwise per 100 deaths from tuberculosis. (MH55/131, Ministry of Health Memo 131/cT, November 1930).

Columns E and F afford an indication of the amount of supervision given to patients in their

homes. Column E is defined as the number of other home visits by Tuberculosis Officers per 100 deaths from tuberculosis. Column F is defined as the number of visits by nurses or home visits per 100 patients on the dispensary register on 31st December. (MH55/131, Ministry of Health Memo 131/cT, November 1930).

Table 1.8 **Efficiency of Diagnosis in 1929: Summary Statistics**

County Councils	*Per cent on dispensary register diagnoses not completed by 31 Dec*	*Diagnosis not complete within 3 months*
Mean	65.56	4.49
Median	68	2.6
Std. Dev.	18.03	6.32
Max	97	32.4
Min	25	0
Metropolitan Boroughs		
Mean	56.41	3.55
Median	57	1.9
Std. Dev.	16.99	4.02
Max	89	17.6
Min	32	0.3
County Boroughs		
Mean	65.56	4.49
Median	68	2.6
Std. Dev.	18.03	6.32
Max	97	32.4
Min	25	0

Source: Ministry of Health Memo 131/cT p.1, November 1930 : National Archives MH55/131.

Notes: 'Per cent on dispensary register diagnoses not completed by 31 Dec' is defined as the percentage of all persons on the dispensary register on 31st December whose diagnosis was not completed. 'Diagnosis not complete within 3 months' is defined as the percentage of new cases and contacts whose diagnosis was completed within three months of first examination.

Table 1.9 The socio-economic and medical determinants of case rates (OLS)

	Dependent Variable ; Case rate per million population				
Living conditions and socio-economic status	OLS (1)	OLS (2)	OLS (3)	OLS (4)	OLS (5)
LAGPDENSITY	3.290*	3.582*	3.211*	2.392	3.684*
	(1.429)	(1.454)	(1.437)	(1.365)	(1.446)
LAGTHREEPROOM	8.384***	6.611*	8.468***	8.293***	4.807
	(2.312)	(2.844)	(2.320)	(2.320)	(3.023)
FAMSIZE	433.027*	374.566*	443.088*	444.864*	337.001
	(177.578)	(185.708)	(178.525)	(179.567)	(185.832)
MALEUNEM	-	9.900	-	-	21.371
	-	(9.256)	-	-	(11.472)
FEMUNEM	-	-	-5.763	-	-17.278
	-	-	(8.360)	-	(10.336)
Medical Provision					
DOCTORS	-14.051	-7.428	-20.017	-26.327	-17.644
	(52.342)	(52.679)	(53.154)	(52.068)	(52.683)
SNURSES	-2.037	-1.175	-2.439	-0.411	-1.379
	(6.945)	(6.988)	(6.983)	(6.999)	(6.942)
Administrative Efficacy					
COMP	4.172*	4.447*	4.172*	-	4.764*
	(2.064)	(2.079)	(2.068)	-	(2.074)
HOMEVISIT	-	-	-	0.241	-
	-	-	-	(0.140)	-
RURAL	-355.124***	-306.490**	-373.893***	-452.197***	-306.414**
	(103.084)	(112.62)	(106.815)	(106.557)	(111.864)
Obs	146	142	142	142	142
F-Stat	12.44	11.04	10.90	12.18	10.26
R-squared	0.394	0.399	0.396	0.389	0.412
Breusch-Pagan p-value	0.477	0.424	0.433	0.361	0.257
VIF (mean)	1.49	1.78	1.48	1.48	1.98
RESET p-value	0.566	0.910	0.369	0.194	0.700

Notes: Standard errors in parentheses. * P<0.05, **P<0.01, ***P<0.001.

Table 1.10 The socio-economic and medical determinants of death rates: robust OLS

Dependent Variable; Crude Death Rate								
Living conditions and socio-economic status	ROLS (6)	ROLS (7)	ROLS (8)	ROLS (9)	ROLS (10)	ROLS (11)	ROLS (12)	ROLS (13)
PDENSITY	1.10	-	-	-	-	-	-	-
	(0.59)	-	-	-	-	-	-	-
THREEPRM	4.99***	-	-	-	-	-	-	-
	(1.15)	-	-	-	-	-	-	-
LAGPDENSITY	-	1.90***	1.88***	1.91***	2.02***	1.86***	1.92***	1.86***
	-	(0.50)	(0.50)	(0.51)	(0.48)	(0.51)	(0.51)	(0.50)
LAGTHREEPROOM	-	2.97***	2.93***	2.98***	2.94***	3.01***	2.75***	2.86***
	-	(0.82)	(0.81)	(0.83)	(0.82)	(0.82)	(0.83)	(0.82)
FAMSIZE	75.87	121.20^	131.78*	119.38^	131.62*	120.23^	129.19*	128.84*
	(63.98)	(63.18)	(62.99)	(63.92)	(64.64)	(64.42)	(63.64)	(62.86)
Medical Provision								
DOCTORS	-19.02	-15.15	-13.07	-15.89	-13.54	-18.78	-13.27	-13.49
	(18.15)	(18.57)	(18.55)	(18.87)	(18.52)	(17.64)	(18.58)	(18.44)
SNURSES	-1.34	-1.57	-8.23	-0.81	-1.72	-	-7.94	-7.89
	(2.44)	(2.50)	(5.75)	(3.90)	(3.06)	-	(5.73)	(5.68)
MWIVES	-	-	-	-	-	-70.36	-	-
	-	-	-	-	-	(79.25)	-	-
XRAY	0.11	0.11	0.17	0.12	0.10	-	-	-
	(0.43)	(0.44)	(0.44)	(0.44)	(0.43)	-	-	-

Table 1.10 continued…

	Dependent Variable; Crude Death Rate							
Medical Provision cont…	ROLS (6)	ROLS (7)	ROLS (8)	ROLS (9)	ROLS (10)	ROLS (11)	ROLS (12)	ROLS (13)
SPUTUM	-	-	-	-	-	0.08	-	-
	-	-	-	-	-	(0.33)	-	-
BEDS	-	-	-	-	-	-	-0.58	-
	-	-	-	-	-	-	(0.68)	-
TREATED	-	-	-	-	-	-	-	-0.22
	-	-	-	-	-	-	-	(0.26)
Administrative Efficacy								
DIAG	2.91	2.92	3.08	2.91	3.06	2.74	2.82	2.86
	(2.21)	(2.25)	(2.24)	(2.28)	(2.29)	(2.27)	(2.28)	(2.24)
NODIAG	-	-	-	-	-	-	-	-
	-	-	-	-	-	-	-	-
COMP	-0.69	-0.54	-1.38	-0.57	-	-0.87	-1.21	-1.31
	(0.73)	(0.75)	(0.92)	(0.77)	-	(0.85)	(0.93)	(0.92)
CONSULT	-0.01	0.00	0.01	0.02	-	0.01	0.03	0.04
	(0.07)	(0.07)	(0.07)	(0.10)	-	(0.07)	(0.07)	(0.07)
HOMEVISIT	-	-	-	-	0.01	-	-	-
	-	-	-	-	(0.09)	-	-	-
Interactions								
SNURSESCOMP or	-	-	0.11	-	-	0.71	0.11	0.11
MWIVESCOMP	-	-	(0.09)	-	-	(1.17)	(0.09)	(0.09)

Table 1.10 continued…

	Dependent Variable; Crude Death Rate							
Interactions cont…	ROLS (6)	ROLS (7)	ROLS (8)	ROLS (9)	ROLS (10)	ROLS (11)	ROLS (12)	ROLS (13)
SNURSESCONS	-	-	-	0.00	-	-	-	-
	-	-	-	(0.02)	-	-	-	-
SNURSESHOME	-	-	-	-	0.00	-	-	-
	-	-	-	-	(0.01)	-	-	-
RURAL	-157.56***	-144.41***	-146.52***	-142.81***	-141.23***	-143.94***	-151.67***	-154.72***
	(36.37)	(37.67)	(37.38)	(38.64)	(37.88)	(37.27)	(37.34)	(37.44)
Obs	143	143	143	143	143	143	143	143
F stat	14.62	13.82	13.12	12.32	13.56	12.58	13.06	13.15

Notes: MWIVESCOMP replaces SNURSESCOMP in ROLS (11). Standard errors in parentheses. * P<0.05, **P<0.01, ***P<0.001.

Table 1.11 **The socio-economic and medical determinants of death rates: Quantile Regressions**

	Dependent Variable; Crude Death Rate							
Living conditions and socio-economic status	QR (14)	QR (15)	QR (16)	QR (17)	QR (18)	QR (19)	QR (20)	QR (21)
PDENSITY	0.79	-	N/A	N/A	-	-	-	-
	(0.50)	-			-	-	-	-
THREEPRM	4.81***	-			-	-	-	-
	(0.96)	-			-	-	-	-
LAGPDENSITY	-	1.73*			1.89***	1.65**	1.62**	1.42*
	-	(0.73)			(0.46)	(0.52)	(0.62)	(0.58)
LAGTHREEPROOM	-	2.80*			2.93***	2.95***	2.91**	2.74**
	-	(1.15)			(0.78)	(0.80)	(0.96)	(0.86)
FAMSIZE	98.14	140.74			127.86*	158.18*	113.56	133.48
	(57.67)	(89.75)			(60.57)	(63.66)	(76.09)	(71.64)
Medical Provision								
DOCTORS	-6.36	-1.18			1.83	-1.00	-2.38	0.10
	(11.54)	(18.53)			(12.35)	(12.58)	(15.62)	(15.14)
SNURSES	-0.09	-0.34			-1.82	-	-0.17	-0.94
	(1.95)	(3.12)			(2.14)	-	(2.68)	(2.81)
MWIVES	-	-			-	-14.25	-	-
	-	-			-	(36.16)	-	-
XRAY	0.37	0.13			-0.09	-	-	-
	(0.37)	(0.61)			(0.42)	-	-	-
SPUTUM	-	-			-	-0.35	-	-
	-	-			-	(0.35)	-	-

Table 1.11 continued…

	Dependent Variable; Crude Death Rate							
Medical Provision cont…	QR (14)	QR (15)	QR (16)	QR (17)	QR (18)	QR (19)	QR (20)	QR (21)
BEDS	-	-			-	-	-0.11	-
	-	-			-	-	(0.80)	-
TREATED	-	-			-	-	-	-0.45*
	-	-			-	-	-	(0.22)
Administrative Efficacy								
DIAG	0.37	1.05			0.89	1.57	1.16	-0.04
	(1.98)	(3.27)			(2.20)	(2.30)	(2.82)	(2.62)
NODIAG	-	-			-	-	-	-
	-	-			-	-	-	-
COMP	-1.27	-1.08			-	-1.01	-0.91	-1.01
	(0.65)	(1.08)			-	(0.77)	(0.95)	(0.88)
CONSULT	0.03	0.05			-	0.07	0.07	0.09
	(0.06)	(0.10)			-	(0.07)	(0.08)	(0.08)
HOMEVISIT	-	-			-0.01	-	-	-
	-	-			(0.03)	-	-	-
RURAL	-184.12***	-178.65			-152.64***	-190.39***	-181.71***	-201.27***
	(33.01)	(53.49)			(35.28)	(38.24)	(45.89)	(43.67)
Obs	143	143			143	143	143	143
Pseudo R squared	0.37	0.37			0.36	0.37	0.37	0.37

Notes: Standard errors in parentheses. * P<0.05, **P<0.01, ***P<0.001.

Table 1.12 **The socio-economic and medical determinants of death rates: Conditional Quantile Regressions**

Dependent Variable; Crude death rate			
Living conditions and socio-economic status	Q=0.25	Q=0.50	Q=0.75
LAGPDENSITY	1.90*	1.90***	1.87**
	(0.75)	(0.58)	(0.64)
LAGTHREEPROOM	4.51***	2.99***	3.03**
	(1.09)	(0.92)	(0.96)
FAMSIZE	-44.94	123.29	209.32*
	(90.29)	(135.09)	(104.21)
Medical Provision			
DOCTORS	-63.34*	1.53	0.66
	(27.92)	(43.30)	(48.17)
SNURSES	-0.44	-1.82	-4.74
	(1.72)	(2.02)	(3.60)
XRAY	0.29	-0.08	-0.17
	(0.35)	(0.35)	(0.44)
DIAG	4.01	1.33	1.67
	(3.64)	(3.95)	(4.48)
RURAL	-84.79*	-148.35***	-166.56***
	(37.43)	(34.94)	(52.65)
Obs			
Pseudo R squared	0.32	0.36	0.37

Notes: Standard errors in parentheses. * P<0.05, **P<0.01, ***P<0.001.

Table 1.13 The socio-economic determinants of the case fatality ratio (OLS)

Dependent Variable ; Case Fatality Ratio					
Living conditions and socio-economic status	OLS (22)	OLS (23)	OLS (24)	OLS (25)	OLS (26)
LAGPDENSITY	-0.0002	-0.0002	-0.0001	0.0002	0.0002
	(0.0003)	(0.0003)	(0.0003)	(0.0003)	(0.0003)
LAGTHREEPROOM	0.0001	0.0001	-0.0002	0.0001	0.0001
	(0.0005)	(0.0005)	(0.0004)	(0.0005)	(0.0005)
FAMSIZE	0.0268	0.0330	-0.0247	0.0417	0.0327
	(0.0399)	(0.0400)	(0.0393)	(0.0400)	(0.0417)
Medical Provision					
DOCTORS	0.0007	-0.0017	-0.0030	0.0084	0.0079
	(0.0117)	(0.0108)	(0.0085)	(0.0123)	(0.0122)
SNURSES	-0.0011	-	-0.0011	-0.0014	-0.0017
	(0.0016)	-	(0.0014)	(0.0016)	(0.0016)
MWIVES	-	-0.0265	-	-	-
	-	0.0216	-	-	-
XRAY	-0.0002	-0.0002	-0.0002	-0.0001	-0.0001
	(0.0003)	(0.0003)	(0.0003)	(0.0003)	(0.0003)
BEDSCASE	0.0067***	0.0067***	-	0.0064***	0.0067***
	(0.0007)	(0.0007)	-	(0.0007)	(0.0007)
TREATEDCASE	-	-	0.0028***	-	-
	-	-	(0.0007)	-	-
Administrative Efficacy					
DIAG	0.0019	0.0019	0.0004	-	-
	(0.0014)	(0.0014)	(0.0015)	-	-
NODIAG	-	-	-	-0.0015	-0.0014
	-	-	-	(0.0012)	(0.0012)
COMP	-0.0016***	-0.0016***	-0.0015***	-	-
	(0.0005)	(0.0005)	(0.0005)	-	-
CONSULTCASE	-	-	-	0.0000	-
	-	-	-	(0.0001)	-
HOMEVISITCASE	-	-	-	-	-0.0001
	-	-	-	-	(0.0000)

Table 1.13 continued…

Dependent Variable ; Case Fatality Ratio					
Other	OLS (22)	OLS (23)	OLS (24)	OLS (25)	OLS (26)
RURAL	0.0170	0.0189	0.0358	0.0317	0.0462
	(0.0230)	(0.0223)	(0.0303)	(0.0243)	(0.0246)
Obs	143	143	143	143	143
F stat	10.36	10.54	3.61	8.57	8.91
R-sqaured	0.44	0.44	0.24	0.39	0.40
Breusch-Pagan p-value	0.1987	0.1708	0.0000	0.7499	0.6353
RESET test p-value	0.0566	0.0522	0.6374	0.2959	0.2078
VIF mean	1.39	1.34	1.38	1.39	1.4

Notes: Specification 23 uses White's heteroskedastic consistent standard errors. Standard errors in parentheses. * P<0.05, **P<0.01, ***P<0.001.

Table 1.14 **Respiratory Tuberculosis: Crude death rate per mill pop at all ages: Summary Statistics**

	Counties				*Towns*			
	Mean	**Max**	**Min**	**Std. Dev**	**Mean**	**Max**	**Min**	**Std. Dev**
1920	735	1,072	554	110	1,399	597	974	198
1921	750	1,081	567	122	1,530	527	967	200
1922	756	1,078	594	105	1,364	453	972	192
1923	693	971	326	119	1,357	439	923	189
1924	697	1,007	272	118	1,431	506	946	181
1925	691	948	439	100	1,398	513	945	197
1926	626	883	428	89	1,261	436	866	176
1927	654	912	388	106	1,401	162	892	197
1928	608	894	385	96	1,634	179	837	213
1929	635	957	364	99	1,413	512	893	194
1930	601	872	369	104	1,364	364	830	195
1931	595	895	223	118	1,464	483	848	215
1932	556	818	403	82	1,447	355	788	191
1933	538	821	325	101	1,376	466	786	193
1934	502	763	281	97	1,268	370	721	167
1935	480	680	243	87	1,150	391	689	170
1936	468	688	352	77	1,145	343	668	151
1937	455	705	284	86	1,029	359	672	158
1938	417	637	280	72	974	303	621	156

Source: *Registrar General's statistical review of England and Wales* (Annual), London H.M.S.O.

Table 1.15 **Efficiency of Clinical Work**

County Boroughs	*Sputum*	*County Boroughs*	*X-Ray*	*County Councils*	*Sputum*	*County Councils*	*X-Ray*
Bottom Ten		**Bottom Ten**		**Bottom Ten**		**Bottom Ten**	
St Helens	20	Burton on Trent	0	Hunts	13	Isle of Ely	0
Birkenhead	26	Bury	0	Yorks North	16	Northumberland	0
Burton on Trent	34	Carlisle	0	Devon	20	Sussex East	0
Sheffield	35	Gr Yarmouth	0	Suffolk West	25	Bedfordshire	0.4
Stoke on Trent	35	Reading	0	Cumberland	28	Yorks North	0.6
Eastbourne	37	West Hartlepool	0	Salop	28	Yorks East	0.8
Middlesborough	40	Worcestershire	0	Isle of Wight	32	Devon	1
South Shields	40	Southend on Sea	0.3	Sussex West	32	Herefordshire	1
Barrow	41	Eastbourne	1	Worcestershire	32	Oxfordshire	2
Chester	41	Exeter	1	Yorks West	33	Somerset	2
Top Ten		**Top Ten**		**Top Ten**		**Top Ten**	
Southampton	120	Leeds	72	Dorset	74	Cornwall	18
West Hartlepool	131	Oldham	79	Lancs	79	Notts	19
Bolton	134	Southampton	85	Lincs (Lindsey)	79	Gloucs	22
Walsall	141	Stoke on Trent	86	Herts	83	Essex	23
Halifax	145	Brighton	89	Leics	95	Derbyshire	29
Southport	152	Bristol	91	Soke of Peterborough	99	Westmorland	32
Worcestershire	157	Southport	94	Lincs (Kesteven)	109	Northants	49
Blackburn	169	Plymouth	119	Warwickshire	110	Warwickshire	76
Birmingham	192	Birmingham	146	Rutland	114	Lancs	107
Bradford	197	Blackburn	184	Suffolk East	161	Cambs	203

Notes: Sputum is defined as the number of sputum exams per 100 new cases and contacts examined. X-ray is defined as the number of X-ray exams per 100 new cases and contacts examined.

Source: MH55/131, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Treatment Schemes of Local Authorities Analysis of progress, 1930-1933. Ministry of Health Memo 131/CT p.1, November 1930.

Table 1.16 **Use of Sanatoria: Summary Statistics**

County Boroughs	*Beds available per 100 deaths*	*Patients treated per 100 deaths*	*Per cent staying more than 3 months*	*Per cent staying more than 6 months*
Mean	60.17	121.35	58.31	24.19
Median	58	114	59.2	16.7
Std. Dev.	23.44	70.67	13.60	25.50
Max	122	605	94.4	100
Min	16	5	16.8	0
County Councils				
Mean	60.10	109.61	65.94	24.78
Median	57	104	67	25
Std. Dev.	24.34	45.09	14.46	17.39
Max	176	349	100	75
Min	26	38	35.4	0

Source: MH55/131, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Treatment Schemes of Local Authorities Analysis of progress, 1930-1933. Ministry of Health Memo 131/CT p.1, November 1930.

Notes: 'Beds available per 100 deaths' refers to the average number of beds available per 100 tuberculosis deaths (all forms of tuberculosis). 'Patients treated per 100 deaths' refers to the total number of patients treated (all forms of tuberculosis) excluding observation cases per 100 tuberculosis deaths. 'Per cent patients staying more than 3 months' refers to the percentage of adult pulmonary tuberculosis patients who stayed over three months. 'Per cent staying more than 6 months' refers to the percentage of those classified on admission as in Class TB + Group 1 who stayed over six months.

Table 1.17 **'Care in the Community'**

Home visits by nurses per no on register				**Home visits by Tuberculosis Officers per number of deaths**			
County Councils		***County Borough Councils***		**County Councils**		***County Borough Councils***	
Bottom Ten		***Bottom Ten***		***Bottom Ten***		***Bottom Ten***	
Warwickshire	38	Exeter	23	Gloucs	6	Burnley	5
Lincs (Lindsey)	76	Eastbourne	56	Somerset	30	Southampton	5
Bucks	105	Gr Yarmouth	89	Middlesex	53	Salford	6
Yorks East	134	Oldham	95	Northumberland	56	Stockport	8
Surrey	135	Canterbury	120	Dorset	63	Rotherham	9
Middlesex	138	Gateshead	132	Surrey	63	Bootle	13
Devon	155	West Ham	142	Southampton	68	Eastbourne	13
Suffolk East	169	Southend on Sea	153	Warwickshire	69	Smethwick	13
Wilts	173	Bournemouth	157	Sussex West	72	Halifax	15
Yorks North	175	Derbyshire	158	Northants	75	Wallasey	16
Top Ten		***Top Ten***		***Top Ten***		***Top Ten***	
Isle of Wight	428	Blackburn	633	Worcestershire	800	Darlington	168
Staffs	516	Kingston upon Hull	649	Lincs (Holland)	942	Tynemouth	189
Oxfordshire	519	Barnsley	715	Oxfordshire	966	Plymouth	207
Derbyshire	550	Norwich	752	Hunts	997	Burton on Trent	227
Herts	575	Carlisle	758	Devon	1063	Norwich	232
Suffolk West	584	Lincoln	776	Berkshire	1304	Northampton	262
Hunts	585	Brighton	804	Cambs	1328	Carlisle	278
Cornwall	612	Newcastle on Tyne	816	Norfolk	1475	Lincoln	313
Berkshire	629	Worcester	837	Rutland	3116	Southend on Sea	470
Isle of Ely	710	Plymouth	1052	Soke of Peterborough	3760	Exeter	1620

Source**:** MH55/131, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Treatment Schemes of Local Authorities Analysis of progress, 1930-1933. Ministry of Health Memo 131/CT p.1, November 1930.

Figure 1.1 ***Crude Death Rate per 1 million lving from respiratory tuberculosis in England and Wales***

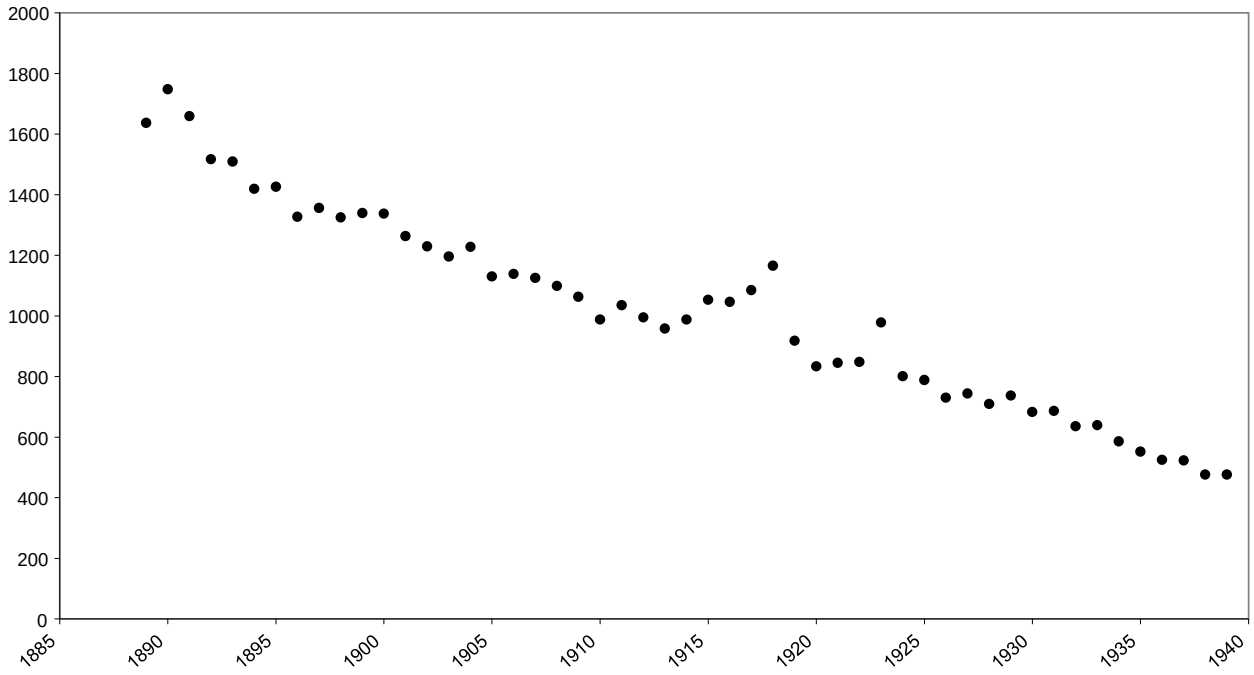


Figure 1.2 **Residual verses fitted plot using data derived from the Registrar General's Annual Review**

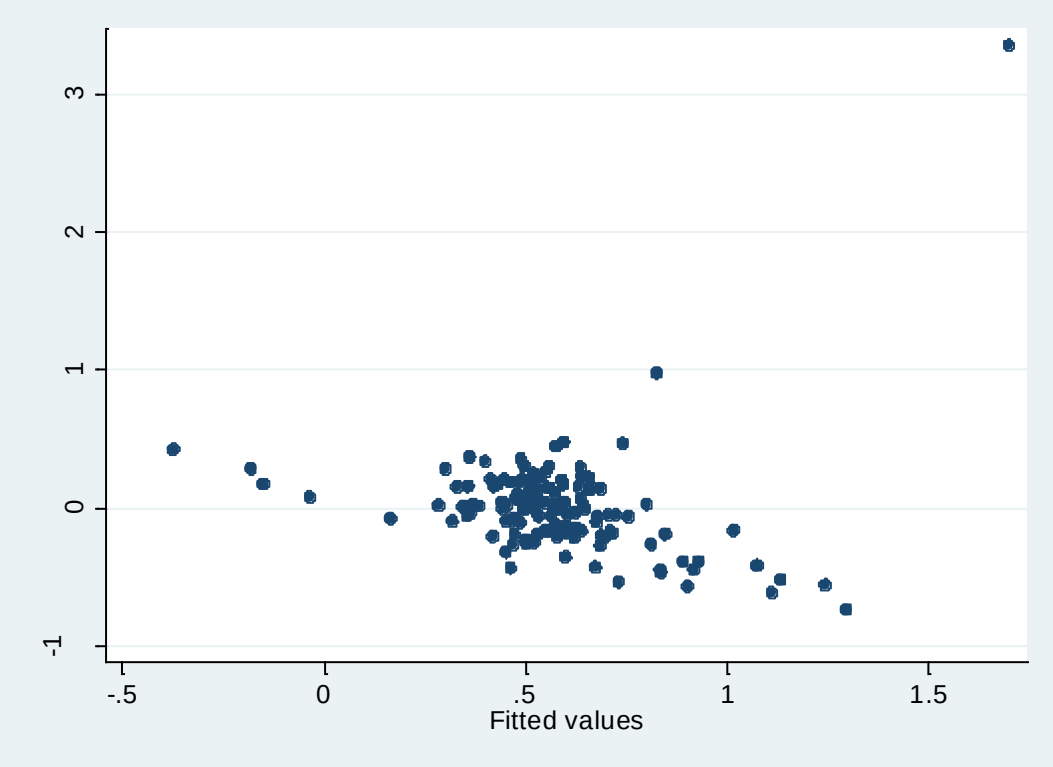
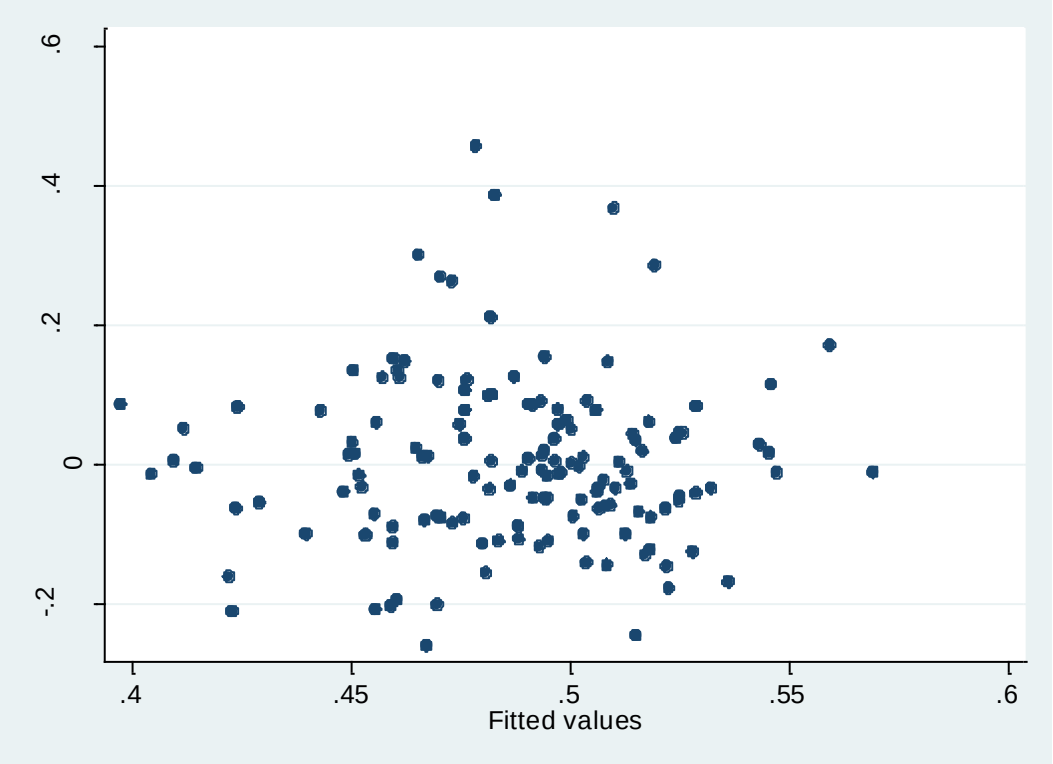


Figure 1.3 **Residual verses fitted plot using data derived from Ministry of Health's notification register**



2. Health Expectations and Health Achievements: Respiratory Tuberculosis in the Global Economy between 1950 and 1980: A Developing Economy Perspective

Sue Bowden and Alex Sadler

2.1 Introduction

In Chapters 2 and 3, we explore the respiratory tuberculosis problem on a global basis between 1950 and 1980. Chapter 2 adopts a more qualitative approach in its analysis of how and why the world became split into three at this time – in terms of the developed, the developing economies and the newly industrialising economies; whilst Chapter 3 extends the analysis and submits the various explanations for revealed differences to statistical testing.

Between the early 1950s and the 1980s, Tuberculosis could be prevented, identified and cured. It represents a unique period of time in history. In theory, all peoples of the world were offered the promise of release from this centuries old killer disease. This chapter is about why that promise was not realised.[6]

Chapter 2 contributes to the literature in three ways. First we have collated evidence on the morbidity and mortality problem over a wider range of countries than previously explored, with specific attention to the developed world. This allows us for the first time to identify a divided world in which some peoples were denied release from a

[6] From the late 1940s, with the advent of chemotherapeutic intervention, a prognosis of respiratory tuberculosis no longer necessarily implied death: in theory and in some parts of the world. The first such intervention was the US discovery of streptomycin from 1944; this discovery was closely followed by that of PAS also in the late 1940s (Dormandy, 1999, 211 and 366). Second, during the 1950s, medical research demonstrated the efficacy of joint PAS/Streptomycin therapy. The third discovery dated from 1952 with the identification of the superior curative and well tolerated effects of isonicotinic acid hydrizide (Isoniazid) (Daniel, 1997, 217-218; Dormandy, 1999, 237). 1980 provides a natural termination date for this research. In the 1980s, drug resistant tuberculosis emerged. This followed from problems in maintaining drug protocols in developing countries (WHO, 1998a, 4) and the decline in public health systems notably in the former Soviet Empire (WHO, 1998b, 35). It further followed from the simultaneous rise and diffusion of HIV/AIDS, a disease that destroys the immune system and increases vulnerability to opportunistic infections, including, importantly, tuberculosis (Barnett and Whiteside, 2006; Raviglione et al., 1996, pp. 87-96; WHO Regional Office for the Mediterranean, 1998c p.5).

disease which should no longer have been a threat of death. Second we have considered the extent to which development itself creates the condition in which the disease spreads by assessing how and why the emerging markets of the 1950s and 1960s experienced and dealt with the disease. Finally, we innovate by asking why, if medical science could prevent, identify and cure the disease, so many people in the developing world continue to contract the disease and die. We consider whether cost was the constraint or whether other institutional issues prevented that promise from being realised.

The implications of our findings are several-fold. First, when we take it as given that a key factor in economic growth is the ability of prime aged adults to work, anything which prevents this from happening is worthy of interest. Second, from a human development perspective, anything which constrains the ability of peoples to live a long and healthy life is of importance. Tuberculosis had a negative effect on both these issues. Third, in an era when drug resistant tuberculosis is emerging, the strategies available to health professionals in curtailing the spread of infection are of significance. We consider how health professionals in former years were constrained in their ability to deal with the disease. Fourth, if as we demonstrate below, development itself creates an increased risk of the spread of infection, then strategies have to be developed to deal with the disease risk that industrialisation and urbanisation creates.

2.2 Data and Findings

In order to ascertain the nature of the tuberculosis problem in the post-war period, we first found that existing datasets were overly developed economy biased and that, if we were to consider the global picture between 1950 and 1980, then a completely new dataset was needed. This dataset had to embrace not only a wider cross section of countries, but also had to include observations for both illness(morbidity) and death (mortality) if we to truly capture the nature of the problem.

In this section we report the findings from our newly created dataset explicitly designed to encompass a far wider range of countries than previously studied and to encompass both the illness and death problem that the illness posed. Our aim was to compile, using a variety of primary sources, up-to-date information to provide comprehensive global

information on different dimensions of the tuberculosis problem.

In the first instance, we used information contained in global, regional and national health reports and statistical yearbooks (for example, Pan American 1981(a), 1981b and 1981c; Pan American, 1983 and 1985; United Nations, Demographic, Annual; United Nations, *Statistical Yearbook for Asia and the Far East*, annual; WHO, Annual; India Statistics, Annual). For both mortality and morbidity we made revisions in the denominator using up to date United Nations data on population (United Nations, 2007).

2.2.1 Mortality

We measured the health significance of respiratory tuberculosis using different definitions. The first is that of mortality, whereby the total number of deaths from the disease in any one year is expressed in terms of deaths per 100,000 of the living population, essentially the crude death rate.

There are several issues appertaining to the reliability of the data for mortality (as for morbidity). First, the recorded information is a reflection of the sophistication of national health systems, the number of health professionals, the ability of these professionals and systems to diagnose and record disease and to specify the cause of death. It follows that for some countries, public health system deficiencies (as evidenced in the limited existence of such facilities to identify and record both cause of illness and cause of death), acted as a constraint on the reporting of reliable information. Second, there are issues relating to the quality of medical diagnosis and 'correct' nomenclature on death certificates, where indeed the authorities issued certified death certificates. Respiratory tuberculosis in particular, in the absence of radiographic or sputum test evidence, could be confused with other chest diseases. Third, as with HIV/AIDS, so with tuberculosis, social stigma deterred diagnosis. In some countries, as for example, Greece:

'pressure (was) brought to bear on doctors by relatives of deceased patients in their anxiety to escape from the verdict of death from tuberculosis, which Greek people still

regard as a stigma on the family as a whole' (McDougall, 1945, 844).

The stigma prevailed through our period of analysis (WHO, 1998b, 29-30). Fourth, there was a financial disincentive to be diagnosed in some countries. For example when X-ray was introduced for miners in New South Wales, Australia, those whose X-rays were positive were discharged without provision of medical care or economic assistance. The reaction was not surprising – men 'resolutely refused to have any more X-rays taken' (National Association, 1947, 203). This implies that the extent of the tuberculosis problem is underestimated by the recorded mortality data.

Our new estimates offer an expanded dataset by including a wide range of countries in Africa, Asia and the Americas. Our key finding is that respiratory tuberculosis accounted for a significant number of deaths globally as late as 1965 when measured in terms of deaths from respiratory tuberculosis per 100,000 living (Table 2.1). In Table 2.1 we present our findings for countries where the death rate was between 10 and 20 per 100,000 living peoples and over 20 per 100,000 living peoples.

Three further observations follow from Table 2.1. First, it is apparent that in the developed world, respiratory tuberculosis was not a significant cause of mortality. Second, in many developing countries, particularly in Africa, South America and the Eastern parts of Europe, the disease continued to be a major cause of death. Third, we find that respiratory tuberculosis was a significant mortality problem in those emerging economies in the 1960s, as for example Hong Kong, Japan, and Singapore. Development in other words had different implications as revealed in the tuberculosis mortality data. It remained a serious health issue in developing and emerging market economies but not in the developed world.

We next considered how important tuberculosis was as a cause of death. In 1974, WHO reported on the ten leading causes of death between 1969 and 1971, but only dealt with deaths from all forms of tuberculosis (WHO 1974 a and b). According to this analysis tuberculosis ceased to be a major cause of death by the start of the 1970s. The position of the emerging markets of this time is significant. In Hong Kong, Japan, and Singapore Tuberculosis was a major cause of death. WHO's findings, however, relied on a highly limited number of countries. We built on this work by collecting information on respiratory tuberculosis as opposed to tuberculosis per se and by increasing the sample

size. We found that as late as 1975, respiratory tuberculosis constituted a major cause of death. First, we note it was a major cause of death in the emerging markets of Singapore (26.8 per cent of all deaths), and Japan (13 per cent) and in the 'developing' parts of Europe including Yugoslavia (12.4 per cent of all deaths), Hungary (8.4 per cent), Bulgaria (6 per cent), and Romania (5.4 per cent). Second, the disease continued to be a major problem throughout the developing world despite receding in the developed. This is illustrated by its importance in Chile and Paraguay (23.3 per cent and 22.8 per cent respectively), Colombia (16.1 per cent), Belize (11.5 per cent), Venezuela (11.4 per cent) and Guatemala (12.3 per cent).

To the extent to which the disease constituted a health problem per se, we find that by the mid to late 1960s, the major health problems by descending order of magnitude were in Africa: tuberculosis, malaria, leprosy, schitstosomiasis, onchocerciasis and trachoma. Hence the observation, 'tuberculosis ranks with malaria and malnutrition as a main cause of illness and death in Africa, and in some African countries is even acknowledged to be the chief public health problem' (WHO Chronicle, (1960), Vol. 15, No. 11, p. 398 and 406). By the mid to late 1960s, the major health problems by descending order of magnitude in America were (save in the USA and Canada), tuberculosis, intestinal parasitosis, malnutrition, malaria, polio and accidents; and in Asia (excepting Japan) tuberculosis, diarrhoeal diseases, malaria, intestinal parasitosis, respiratory infections and leprosy. In the mid to late 1960s, tuberculosis was the second major cause of death (after accidents) amongst those aged 15 to 44, the prime aged income-producing agents, in the less developed countries; 'tuberculosis remains one of the principal causes of death in Latin America' (WHO Chronicle, (1962), Vol. 16, No. 5 (May) p. 154).

In terms of mortality, we find a tripartite division of the world: the developed economies whose citizens no longer needed to fear death from the disease; the emerging markets, where respiratory tuberculosis was a major cause of death and the developing world where it continued to be a major health problem. Economic growth did not bring health benefits in the short term; rather economic growth was associated with a rising mortality problem in terms of this disease. Interestingly, we note that 'developing' world status in this respect applied to parts of Europe.

2.2.2 Morbidity

Illness impedes the ability of income producing agents (that is, prime aged adults) to be active contributors to economic activity. Tuberculosis is an increasingly debilitating disease if untreated. The symptoms of active tuberculosis of the lung are coughing, sometimes with sputum or blood, chest pains, weakness, weight loss, fever and night sweats.

Data on morbidity become more reliable and more available from the 1960s. Morbidity returns relied on local diagnoses and returns reported to national health authorities: a medical and a bureaucratic need, which not all countries had. The number of cases reported depended not only on the availability of diagnostic facilities, but also on the extent to which case findings covered the population (WHO, Chronicle, 1961 (4), 140). For example, in India tuberculosis was only a notifiable disease in thirteen states in the early 1950s (Nair, 1954, 30).

WHO reported morbidity in different ways: incidence reflecting the number of new cases reported each year and prevalence reflecting the total number of cases of respiratory tuberculosis. In addition, prevalence *surveys* of large samples of the population, selected at random, were conducted which allow us to ascertain the extent of tuberculosis in a variety of countries at given points in time (WHO, Chronicle, 1959 (4), 169-170).

In Table 2.2, we report our findings on *incidence* of respiratory tuberculosis in the Americas, Asia and Africa at a given point of time. The data we have compiled have the advantage of being more comprehensive (cf. other earlier years) and to that extent more developing economy sensitive. Table 2.2 shows how respiratory tuberculosis continued to be a major health issue in the developing world and in the newly industrialising economies and *remained* a significant health issue in parts of the Americas, Asia and Africa.

Finally, prevalence surveys confirm the above. These surveys relied neither on reported cases nor on local diagnoses. From the late 1950s, WHO conducted prevalence surveys of large samples of the population. These surveys were particularly important in those

countries where reliable data were not available (WHO Chronicle, April, 1959, Vol. 13, No. 4, p. 169) - as in developing countries where health systems were not sufficiently sophisticated to monitor and collect data. Prevalence surveys provide point of time evidence for selective countries but not a source of information over time for a large number of countries.

The findings underline the extent of the problem in the developing world, and notably in Africa. Out of the total population in Kenya of around 6 million in the early 1960s, a prevalence survey found that there were about 110,000 definite or suspected cases of pulmonary tuberculosis in adults and older children (WHO Chronicle, 1962, No. 4, p. 122). A similar survey in Mauritania found that although 'for long it was thought that tuberculosis did not exist' in that country, there was indeed in 1964 a high incidence of the disease. (WHO Chronicle, July, 1965, Vol. 19, No. 7 p. 270). Prevalence surveys in Basutoland, Bechuanaland, British Somaliland, Gambia, Ghana, Liberia, Mauritius, Nigeria, Republic of the Congo, Sierra Leone, Somalia, Swaziland, Tanganyika, and Uganda were to confirm the extent of the health problem the disease posed in the late 1950s (WHO Chronicle, February, 1959, Vol. 13, No. 2, 82; WHO Chronicle, November, 1960 Vol. 15, No. 11, 406 and 408).

It was not just in developing countries that prevalence surveys alerted authorities to the nature of the problem. Prevalence surveys equally identified the extent of the health issue of the disease in the then emerging economies. In 1953, the Japanese government conducted its own prevalence survey. The findings revealed that 2,920,000 persons or 3.4 percent of the population needed medical treatment for tuberculosis (Yamaguchi et al., 1959).

To summarise, we find a tri-partite division: the developed economies where the disease had ceased to be a major health issue; the developing economies where the disease remained a major health issue and the emerging markets, which followed a Kuznets curve of rising tuberculosis health issues.

2.3 An emerging market economy problem?

A major finding from our dataset was that respiratory tuberculosis became a major health issue in the emerging market economies of the 1950s and 1960s. Those economies were characterised of course by increased industrialisation and urbanisation. The finding requires further examination not least since it implies that newly emerging markets today could face a similar problem. The following paragraphs explain how and why the newly emerging economies of the 1950s and 1960s experienced and, critically in terms of public health issues today, dealt with and in the event managed to avoid a respiratory tuberculosis health crisis.

Some of the highest recorded levels of respiratory tuberculosis mortality and morbidity in the mid to late 1960s were in the newly emerging markets of Japan, Hong Kong, and Singapore (Tables 2.1 and 2.2). The number of new cases in those countries far exceeded many if not most of the developing economies, while death rates were among the highest in the world. How do we explain these findings?

High levels of prevalence and incidence of the disease could be a spurious finding. As with HIV/AIDS, the incentive for an individual to be diagnosed with respiratory tuberculosis increased where effective treatment was more likely. An increase in recorded prevalence and incidence could be a reflection of the strength and effectiveness of health regimes, which in turn could be a reflection of the growing economic power of emerging economies. The newly emerging economy of Hong Kong provides such an example. The increase in the number of tuberculosis cases in Hong Kong in the late 1940s and 1950s has been attributed to the fact that more people presented themselves for assessment, 'because of growing confidence in modern medical treatment' (Phillips, 1988, 56).

A second explanation refers back to the historical literature. Scholars familiar with the historical literature will not find our finding surprising. There is a large literature linking tuberculosis mortality with increased overcrowding conditions associated with industrialisation and urbanisation in the West and in North America (Bates, 1992; Barnes, 2000; Bryder, 1988; Burnet, 1932; Daniel, 1997; Dormandy, 1999; Dubos and Dubos, 1952; Ott, 1996; Rothman, 1994; Smith, 1988; Teller, 1985). S*ustained* exposure to the bacteria is the crucial issue. Unlike influenza, only *prolonged* exposure to the bacteria led to infection. In the post-war period, as development with its consequential growth in urbanisation and industrialisation took place, tuberculosis

started to become a major health issue given sustained exposure in industrial workplaces and urban living environments. To that extent, high morbidity and mortality levels could reflect both a growing incentive to be diagnosed and a greater risk of infection which followed from prolonged exposure in an environment of industrialisation and urbanisation.

A third theme in the literature debated the relationship between economic growth and health. In part, this derives from an endogenous growth theory perspective which stresses the importance of the quality of labour (Lucas, 1988) and from a capability perspective (Sen, 1999), which sees the importance of peoples' ability to both acquire and use the capability of health as critical ingredients in human development. In *empirical* studies, debate centres on the causal relationship between health and economic growth. Does improved health (in terms of life expectancy and falling mortality trends) follow from economic growth per se or can public health mediate and instigate health gains at low levels of growth? Pritchett and Summers (1996) prompted the debate by arguing that income was more important than any other factor in explaining mortality decline. In response, Arora (2001) provided convincing evidence that health is not the result of, but a crucial ingredient to achieving economic growth. This article re-instated the notion that improvements in health can push economic growth and development complementing theses maintaining that health be treated as a luxury or by-product of that development. Likewise, Riley (2001, Chapter 4) argued that income per se is not enough to deliver improvements in health. The mediating effects of improvements in political and civic institutions are, he claims, crucial. The Arora/Riley thesis has been corroborated by Cutler *et al.* (2006, 110). They argued that economic growth could only improve health if it were accompanied by deliberate public action; 'health comes from institutional ability and political willingness to implement known technologies, neither of which is an automatic consequence of rising incomes' (Cutler et al., 2006, 116).

How then do our findings on Hong Kong and Japan fit into these debates?[7] We find a close relationship in both countries between the rise in economic growth and the decline in respiratory tuberculosis mortality and morbidity (Figures 2.1 to 2.4). The direction of

[7] We have insufficient data to conduct such analyses for Singapore.

causation is however unclear.

We further note that the decline in the respiratory tuberculosis problem undoubtedly reflected significant investments in the health services in those countries. In 1950, there were 463 people to each hospital bed in Hong Kong. By 1960, this had fallen to 380; it fell again to 240 people per bed by 1970. Achievements that are even more impressive were recorded in Japan. In 1950, there were 258 people per hospital bed; this had fallen to 111 in 1960 and 79 in 1970 (World Health Statistics 1983, Table19 and Statistical Yearbook for Asia and the Far East, annual, United Nations). In 1950, there were 9.22 physicians per 10,000 population in Japan. By 1960, this had risen to 10.83 and by 1970 to 11.33 physicians per 10,000 population. The achievement of Hong Kong was even greater in this respect. In 1950, there were 2.6 physicians per 10,000 population; by 1960, this had risen to 3.25 and by 1970 to 6.59 (World Health Statistics 1983 (Geneva, 1983) Table 17). In terms of specific interventions in relation to tuberculosis, all active cases in Japan by 1958 received chemotherapeutic treatment (Yamaguchi et al., 1962, pp. 36 and 37). As Japan offered real hope of cure, so voluntary visits to clinics for diagnosis rose (Yamaguchi et al., 1962, p. 41). BCG[8] campaigns, carried out annually among the young in Japan, led to a marked reduction in infection amongst younger age groups (Wakamatsu et al., 1969, 130; Yamaguchi et al., 1962, p. 44).

There is evidence that suggests that as those countries developed, peoples gained from the significant improvement in health services – and that those improvements did not lag far behind economic growth. The key finding is that the health problems caused by industrialisation and urbanisation were dealt with rapidly in both Hong Kong and Japan in a period of less than twenty years.

Two questions follow. One is the extent to which urbanisation or urbanisation accompanied by industrialisation created the health problem; the second is the extent to which health services were able to keep pace with the problem. Africa is a case in point. Iskander argued that the disease was 'liable to spread like wildfire because of rapid urbanisation' in Africa (Iskander, 1987, p.2). But the evidence suggests that the explanation is more complicated. The experiences of the Bantu in South Africa provide a telling example of the effect on tuberculosis morbidity and mortality of

[8] BCG (Bacillus Calmette-Guérin) is a vaccine against tuberculosis derived from *attenuated mycobacteria bovis.*

industrialisation/urbanisation.[9] Attracted by high wages, Bantu men migrated to towns in large numbers and into 'severe living and working conditions to which he is entirely unaccustomed' (Wiles, 1947, 17). Urban deaths rates from tuberculosis amongst the Bantu nearly doubled between 1939 and 1947 to the extent that 'in some industrial towns it is now over 1,000 per 100,000 – that is thirty times as high as the European rate'(Wiles, 1947, 16).

Two pieces of evidence suggest that industrialisation was the key factor. First, amongst Bantu men in the urban environment, those who worked as domestic servants had far lower rates of tuberculosis than those who worked as industrial labourers. The incidence of tuberculosis was 2.8 times as great among men who worked as industrial labour as against those who worked in the towns as domestic servants. Second, infection was just as high amongst Bantu in the native rural as in the urban environment, yet active cases were far higher in the latter: 'there must, therefore, be ample opportunity for infection in the reserves, but despite this the mortality is low' (Wiles, 1947, 18). Why then did Bantu men who migrated to towns and worked as industrial labour develop active symptoms of the disease and high mortality rates? The explanation, according to Wiles was 'a complete break-down of resistance due to inadequate food and severe living conditions' (Wiles, 1947, 19). Japan achieved its reduction in respiratory tuberculosis mortality and morbidity whilst experiencing an increase in its urban population from 46 per cent of the population in 1963 to 57 per cent in 1975. Hong Kong's health achievement was set against a background of a 86 per cent urban population in 1963 to that of 90 per cent in 1975. The crucial difference was economic development and the improvement in living standards[10] and the growth in health services in those countries – development and gains most developing countries did not experience.

Second, the issue is the extent to which health services were able to keep pace with urbanisation and industrialisation. We indicate above that both Japan and Hong Kong were successful in this respect. Yet signal improvements could be and were achieved in

[9] The term Bantu includes practically all the native tribes of South Africa. In 1947, the Bantu comprised 87 per cent of the 8,900,000 non-European population, comprising Bantu, Asiatic and coloured (sic) peoples of South Africa. The European population at the same time numbered 2,300,000.

[10] Living standards in terms of nutrition improved for the Japanese. Japanese peoples did not suffer from an inadequate calorie intake. In 1963, the average calorie intake for the Japanese was 2551 calories a day. By 1974, this had risen to 2719 calories a day.

some developing economies with little or no economic growth and development, given the will and the commitment of governments to commit funds to the problem. In an overview of health conditions in Nigeria in the 1970s and 1980s, Adegbola stressed the superior provision of medical services in urban environments compared with rural areas where 'the majority of health units were dispensaries and maternity clinics' (Adegbola, 1987, p. 40). Similar findings were reported in research in Brazil where again the critical factor was the extent to which health care services and social investment kept pace with urbanisation (Sawyer et al., 1987). A further example is that of Korea.

The Republic of Korea is an outlier among emerging economies in terms of its record of low mortality and morbidity. Data suggest that mortality rates were twice developed country levels in the 1960s and declined significantly thereafter. Other emerging economies in the region including Philippines, Hong Kong, China, Singapore and Japan, presented rates which were up to ten times the prevailing rate in developed economies. The Republic of Korea therefore is an interesting case; a country which experienced rapid economic development, yet not the concomitant tuberculosis problem. Some authors have argued (Seung, 1999) that the relatively good performance of the Republic of Korea into the 1990s can be explained by co-operation between the public and private sectors. By 1993, the private sector carried out roughly half of all tuberculosis treatment (47%). The principal policy component are subsidies offered to patients of private clinics which offer to cover up to 55 per cent of the cost of treatment, paid for through a national health insurance scheme (Seung, 1999, p.914). Public sector treatment is free for the patient.

Government in the Republic of Korea took the tuberculosis problem seriously with the establishment of the National Tuberculosis Program (NTP) in 1962, which attempted to improve cure rates for new cases of classical tuberculosis infection. Two National Tuberculosis Hospitals and numerous NTP health centres comprised the public offering. The private offering included private doctors operating in the large national hospitals, Korean National Tuberculosis Association chest clinics, and at private specialists. Engineering an environment in which private sector practices propagated led to the highly beneficial but unintended consequence that 90 per cent of the population lived within one hour of treatment and diagnosis facilities. In addition, public and private sectors assumed specialised roles. The public sector concentrated on the mass treatment of new cases, whilst the private sectors focused on more difficult cases of tuberculosis

and, after 1980, on the treatment of drug resistant tuberculosis. Republic of Korea may offer an example, therefore, of a country that did find a way to both achieve economic development and avoid high burden of tuberculosis disease. Medical and bureaucratic structures were developed to solve the tuberculosis problem despite the collapse in living standards following the Korean War.

The message of course for today is that as countries modernise, overcrowding in living and working conditions can inevitably follow - creating the conditions in which tuberculosis can spread. It further follows from our work that where and when countries dedicate resources to dealing with the problem, success can be achieved. It finally follows that economic growth creates the conditions in which the disease can be treated and cured: but crucially that health service investments create the conditions in which those diagnosed have the incentive to be diagnosed – and cured.

2.4 The limitations of 'developed world' medical science and public health innovations in a developing economy perspective

The creation of a new global database on the tuberculosis problem between 1950 and 1980 revealed a disturbing finding in terms of the developing world. We found that peoples in the developed world enjoyed a unique period of time when the disease could be prevented and cured. Peoples in the developing world were denied such a 'luxury'.

The question is why. Two explanations immediately apply. The first, following the 'in vogue' literature of the moment, is that there were institutional constraints (Acemoglu and Johnson, 2001 and 2002). The second is that these countries could not afford to purchase the medical technologies (drugs and apparatus) available in the developed economies. We consider both explanations in the following paragraphs. In terms of the institution approach, we turn the problem on its head. Instead of looking at what institutions were available, we discuss what needed to be done and why institutions could not deal with the problem. We further extend the definition of institutions to its most micro level to embrace the availability and training of health professionals, the logistical problems involving transportation of drugs and technology and the availability of reliable laboratories.

Tuberculosis posed a three-fold problem for international agencies, for health care professionals and for governments: how to take adopt and implement the appropriate interventions to a) avoid infection; b) prevent latent infection becoming active and c) prevent mortality amongst the active cases. In terms of the first two issues, the one obvious solution was to raise the standard of living in terms of working and living conditions and in terms of nutrition. Yet as Black has argued, for developing economies at this time 'There was no treatment for the victims, no sanitoria where they could be kept from infecting anyone else, nor any systematic improvement of housing or sanitation—the factors that had gradually cut down the tubercular toll in the West' (Black, 1986, 100). Hence, the generic solution to the problem did not apply in many parts of the world. Where economic growth was low and poverty endemic, and where modernisation led to overcrowding in living and working conditions, one might argue that the population could not wait for economic performance to improve to see a decline in the tuberculosis problem.

Here the approach is to specify, in medical and public health terms, that which could be done to reduce the risks of infection, to stop the disease becoming active and to reduce mortality under developing economy cost constraints. The methods of local health professionals and global non-government agencies such as WHO and UNICEF shared in these developing economy specific problems. They sought solution to the tuberculosis health problem by containing transmission of the disease, preventing transmission of the disease and curing the infected via fusion of the expertise of medical and public health. Our findings are based on a comprehensive review of the primary literature. We outline how and why medical and public health initiatives were constrained in a developing economy context and whether cost constraints rendered the tuberculosis problem unavoidable.

2.4.1 Containing Transmission

The first step toward containing transmission was to identify those infected. Three diagnostic tools were important. After 1895, X-ray provided the means to identify tubercular lesions although it was not until the 1920s that the utility of chest

radiographic examinations for diagnosis was recognised (Bhagi, 2001; Daniel, 1997, 45). X-ray may have been a basic tool in the developed world, but making its use effective was highly problematic in a developing economy context. The problem was threefold: the availability of X-ray units, the existence of laboratory facilities to interpret radiographs, and finally the existence of experienced personnel to take and interpret the X-rays. Developed world technology in other words could not easily be applied in developing economies. In developed economies, with relatively sophisticated health services, the X-ray was usually located in hospitals, serviced by trained personnel and appropriate laboratory facilities. Such a service was not the norm in developing economies where there were limited numbers of hospitals, often located miles from the afflicted. One solution was the travelling X-ray vehicle, which could travel to the afflicted, rather than expecting the afflicted to travel to the X-ray site (WHO, 1950, 7). In many developing economies, however, there was a lack of X-ray apparatus 'sufficiently sturdy, sufficiently portable or mobile, and sufficiently simple to operate and maintain' (WHO, 1960, 6). Radiography further required ancillary services (personnel and laboratory facilities) able to establish exact diagnoses (WHO, 1951, 6). The absence of these ancillary services compromised diagnostic tests in the developing economies. There was, in place, both a capital and a labour constraint on the realisation of effective X-ray interventions in the developing world.

Tuberculosis infection could also be identified by direct smear and/or culture from secretions or diseased tissue. In the 1950s, there were three laboratory diagnostic methods: a) the Mantoix test and b) sputum examination by direct smear or culture (Bhagi, 2001,118). Third, c) the development of tuberculin in 1890 by Koch allowed the infected to be distinguished from the uninfected via the so-called tuberculin test (Comstock, 1980, 445). As with X-ray, so with laboratory dependent identification of infection: developed world technology could not easily be translated in a non-developed economy context. The problem was the existence of laboratories and skilled technicians able to identify tuberculosis infection. Identification required health services and personnel, in particular tuberculosis laboratory services, able to conduct and interpret the tests. As late as 1982, laboratory services in many countries were unable to fulfil this service (WHO, 1982, 14-17). We find that despite the advances of medical and public health initiatives in the developed world, many economies lacked the health infrastructure (in terms of capital and labour) to provide effective diagnosis. In contrast to delivering vaccinations or administering treatment, identifying those who were

infected was not a simple matter of cost, but of the absence of sufficient development.

2.4.2 Preventing infection via vaccination

In terms of prevention, vaccination was the key intervention. In the post Second World War years, BCG was widely used. It is now acknowledged that BCG should not be used for children over one year of age because protection afforded by the diseases is considered to be variable and uncertain. Writing in 2006, Reingold et al noted that the efficacy of immunisation at birth against pulmonary tuberculosis group is probably no better than 50 per cent to 60 per cent (Reingold and Phares, 2006, 150).
There is, however, a significant rider relevant to this research. BCG was an inappropriate strategy where infection rates were *low* (Drolet and Lowell, 1962). Infection rates, however, were high in many of the countries we study in the 1950s, 1960s and 1970s and therefore BCG was an appropriate intervention. BCG was considered the primary intervention in developing economies.

Yet serious problems applied. One constraint related to the reliability of the vaccine: 'BCG produced in one laboratory looked very different from that produced in another laboratory' (Daniel, 1997, 140). As BCG came into general use, a number of different sub-strains were generated in a number of production laboratories (Milstien, 1993, 9). Hence, 'the protective efficacy of BCG, the most widely used vaccine against pulmonary tuberculosis, varied from 0 per cent to 80 per cent (Borgdorff, et al., 2002, 218.) Explanations for this variability included differences in the prevalence of infection with environmental mycobacteria and differences between BCG strains. A second problem related to the storage, delivery and transport of vaccines – as well as parents willing to have their children vaccinated. As Bloom et al argued:

'Delivering vaccines to patients requires functioning freezers and refrigerators (which in turn require a constant supply of energy); good roads and reliable transport to move the vaccines from port to clinic; clinics with access to people who need to be immunised; parents who know the value of vaccination; trained medical staff to deliver the dose; and sterile syringes' (Bloom et al., 2005, 22).

Micro studies revealed storage and delivery problems in a developing world

perspective. Togo exemplifies the problems such conditions created. National and regional cold storage conditions existed, but in rural areas, there was little or no electricity. Kerosene refrigerators were installed in rural areas but 'kerosene refrigerators proved fragile, needed clean fuel and required maintenance…' (Fitzgibbon, 1993, 15). Under kerosene conditions, the maximum duration of storage for vaccines was one month. If the vaccine were held in cold boxes or vaccine carriers, then the maximum duration of storage fell to 7 days and 24 hours respectively (Foster, 1993, Chapter 6, Table 6.6, 41). If, and when, mobile units had to travel more than 24 hours to reach populations, then the efficacy of the vaccine was negated. This would support modern analysis that argues that distance from the equator is strongly associated with vaccine effectiveness, such that the further from the equator countries are, the higher is vaccine efficacy (Roworth et. al., 2002, 246). Again, the evidence suggests that whilst developed economies benefited from the luxury of being able to store and deliver the vaccine, many economies enjoyed no such luxury.

In a developing economy context where many people lived in rural areas, a mobile unit equipped to take the vaccines to the people might have constituted an effective immunisation strategy. When well equipped (vehicles, spare parts and fuel), funded (per diem) and supervised, mobile teams were a highly efficient, cost effective strategy for vaccinating large numbers of people in a short time. Yet analysis of such activities in African countries as late as the early 1980s revealed significant problems. Mobile strategies in such countries were not sustainable over time because of the high cost of running mobile teams, the short cycle frequency required to immunise three-month cohorts and the relatively small number of target age individuals available at a given site eligible for vaccination (Foster, 1993, 38-39).

As a consequence mobile units could be both an expensive and a limited strategy where mobile units, as in Burkino Faso, were only able to visit nomadic or very sparsely populated areas at limited intervals. This meant that a limited number of children were fully immunised with all vaccines during their first year of life. In the late 1980s, whereas fixed facilities cost $7 per fully immunised child, mobile team delivery of *full* immunisation rose to $11 per immunised child (that is, for not just tuberculosis) (Gulaid, 1993, 75). A $4 dollar per child premium was a high cost in resource constrained developing economies – and this assumes that the mobile units were capable of storing and delivering the vaccine. Cost issues were compounded by the fact

that whereas developed economies largely used domestic sources of the vaccine, developing economies were dependent on imported vaccines. In the late 1960s, developing economies imported 80 per cent of the vaccines they used (Grzybowski, 1972, Table 8, 131).

The cost of protection against a number of childhood diseases was estimated to be between $1 and $7. Nevertheless, studies from the 1980s found the costs of fully vaccinating a child against diphtheria, tetanus, polio, measles, tuberculosis and pertussis varied widely between countries. These variations were attributed to delivery strategies (that is, whether the vaccination was delivered via fixed facilities, mobile services or mass campaigns), the local cost of personnel and vaccine procurement and distribution. The average cost for fixed facilities in low- income countries in the 1980s was US$15 per fully immunised child for the traditional antigens of diphtheria, tetanus, polio, measles, TB and pertussis (WHO, 2005, 12.).

It is not altogether surprising, given the above, that by 1968, only 2.2 per cent of the population of 14 African countries (that is 3,481,583 people vaccinated out of a total population for those countries of 163,360,000) received BCG vaccination. The coverage in Asia was only marginally better: among 24 Asian countries with a population of 1,087,556,000 only 27,850,381 people (2.6 per cent of the population) were vaccinated via BCG (Grzybowski, 1972, Table 1, 128). The chances of people acquiring the infection rose where protection was limited.

It is a reflection of the concerns of policy makers and the interests of health economics that it is only from the 1990s that the issue of the cost effectiveness, or value for money, in tuberculosis control appears in the literature. This reflected not only the concerns of policy makers and NGOs with ‘value for money’ concerns but also the new interest in Disability Adjusted Life Years and Disability Adjusted Life Expectancy. In a series of major contributions, looking at the position in the late 1980s and early 1990s, Murray evaluated ‘value for money’ in tuberculosis control (Murray, 1994). Murray found that the most cost effective intervention for tuberculosis was BCG added to chemotherapy for smear positive tuberculosis cases. This worked out at a cost per disability adjusted life year of $7 (Murray, 1994, Table 9.4, 201).

Short course chemotherapy and the inclusion of BCG in the expanded programme of

immunization were applied by the late 1980s. Short course therapy did not apply in the period we are examining. EPI only applied from 1974. Unfortunately, we cannot therefore apply the Murray estimates to prior periods. For the purposes of this research, comprehensive data sets distinguishing the components of capital and current expenditure on health expenditure are not available. World Health returns exist which give total amounts spent on health, but the returns themselves carry the health warning that they should not be used for comparative cross sectional analysis (WHO, 1970).

2.4.3 Curing the Infected

The advent of effective chemotherapeutic drugs offered explicitly curative treatment. In the mid-1960s, between eighteen and twenty four months of treatment with drugs was required to achieve stable bacteriological negativity. Initially it was thought that daily administration of chemotherapy was essential. In the 1960s, however, researchers found that therapy was just as effective when given two or three times a week. (Snider, 1994, 19) A ground-breaking study conducted by the Tuberculosis Chemotherapy Centre in Madras found that out-patient treatment was as effective in curing the ill as in hospital treatment. This implied to medical practitioners that treatment at home was now a viable and effective option. (Brimnes, 2008; Kamat et. al., 1959 and 1966).

For developing economies, the issue (as with BCG) was the cost and ease of administration of chemotherapy. The ease of administration was both facilitated and complicated by the knowledge that drug therapy could be provided effectively in the home. Facilitation derived from the fact that drugs no longer required hospitalisation. Complication derived from the concerns, despite the findings of the Madras study, that both completion and adherence to drug regimens were harder to monitor in a domestic setting (Pan American, 1981 (b) 3).

By contrast to short course chemotherapy, benchmarks for the cost of drugs and for the amount developing economies could devote to indirect expenditure (which of course includes the cost of drugs) do apply. In the mid-1960s, the cost per year of Isoniazid was estimated to be 6.25s a year (King, 1966, 21:8). If we take the cost of Isoniazid and eighteen months as the length of treatment, then curing one person via treatment of Isoniazid would cost 12.50s (£0.625). This translates into £9.01 per person as the cost of

effective cure in 2009 prices using the retail price index to measure the relative value of the pound in 2009 terms – a reasonable amount at first sight.

In 1963, Abel-Smith published his findings on the costs of health finances in six countries in the late 1950s (Abel-Smith, 1963).[11] His study has the advantage of the distinction he drew between capital and current costs with current costs incorporating staffing costs (medical and other), the costs of equipment and drugs, as well as replacement and maintenance costs. His estimates of current operating costs expenditure per head in 2009 prices were for the three non-developed economies of Ceylon, $38.4; Chile, $69.50; Czechoslovakia, $493.[12]

Herein, we take Ceylon as representative of the total indirect expenditure available for health in developing economies and then express our estimates of the cost of Isoniazid in terms of such budgets. In 1963, there were 9,977 confirmed cases of respiratory tuberculosis in Ceylon.[13] An annual course of treatment with Izoniazid for the 9,997 infected people cost £3,118. According to Abel-Smith, the total amount available per head for indirect costs associated with all health care needs in Ceylon were £2.4. At 6.25s for a year's treatment, Izonizaid counted for 13.02 per cent of the *total* amount available per head of *all* indirect costs in Ceylon.

Although Ceylon's commitment to health was in line with developed economies, the funds at its disposal were woefully inadequate in comparison. In 1957, Ceylon committed 4.3 per cent of gross national expenditure to the health services. In the same year, the USA committed 5.3 per cent of gross national expenditure to the health services, whilst in 1956 Sweden committed 4.9 per cent of gross national expenditure to the health services (WHO Chronicle, Vol. 17, No. 4 April, 1963, Table 3, p. 126). However, in 1957/58, expenditure per head in Ceylon was 5.3 US $, comprising 3.5 US $ indirect expenditure and 1.8 US $ direct expenditure. By comparison in the same year expenditure per head was 128 US$ in the USA, 58.3 US$ in Sweden in 1956 and 62.2 US$ in Czechoslovakia in 1959/60 (WHO Chronicle, Vol. 17, No. 4 April, 1963, Table

[11] Also reported in WHO Chronicle, Vol. 17, No. 4 April, 1963: 121-132.
[12] Conversions calculated using (http://www.measuringworth.com/ukcompare/result.php).
[13] World Health Statistics, 1963, Volume III, 1967.

5, p. 126).

Three considerations apply. First, tuberculosis was only one of the health problems Ceylon faced at this time. Ceylon also had a high infant mortality and general mortality rate (Abel Smith, 1963, Table 2, 38). Ceylon thus represents the problem many countries faced: tuberculosis was but one of many health problems. Second, drugs were only part of the indirect costs – indirect costs included, inter alia, the costs of all staff. Third, drugs were only part of the tuberculosis programme – BCG was also part of the tuberculosis programme and BCG itself was only part of a wider preventative health programme. When resources were limited and other health problems applied, dedicating 13 per cent of the total per head health expenditure to just one plank of a tuberculosis programme was undoubtedly beyond the reach of most developing economies. Finally, there is the obvious health warning that Ceylon may or may not be representative of the developing economy experience.

India illustrates the contextual problem well. For most developing economies, the critical problem was that the disease was but one of many health issues. India provides a telling example. Whilst WHO returns largely omit respiratory tuberculosis details for India, contemporary local work and statistical abstracts for India very specifically include observations on respiratory tuberculosis. In 1954, Nair estimated that the number of tuberculosis cases was likely to be about 2.3 million and, on the assumption that one out of five cases died every year in India, the number of deaths was about 500,000 a year (Nair, 1954, 50). Yet India also faced other critical health issues (Tables 2.3 and 2.4). Where cholera and malaria were major killers, tuberculosis was but one of many major health issues.

Governments face hard choices. It may be that a reluctance to address the tuberculosis problem reflected greater and more pressing health issues. In 1952, the tuberculosis adviser to the health services of India[14] made the telling remark that:

> ‘finances for dealing with the tuberculosis problem are completely inadequate,

[14] Dr. Peraketh Verghese Benjamin, Adviser in Tuberculosis, Directorate General Health Services, Government of India speaking at the Commonwealth Conference on Tuberculosis in India in July 1952.

> especially when we take into consideration not only other medical needs, but also the needs for development in all other departments of government including those urgently needed for food production' (National Association for the Prevention of Tuberculosis, 1952, p. 14).

To summarise, we suggest that medical science offered the means for identification, prevention and cure. These interventions, feasible in the West, were constrained operationally in the developing world by the costs of implementation (in terms of transport and storage), the lack of trained personnel and the multiplicity of health problems such countries faced. For many developing economies where resources were limited, and the health problem was infinite, the costs of identifying, preventing and curing tuberculosis were prohibitive. Deaths were avoidable in theory, but not always in practice.

2.5 Conclusion

This chapter has considered how and why there were revealed differences in the 'tuberculosis problem' between developed, developing and newly developing economies in the world, at a time when in theory medical science could prevent, identify and cure the disease. There are several key findings from this chapter. First, significant differences in patterns of respiratory tuberculosis morbidity and mortality globally indicate that between 1950 and 1980 economies experienced varying success in facing the tuberculosis challenge. Second, we note that development created its own health problems and solutions. Urbanisation and industrialisation provided an environment in which the disease spread but concurrent economic growth over the medium term created condition in which the disease could be controlled and cured. Third, we find that despite the signal gains from medical science and public health, the benefits of those gains were limited to developed economies. Finally, we find that the costs of preventing and curing respiratory tuberculosis were prohibitive in an environment of multiple health issues. The lessons for contemporary policy makers are clear: development can create the very conditions in which disease can spread, and whilst medical science and public health can provide the means if those means are adapted to developing economy conditions. If not, the tuberculosis – and surely other health - problems will remain. This chapter adopts an explicitly qualitative approach

because we wanted to explore what could – and was done – throughout the global economy in this period of time. In the next chapter we take the research one step further by undertaking statistical analysis to explore explanations for the revealed variations in the tuberculosis problem.

2.6 Appendix

Table 2.1 **Respiratory Tuberculosis Death Rates in 1965**

Countries categorised by their respiratory tuberculosis death rate per 100000 per population					
	Fewer than 10		Between 10 and 20		Greater than 20
Oceania		**Oceania**		**Oceania**	
Samoa	1.6	Soloman Isles	10.2	French Polynesia	31.2
Papua	1.7			Cook Islands	63.2
Australia	2.4				
Tonga	2.4				
New Zealand	2.9				
New Caledonia	6.6				
Fiji	7.5				
Guam	7.9				
Africa		**Africa**		**Africa**	
Guinea	0.2	South Africa Asiatic	12.0	Nigeria	22.3
Tunisia	0.3	Reunion	14.4	Tanganyika	28.5
Cameroom	0.4	Lesotho	15.0	Cape Verde	29.2
Ethiopia	0.4	Morocco	16.0	South Africa 'coloured'	59.4
Niger	0.4	Botswana	17.5		
Senegal	0.4				
Gambia	0.5				
Mauritania	0.9				
Chad	1.4				
Angola	1.8				

Table 2.1 continued…

Countries categorised by their respiratory tuberculosis death rate per 100000 per population					
	Fewer than 10		Between 10 and 20		Greater than 20
Africa cont…		**Africa cont…**		**Africa cont…**	
Mali	2.0				
Malawi	2.1				
Burundi	2.2				
S Rhodesia European pop	2.4				
Swaziland	2.4				
Rwanda	2.5				
Kenya	3.1				
United Arab Rep	3.2				
Libya	3.4				
South Africa White	3.7				
Zanzibar	3.7				
Madagascar	4.3				
Congo	4.4				
Ghana	5.2				
Mauritius	6.1				
Mozambique	7.3				
Egypt	9.1				
Americas		**Americas**		**Americas**	
Bermuda	2.0	Guadeloupe	11.7	Mexico	20.5
Bahamas	2.1	Uruguay	12.3	Guatemala	25.7
Greenland	2.6	El Salvador	12.7	Bolivia	28.8
Canada	3.2	Paraguay	13.3	Chile	36.5
Jamaica	3.6	Cuba	14.2		

Table 2.1 continued…

Countries categorised by their respiratory tuberculosis death rate per 100000 per population					
	Fewer than 10		Between 10 and 20		Greater than 20
Americas cont…		**Americas cont…**		**Americas cont…**	
USA	3.8	Venezuela	14.3		
Trinidad & Tobago	4.1	Argentina	14.7		
Barbados	4.3	Puerto Rico	16.6		
Antigua	4.8	Martinique	16.7		
Dominican Republic	6.2	Colombia	17.3		
Nicaragua	6.4	Panama	17.7		
Montserrat	8.3	Dominican Republic	18.0		
Costa Rica	9.7	Ecuador	19.7		
Asia		**Asia**		**Asia**	
Laos	1.3	Jordan	10.8	Japan	21.3
Syria	2.1	Kuwait	12.5	Singapore	29.8
Iraq	2.9	Sri Lanka	13.0	Hong Kong	31.2
Israel	2.9			Brunei	41.7
Iran	3.7			Korea Dem Rep	67.9
Turkey	8.6			Macau	69.9
Bahrain	9.9			Philippines	85.4
Europe		**Europe**		**Europe**	
Netherlands	1.4	Belgium	10.0	Romania	22.1
Iceland	1.6	Ireland	10.5	Yugoslavia	23.8
Denmark	1.8	Italy	11.9	Hungary	23.9
Norway	3.3	GFR	12.0	Portugal	27.1
Eng & Wales	4.2	Greece	12.0	Poland	36.0

Table 2.1 continued…

Countries categorised by their respiratory tuberculosis death rate per 100000 per population					
	Fewer than 10		Between 10 and 20		Greater than 20
Europe cont…		**Europe cont…**		**Europe cont…**	
Sweden	4.2	Bulgaria	12.4		
N Ireland	4.6	Czechs	12.9		
Malta	5.0	Finland	13.1		
Scotland	6.1	France	13.2		
Switzerland	7.4	Spain	16.1		
		Austria	18.0		

Sources: A Bulla, Global Review of Tuberculosis Morbidity and Mortality in the world 1961-1971, 2-38; WHO, *World Health Statistics Report*, Volume 30, No. 1 (1977), Table 4, 19; WHO, *Annual Statistics*, 1965-1970; United Nations, Population Division, annual *Demographic Yearbook.*

Notes: Countries in the column under 'fewer than 10' are those countries in which the death rate from respiratory tuberculosis per 100000 population is less than 10 per 100000. Countries in the columns under ' between 10 and 20' and 'greater than 20' are countries those in which the death rate from respiratory tuberculosis per 100000 population is between 10 and 20 and greater than 20 respectively.

Table 2.2 **Incidence of Respiratory Tuberculosis in 1967**

Africa		The Americas		Asia	
Upper Volta	12.6	Dominican Republic	12.4	Lebanon	12.8
Egypt	13.1	Jamaica	15.6	Muscate Oman	38.1
Benin	15.8 [a]	Bermuda	18.0	Israel	42.6 [e]
Cameroon	21.3	Bolivia	18.8	Syrian Arab Republic	45.3
Mozambique	23.3	Canada	18.8 [c]	Iran	66.8 [f]
Niger	25.1	United States	20.4 [c]	Sri Lanka	86.0 [g]
Malawi	25.7 [b]	St Kitts	22.8	South Viet Nam	93.1
Mali	26.3 [b]	French Guiana	26.3	Iraq	108.3
Central African Rep.	33.9	Costa Rica	35.2	Lao	111.4
Togo	42.6	Cuba	36.9	Bahrain	114.5 [g]
Chad	48.6	Surinam	38.9	Malaysia West	121.8 [h]
Kenya	52.8	Puerto Rico	42.3	Malaysia Sarawak	122.8 [g]
Angola	55.8	Belize	44.3	Brunei	133.3
Madagascar	56.4	Guadeloupe	56.6	Turkey	162.9 [i]
Ivory Coast	58.3	Uruguay	63.8	Singapore	168.7 [g]
Sudan	58.8	Bahamas	81.6 [d]	Kuwait	179.2
Gabon	60.7	Ecuador	85.6	Malaysia Sarah	210.0 [g]
Guinea Bissau	65.5	Honduras	90.5	Japan	239.0
Sao Tome & Principe	69.8	Colombia	95.5	Philippines	333.2
Zambia	73.2	Panama Canal Zone	96.2	East Timor	343.9 [j]
Rwanda	74.1	Dominica	100.0	Hong Kong	393.0
Reunion	76.3	Guatemala	113.6	Macau	495.5
Southern Rhodesia	76.8	El Salvador	145.5 [a]		
Senegal	80.7 [b]	Peru	151.2		
Congo	92.9 [a]	Mexico	313.0		
Ethiopia	103.1				
Tanzania	118.0				
Mauritania	124.5				
Comoros	149.2				
Gambia	184.5 [a]				
Lesotho	187.7				
Cape Verde	188.7				
Libyan Arab Republic	236.9				
Swaziland	256.8				
South Africa	361.7				

Notes: Incidence is defined the number of people per 100,000 infected by tubercle bacilli during one year. [a] Datarefer to cases reported to tuberculosis control units only. [b] In patients ad outpatients. [c] Data refer to new active cases. [d] Data refer mainly to New Providence. [e] Data

excludes recovered cases. [f] Data refer to selected urban areas. [g] Data refer to inpatients only. [h] Data refer to hospital and dispensary patients. [i] Data refer to new and old cases. [l] Data includes relapses.

Table 2.3 **Patients Treated in Various Hospitals and Dispensaries by Disease: All India**

	1949	1950	1951	1952	1953
Respiratory TB					
Number treated	226,723	214,826	315,654	333,723	212,550
Deaths	4,980	2,953	4,899	3,118	1,803
Cholera					
Number treated	21,728	262,737	34,554	34,938	72,992
Deaths	3,167	4,618	7,189	5,889	25,131
Malaria					
Number treated	8,686,357	5,665,056	8,029,776	6,802,736	5,084,618
Deaths	2,125	1,511	5,091	1,773	1,429

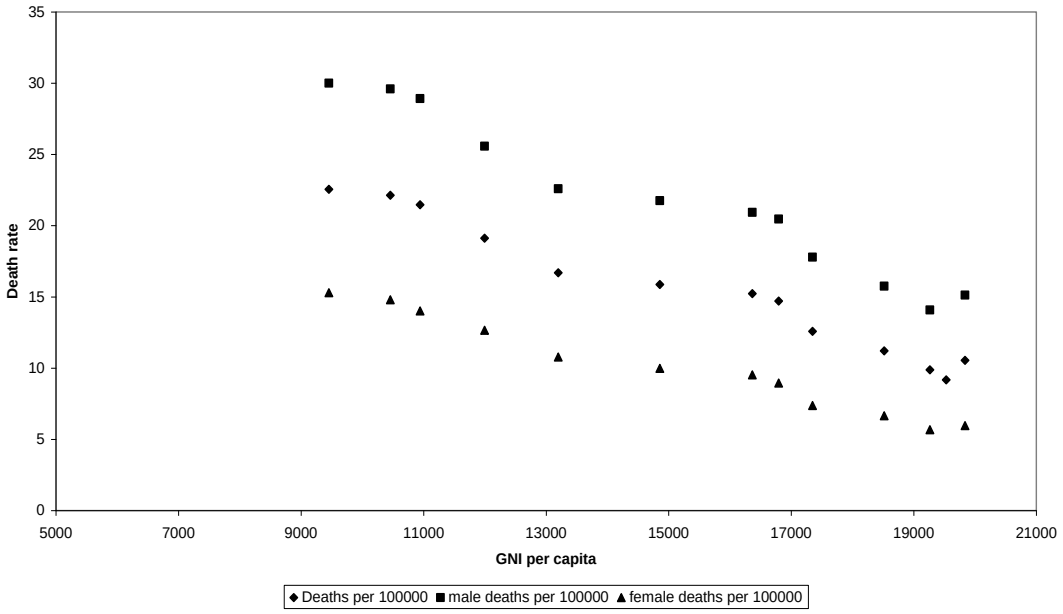
Source: Statistical Abstract, India, 1955-1956, No. 6 (New Delhi, 1957), Table No. 31, p. 69.

Table 2.4 **India: Patients Treated in Hospitals and Dispensaries for Respiratory Tuberculosis**

	1956		1978	
State	**Treated**	**Deaths**	**Treated**	**Deaths**
Andhra Pradesh	-	-	323,134	1,615
Assam	-	-	163,097	210
Bihar	17,785	-	-	-
Gujarat	-	-	44,268	920
Haryana	-	-	45,939	-
Himachal Pradesh	1,496	6	-	-
Karnataka	40,202	570	159,762	1311
Madhya Pradesh	119,923	327	-	-
Maharashtra	-	-	64,422	2,682
Manipur	598	6	-	-
Nagaland	-	-	5,184	32
Orissa	4,413	41	-	-
Punjab	30,351	114	43,051	582
Rajasthan	30,568	82	-	-
Tamil Nadu	156,799	844	-	-
Tripura	293	0	-	-
Uttar Pradesh	121,150	339	-	-
West Bengal	55,295	620	-	-

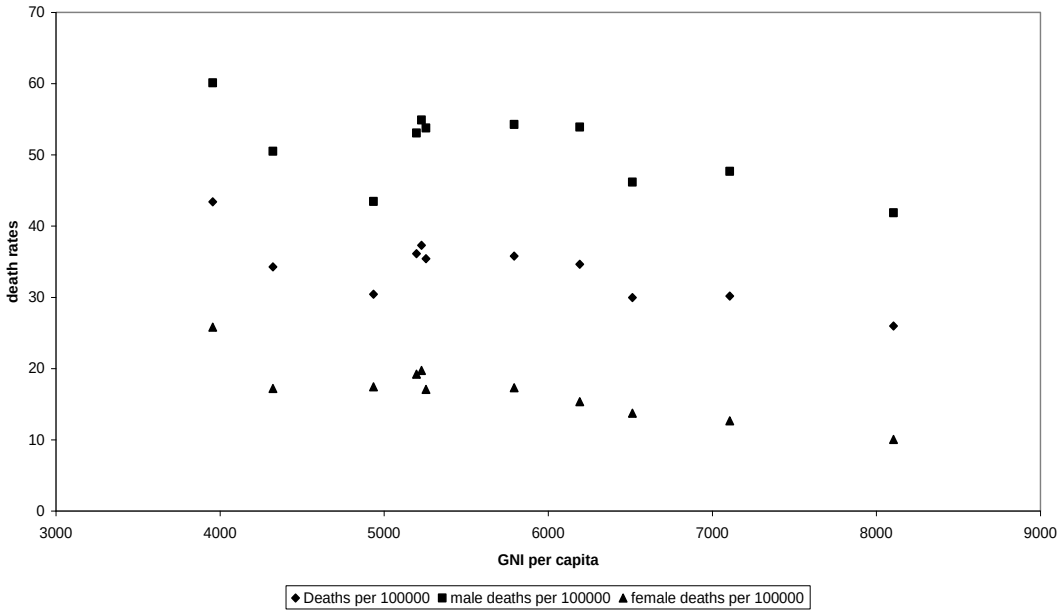
Source: India Statistics, Public Health Statistics, Table 232

Figure 2.1 **GNI per capita and respiratory tuberculosis mortality: Japan**



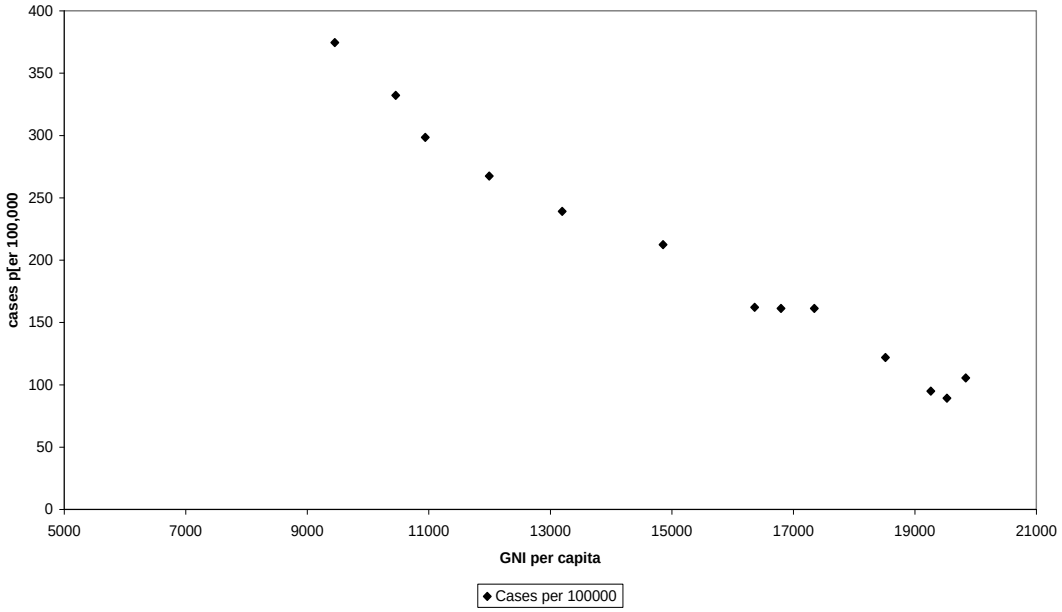
Notes: GNI per capita is GNI/cap (2000 $).

Figure 2.2 **GNI per capita and respiratory tuberculosis mortality: Hong Kong**



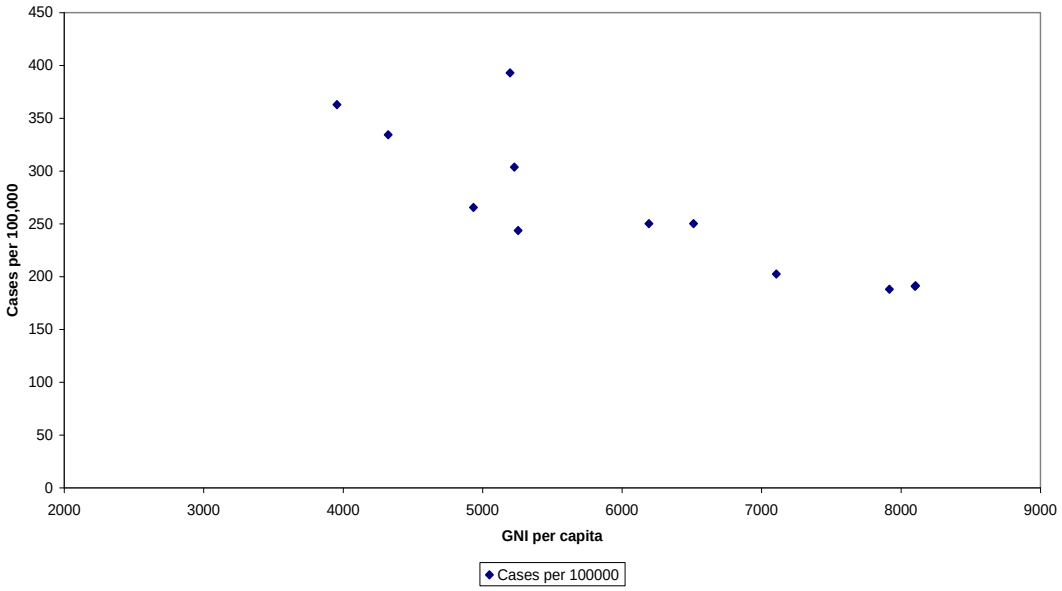
Notes: GNI per capita is GNI/cap (2000 $).

Figure 2.3 **GNI per capita and respiratory tuberculosis morbidity: Japan**



Notes: GNI per capita is GNI/cap (2000 $).

Figure 2.4 **GNI per capita and respiratory tuberculosis morbidity: Hong Kong**



Notes: GNI per capita is GNI/cap (2000 $).

3. Exploring Reasons for the Variation in the Global Tuberculosis Problem 1950-1980

Sue Bowden and Alex Sadler

3.1 Introduction

In Chapter 1 we assessed explanations for revealed differences over a cross-section of rural, urban and metropolitan areas in England and Wales in the inter-war period. This chapter identified the role played by a variety of standard of living and medical interventions in explaining those differences – at a time when no reliable chemotherapeutic cure existed. Chapter 2 then extended the analysis to identify differences between economies on a global basis at a time when in theory the disease could be prevented, controlled and cured. Adopting an overtly qualitative approach, Chapter 2 identified a number of micro-institutional constraints which helped explain why peoples in some parts of the world did not benefit from the promises of prevention and cure enjoyed in the developed world. This chapter also identified how and why industrialisation in the then NICs brought with it an increased risk of peoples being infected – but equally how and why some economies identified and dealt with the problem. In this Chapter, the analysis of the years between 1950 and 1980 is extended to encompass the application of statistical testing.

In Chapter 2 we hypothesised that a Kuznets curve type relationship could exist between historical rates of tuberculosis morbidity and mortality and the level of a country's development. Analogous to the relationship hypothesised by Simon Kuznets (1955) where inequality first rises with rising income, reaches some turning point and then falls thereafter, we hypothesise that as income rises, rates of tuberculosis disease and death rise, reach some turning point and then fall as economic development proceeds. This hypothesis is motivated by the extant literature described in chapter 2 which argues that tuberculosis disease is a reflection of worsening living and working conditions (from an epidemiological viewpoint) in the early stages of economic development. Initially income grows through rapid urbanisation and industrialisation. Improvements in health systems, working conditions and conditions in the home, however, have not historically kept pace to abate the health - related costs of

development. Thus, from a developing economy perspective, the problem is rooted in its technical sophistication and its ability to mobilize resources for abatement of tuberculosis disease in the workplace, the home, and in terms of medical provision. The increasing share of services in the economic activity can be viewed as a structural abatement mechanism. On the supply side, therefore, it is reasonable to expect that countries will pass through stages of development where at first the risks of contracting, developing and dying from tuberculosis increase and then diminish.

Demand side factors, may also be in operation. That is, when income is low, the demand for health may also be low. Initially, the generation of income at the margin is preferred to improvements in health. When income rises, so too does the relative importance of health and this in turn leads to demand for wider health coverage and more sophisticated health services. Living and working environments that are more conducive to good health may also be increasingly demanded as individuals become more educated, including better nutrition. The hypothesis that a tuberculosis morbidity / mortality Kuznets curve relationship exists is further motivated by descriptive statistics, provided in the next section, that reveal a tripartite divide; one in which developing countries have low levels of tuberculosis morbidity / mortality, rapidly industrialising countries have much higher levels and, finally, developed countries have low levels.

Establishing the Kuznets relationship in itself is not particularly informative or interesting. If an inverted U-shaped relationship between income and measures of tuberculosis disease is observed, there are good theoretical reasons to believe that the income measure is capturing omitted variables correlated with income that explain the tuberculosis Kuznets curve. Controlling for truly exogenous explanatory variables in the regression model, the tuberculosis Kuznets curve for an individual country is the locus of the equilibrium levels of supply and demand for abatement of tuberculosis disease.

Two issues arise. The first is, if a Kuznets relationship exists, has policy been an effective strategy historically to attenuate the rise in the burden of tuberculosis disease and accelerate the fall in NICs or elsewhere? The second concerns the specification of the model and a distinction between variables that are the endogenous consequences of growth and those which are exogenous. Endogenous variables include the number of doctors per capita, the share of the manufacturing sector in the economy, calorie consumption and urbanisation measures. Exogenous variables include the amount of

sunlight reaching the surface of the Earth on average in a given country, a time trend that proxies progress in technology available to all countries (as well as other variables correlated with time) and population density. If the purpose of the analysis is to assess both direct and indirect consequences of income growth, then endogenous variables ought to be excluded from the model.

In section 3.4.1 we test for the existence of a tuberculosis Kuznets curve without including additional regressors. We also distinguish between those countries that practised "good" anti - tuberculosis policies and those that did not. In chapter 2 it was argued that good policies include 1) administering BCG to those under one year of age, 2) treating tuberculosis disease with chemotherapeutic drugs for a sufficient period of time and 3) access to X-ray and laboratory personnel to accurately identify the infected. We calculate the proportion of children under one year of age that were vaccinated in 1963 (or nearest available year) using population tables from the 1965 United Nations demographic yearbook using the latest available census data 1955 – 1965. If a country attains a proportion ≥ 0.90 then that country has achieved the Global Immunization Vision and Strategy (GIVS) target of national immunisation coverage of 90 per cent. A country is therefore considered to have good policy if 90 per cent of children under the age of one are vaccinated. We consider countries which routinely offered more than 100 days of treatment in specialised tuberculosis facilities in 1969 to have good policy with respect to 2). We consider countries that offered both medical laboratory technicians and X-ray technicians in excess of 5 technicians per 100000 population in 1969 to have good policy in relation to 3).[15] If a country achieved 1) we consider it to have had good policies with respect to tuberculosis morbidity. If all 3 of the above policy criteria were achieved then the country is considered to have had good policies with respect to controlling mortality. Table 3.6 records whether countries achieved the above policies.

We are, however, not only interested in testing for the existence of a tuberculosis Kuznets curve, but also for determining the relative importance of the socio-economic determinants of tuberculosis morbidity and mortality. There has been extensive focus on the treatment of tuberculosis symptoms and the prevention of infection via vaccination

15 For countries with "good" policies with respect to preventing morbidity see first column of table 3.6. "Good" policies (1963 - 1975) with respect to mortality were followed by Australia, Austria, Cuba, Czechoslovakia, Federal Rep. Germany, Finland, France, Hungary, Iceland, Ireland, Israel, Japan, Luxembourg, New Zealand, Norway, Panama, Poland, Qatar, Romania, South Africa, Sweden, Switzerland, United States and Yugoslavia.

in the medical science literature. Very little attention, however, has been paid to the socio-economic determinants that underlie why individuals contract the illness and why they proceed to develop active symptoms of respiratory tuberculosis (Barnes, 2000). As we have already noted in Chapter 1, a feature of the disease is that those infected need not develop active symptoms. Chapter 1 further noted the literature which links activation of respiratory tuberculosis infection to inadequate nutrition, cramped working conditions common in newly industrialising regions, length of exposure to bacteria and exhaustion amongst new urban/industrial working populations (Wiles , 1947), Doull (1945).

We introduce, therefore, measures of overcrowding, industrialisation, medical provision and nutrition that get closer to the true variables of interest which explain inter - country differences in morbidity and mortality. Whether higher tuberculosis mortality rates in some countries are a reflection of differences in the institutional aspects of medical provision or in differences in standards of living and working conditions is not clear. There has been an association between overcrowding and tuberculosis disease in urban populations since at least the sixteenth century (Daniel, 1997, pp. 19-21; Dormandy, 1999, pp. 2-3). It was, however, industrialisation with its concomitant rise in urban population and overcrowding that facilitated the rapid spread of infection and kindled the interest of scholars. A large historical literature has emerged that links tuberculosis mortality with overcrowding, industrialisation and urbanisation in the West (Bates, 1995; Barnes, 1995; Bryder, 1988; Burnet, 1932; Dormandy, 1999; Dubos, 1952; Ott, 1996, Rothman, 1994; Smith, 1988; Teller, 1985). However, work that focuses on the non-developed world is limited with the exception of Packard (1989). Thus, elevated tuberculosis mortality has been attributed to low standards of living, cramped working conditions and inadequate treatment programmes, but without assessment of the relative importance of each factor (Johnston, 2003, p. 342).

In the post-war era the focus has been on the economic burden of both tuberculosis morbidity and mortality. Winslow (1951) has reported that deaths from tuberculosis in the USA cost that country more than one million years of future working life and $350 million each year for medical care and related services (Winslow, 1951, p. 13). Thus economies that rely on increasing inputs of labour suffer significant losses in potential output as a result of the tuberculosis mortality burden. Because tuberculosis is a disease which affects not only the elderly, but also income producing agents (as discussed for

the case of inter-war Britain in chapter 1) there exists a theoretical basis for endogeneity between the tuberculosis morbidity / mortality and income.

Finally, we investigate the role of nutrition in explaining variation in morbidity / mortality. The association of adequate nutrition with the prevention of the development of active tuberculosis disease and in the reduction of the risk of tuberculosis mortality has been documented in the historical literature. Higher mortality rates during the Second World War were attributed, not only to damage to health infrastructures and displacement of populations, but also to food shortages and malnutrition (Biraud, 1943/4, p. 572). That malnutrition and poverty are closely associated has led some authors to argue that poverty is chief among factors predisposing individuals to the disease (Grange, 1999; Hillbeboe and Holm, 1947). More recently Comstock (1980) has identified underweight as a risk factor for developing disease. This conclusion is supported by modern nutrition science which identifies a consistent log-linear relationship between body mass index and the risk of entering the disease state once infected (Lönnroth, 2008). The evidence suggests that those with higher body mass index have lower risk, relative to normal weight and underweight individuals, of going on to develop the disease.

To our knowledge, no statistical evidence has been provided to support either the existence of an historical cross country tuberculosis Kuznets curve, or socioeconomic factors that explain inter country differences in tuberculosis morbidity / mortality, except for in Bowden et al. (2014) which found within Europe in the post-war period, strong positive correlation between percentage of the population living in an urban environment and incidence of tuberculosis cases. In this chapter, we have collated a panel dataset for the period 1963 – 1980 with a view to a greater understanding of the economic mechanisms which procure disease activation for a cross-section of developed, newly industrialising and developing countries. These data also support an investigation into whether health provision and policy affected mortality rates. As such, we focus on the one period in time when, in theory, respiratory tuberculosis could be prevented, identified and cured.

3.2.1 Data sources

Measures of morbidity and mortality, country area, as well as physicians and beds per 100000 population, are derived from the World Health Statistics annual (WHS). The morbidity measure used is the number of newly reported cases per 100,000 of the population.[16] In Chapter 2 we presented our findings for 1967.

Data relating to both the growth rate of the urban population (measured as the percentage change) and the level of urbanisation (measured as the percentage of the population living in urban areas) are derived from The United Nations World Population Prospects (2007) revision (UN, 2007) for developing, newly industrialised and developed economies. Nutrition data are derived from the World Health Organisation's Global Database on Body Mass Index. GDP per capita (Int. 1990 GK$) and population data are derived from Maddison's historical GDP data set. We use data from the International Labour Organisation on paid employment by economic activity for the proportion employed in services and manufacturing.

Insolation data were derived from the National Aeronautics and Space Administration Surface Meteorology and Solar Energy database. Contemporaneous data were not, however available. For the purposes of this research we assume that insolation is a stationary process and that the average kWh per metre squared per day for the period July 1983 to June 2005 is representative of insolation for the period of interest. Table 3.1 presents representative data for total calorie consumption and urban population growth rate for the year 1967 for select countries.

3.2.2 Data Issues

There are a number of considerations related to interpreting the data on morbidity. Firstly, the number of cases reported in any country depends not only on the availability of diagnostic facilities, but also on the extent to which case finding covers the

[16] In the official WHO returns, morbidity was reported in different ways: incidence reflecting the number of new cases reported each year and prevalence reflecting the total number of cases of respiratory tuberculosis. In addition, prevalence *surveys* of large samples of the population, selected at random, were commissioned by WHO to ascertain the extent of tuberculosis in a variety of countries – but such surveys were taken at only limited intervals and hence merely yield point in time estimates of the illness problem (WHO *Chronicle*, 4, (1959), 169-170).

population. The number of cases reported depended first on the availability and reliability of diagnostic facilities, and the medical personnel able to interpret the findings - facilities and personnel not all countries had (Groth Peterson et al., 1959, p. 6). Second, it depended on the extent to which case finding covered the population (WHO Chronicle, 1961, p. 140). For example, in India tuberculosis was only a notifiable disease in thirteen states in the early 1950s (Nair, 1954, p. 30). Data on morbidity become more reliable and more available from the 1960s.

Thirdly, as with HIV/AIDS, where the chance of effective treatment and cure increased, so the incentive for an individual to be diagnosed with respiratory tuberculosis increased. Phillips, for example, attributed the increase in the number of tuberculosis cases in Hong Kong in the late 1940s and 1950s to the fact that more people would present themselves for assessment, 'because of growing confidence in modern medical treatment'(Phillips, 1988, p. 56).

Mortality data are also derived by WHO in WHS are also potentially problematic. The mortality variable recorded by WHS is the number of deaths per 100000 population in the calendar year. The reliability of these data depends on the existence of adequate institutional frameworks at a national level and the quality of reporting to WHO. It also depends on the quality of diagnostic equipment within countries and accurate nomenclature on death certificates, if these were issued. The possibility exists that developing countries did not have adequate chest radiographic facilities or laboratories for interpreting sputum tests to accurately diagnose respiratory tuberculosis as distinct from other chest diseases. The reliability of mortality data may furthermore be undermined in many parts of the developing and developed world because of the stigma generated by the association of tuberculosis with poverty. Thus the reliability of the data is challenged by the possibility that where death certificates were issued, doctors were pressured by relatives not to report a verdict of tuberculosis as the cause of death and so avoid social stigma. Hence, available data may underestimate the extent of the mortality burden (McDougall, 1945, p.844; WHO, 1998b).

To investigate the effect of industrialisation, independent of urbanisation, on the burden of tuberculosis we utilised the available historical sectoral employment records of the International Labour Organisation (ILO). Returns are only available on a yearly basis from 1969 for some countries, with availability improving towards the present.

Employment is disaggregated, for some countries, by activity into a) agriculture, hunting, forestry and fishing, b) mining and quarrying, c) manufacturing, d) electricity, gas and water, e) construction, f) wholesale and retail trade and restaurants and hotels, g) transport, storage and communication, h) financing, insurance, real estate and business services and finally, i) community, social and personal services. Our interest is in the proportion of employment in manufacturing and the proportion of employment in services specifically.[17] [18]

As table 3.5 illustrates, data series from some countries are not comparable with others. ILO sectoral employment data are reported by three measures that are not comparable. These are the total number of persons engaged in a particular sector or the total number of employees engaged or the number of insured persons. Most countries report only one of the above measures. In countries that report more than one measure of sectoral employment the totals differ substantially. In addition, these data are available from 1969 for select countries, reducing the length of the sample period by a minimum of 6 years. We address the issue of comparability by only using data from strictly comparable survey methods. We utilise data only from surveys that measured "employees" because this type of survey is the most numerous. We also extrapolate backwards in time to 1963 the proportions employed in manufacturing based on data available from 1969 onwards in order to avoid losing observations where data for other variables of interest are available.

To measure the proportion of the population employed services we wanted to sum f) wholesale and retail trade and restaurants and hotels, g) transport, storage and communication, h) financing, insurance, real estate and business services and i) community, social and personal services and divide by the total population. In addition, few countries include the full sectoral breakdown. Of the 93 countries in the sample,

17 Employment in manufacturing in Nigeria available from 1969 but unavailable for other sectors except mining and quarrying. Data refer to establishments with more than 10 employees. Employment in manufacturing is not disaggregated for Costa Rica. Employment in manufacturing available in Bulgaria, Cameroon, Czechoslovakia, Finland, Ghana, Hong Kong, Iceland, Japan, Panama, Puerto Rico,United States and Yugoslavia from 1969, Belgium, Brazil, Denmark, Dominican Republic, Hungary, Mauritius, Poland, Singapore, South Africa, Swaziland, Sweden from 1970, Cuba, Federal Republic of Germany, New Zealand, Nicaragua, Philippines, Portugal from 1971, Austria, Botswana, Gibraltar, Israel, Jordan, Kenya and Venezuela from 1972, Gambia, Suriname from 1973, Guatemala, Iraq, Peru and Uruguay from 1974, Colombia, Netherlands from 1975, Bahamas from 1976, Barbados, El Salvador, Italy from 1977, Bermuda, Luxembourg from 1978, Paraguay, Romania from 1980, Qatar from 1983, Macau from 1984, Kuwait from 1985. Data for Iceland and Nicaragua are derived from insurance records.

18 See Table 3.5 for countries available in ILO and the sources used by ILO.

only 17 adequately describe service sector shares in any of the periods 1969 - 1975 and many begin to do so only towards the end of the period. Unfortunately, even with the aid of extrapolation, data were too scarce to include in the analysis.

3.2.3 Descriptive statistics

In this section we motivate the hypothesis that a tuberculosis Kuznets curve existed within and between countries in the period 1963 – 1980. Figures 3.1 and 3.2 reveal a higher level and volatility in cases per 100,000 in the new industrialising and developing countries over the developed countries. Figure 3.3 indicates that for developed countries there is a steady downward trend over the twelve years in which year-on-year reductions in tuberculosis morbidity are typical. These figures illustrate to an extent the conventional wisdom; developing countries had lower rates of tuberculosis morbidity than their newly industrialising counterparts. These economies were dominated by agriculture and other primary production activities that necessitated greater dispersion of the population. Lower population density in addition to more frequent exposure to sunlight favoured lower morbidity rates. But unlike newly industrialising countries there is little evidence that in these countries the tuberculosis problem could be controlled.

Newly industrialising countries (NICs) are characterised by very high incidence of tuberculosis disease and for some there is the possibility of declining rates of tuberculosis. As mentioned, a feature of NICs is their rapid rates of urbanisation and potential for overcrowding. The observation that overcrowding facilitates the spread of disease was not only noted in wartime England (McDougall, 1952, p. 116) but the movement of peoples into urban areas as a factor that encouraged the spread of the disease was also apparent in Iceland (McDougall, 1952, pp. 122-4). The crucial issue here is *sustained* exposure to the bacteria. Unlike influenza, only *prolonged* exposure to the bacteria led to infection. As discussed in chapter 2 there is a literature that proposes that a similar effect was observed in developed countries in the West and North America. (Bates, 1992; Barnes, 1995; Bryder, 1988; Burnet, 1932, pp. 502-510; Dormandy, 1999; Dubos R & J, 1952; Ott, 1996; Rothman, 1994; Smith, 1988; Teller, 1985).

Though these societies offered greater material rewards in the long-run, health risks were greatly exacerbated in the short-run. For example, the lower incidence of morbidity amongst developing countries vis-a-vis industrialising countries supports a generalisation of the case evidence from the experience of Bantu men – industrialisation implies a greater risk of activation of latent infection. Hong Kong and Japan stand out, however, in their ability to reduce the impact of rapid industrialisation and urbanisation over time. The varied ability of NICs to control respective tuberculosis problems could due to the extent to which public health institutions kept pace with and dealt with changes in the epidemiological environment (Cutler et al., 2006).

The decline in *mortality* pre-dated the introduction of therapeutic drug regimens – in the developed world. Bowden et al. discuss the pre-war decline in mortality in Europe (Bowden, Jalles, Pereira and Sadler, 2014) and find that for many countries the decline began as early as the 1880s. In Ministry of Health reports discussed in Chapter 1, the decline in mortality may have begun in Britain as early as the middle of the nineteenth century. It is evident from Figure 3.6 that the selected developed countries were successfully managing a controlled decline in the number of tuberculosis deaths that mirrors the steady fall in cases observed over the period following the Second World War. Though the period for which comprehensive data are available is a relatively short - 13 years - the trend is towards low and steadily declining death rates in the selected countries. Where moderate increases in the case rate did occur in the developed world, for example in Belgium (see Figure 3.6), health systems were able to accommodate and death rates continued to decline. In these countries, the identification of infection, via smear and X-ray and prevention via vaccination as well as curative chemotherapies continued the decline that had begun in the late 19th century following improvements in working and living conditions, and crucially, diet.

Newly industrialising countries suffered the highest mortality burden in general. In these countries higher mortality rates followed from greater incidence but notably Figure 3.5 suggests that in the selected countries the decline in tuberculosis mortality proceeded despite volatility in case rates. That there is a decoupling of case and death rates over the period suggests that death rates were not only a function of case rates, but could be manipulated downward through public health initiatives. In Japan and Hong Kong in particular, declines in respiratory tuberculosis could be attributed to investment

in health services. The number of hospital beds per person and the number of physicians per person increased in both countries between 1950 and 1970 (World Health Statistics, 1983). In Japan, the wealth created during the process of rapid industrialisation facilitated implementation of effective treatment regimens whereby all patients received a regular supply of effective drugs (Yamaguchi, 1962). Chapter 2 investigated the specific problems of tuberculosis disease and mortality in an emerging market economy. Here it was argued that rapid urbanisation increased both the propensity to experience sustained exposure to the bacteria and also the incentive to be diagnosed and receive treatment. The controversy in the literature concerns whether rising incomes facilitate improvements in health and healthcare, or whether improved health is crucial to achieving growth (Arora, 2001; Pritchett and Summers, 1996; Riley, 2001). For these reasons we are interested in exploring in what way life expectancy, as a measure of health, is related to case rates, after having controlled for income.

Developing countries were unable to place sustained downward pressure on death rates. Figure 3.4 reveals that, although tuberculosis death rates were low in comparison to the newly industrialising countries, there is no predictable downward trend in mortality from year to year as in the developed world. This might be a reflection of data issues discussed above but equally it could be as a consequence of greater dispersion in these populations and hence the absence of industrialisation and urbanisation as vehicles for *mycobacterium tuberculosis* transmission.

3.3 Panel Data Methods

Given the above trends, the purpose of the following analysis is to investigate their link between tuberculosis disease in terms of both morbidity and mortality. Firstly, we hypothesise a tuberculosis Kuznets curve between tuberculosis morbidity / mortality and income per capita for historical rates of respiratory tuberculosis cases / deaths. Secondly, we hypothesise that the quality of a country's health policy will lead to changes in the shape of the tuberculosis Kuznets relationship. Thirdly, we hypothesise that urbanisation, the sectoral distribution of the economy, poor living and working conditions and poor nutrition, which facilitate the transmission and development of tuberculosis disease are the underlying explanatory variables in this relationship.

The data collected are panel and contains N = 93 countries for the years 1963

continuous through 1975 (T =13). The panel contains 29 countries in Europe, 22 countries in Africa, 14 in Asia, 19 in North America and the Caribbean, 7 in South America and 2 in Oceania. The panel data are "unbalanced" because for some countries in some years data are missing. As a consequence the number of groups and observations are subject to change when new variables, in addition to GDP per capita are used.

We began by estimating a model for the whole cohort including all countries for which there was at least one observation for all variables in the sample period. These estimations yielded large standard errors because of the paucity of the data and the unexplained volatility in the morbidity and mortality statistics recorded in some countries. For example, we dropped Gambia from the dataset following our subjective judgement that the data were incorrect[19]. Data were then re-estimated following the inclusion only of those countries with plausible statistics on case and death rates.

3.3.1 GMM estimation of the tuberculosis Kuznets curve

We considered the following quadratic logarithmic generalised dynamic specification (Arellano and Bond, 1991, p. 288)

$$lnH_{it} = \beta_2'(L)ln(GDPPC)_{it} + \beta_3'(L)(ln(GDPPC))_{it}^2 + \beta'(L)\boldsymbol{x}_{it} + \gamma_1 H_{it-1} + \gamma_2 H_{it-2} + \gamma_3 H_{it-3} + \gamma_4 H_{it-4} + \alpha_i + \varphi_t + \varepsilon_{it}$$

where Hit denotes either the log of the incidence of diagnosed cases of (CASES), or death rate from (DEATHS) respiratory tuberculosis per 100000 population in country i at time t. *GDPPC* and *(GDPPC)*^{2} are the log of both GDP per capita and its square. $\boldsymbol{x}$ is a vector of strictly exogenous variables, expressed in logs and unrelated to GDP per capita, but hypothesised to influence the tuberculosis heath outcomes. $\beta'(L)$ is a vector of polynomials in the lag operator, α_i is the country-specific time-invariant heterogeneity which drops out of the model when the estimating equation is first

19 For Gambia, in each of the years 1963, 1964, 1970, 1971 and 1972, only 1 person is recorded to have perished from respiratory tuberculosis whereas case rates range from 647 in 1969 to 72 in 1974 (WHS 1963 – 1980).

differenced, as well as other time invariant, but known variables, φ_t time dummies and ε_{it} is the idiosyncratic error.

Inclusion of a dynamic term changes the interpretation of the model from that of static fixed and random effects. Rather than the dependent variable being conditional only on the independent variables, it is now conditional on the independent variables and its own entire history. Because tuberculosis is a transmittable disease, therefore, we hypothesise that case and death rates in the current period, t, are dependent on case rates in the previous periods $t-k, (k \leq t)$. The transmission process suggests that population morbidity and mortality exhibits persistence through time. The available theory does not inform us sufficiently to arrive at any particular auto-regressive (AR) specification. The transmission process could vary between individuals in a population depending on the length of the latency period associated with tuberculosis infection. To test for the appropriate AR(p) structure, we experimented with different lag lengths, (up to a maximum of 4). Lag lengths in the dependent variables greater than 1 were insignificant when a single lag was included in the specification. The difference - Sargan test can be used to test the validity of a subset of instruments and is distributed asymptotically as χ^2 with degrees of freedom equal to the difference between moment conditions and parameters to be estimated. The model containing 4 lags of the dependent variables is the unrestricted model because it imposes fewer moment conditions. The models containing 3 lags, 2 lags, 1 lag and zero lags of the dependent variable contain more moment conditions and are hence, restricted models. The difference between the Sargan statistic for the unrestricted and restricted models is itself asymptotically distributed as χ^2 with degrees of freedom equal to the number of restrictions. The null hypothesis that the full instrument set is jointly valid. If the difference between the unrestricted and restricted values exceed the critical value, the the null hypothesis is rejected. Only the zero lag model is rejected at 95 percent significance for both dependent variables and so based on the observation that additional lags we proceed with an AR(1) .

We adopt an autoregressive-distributive (AD) lag structure of the form AD(1,0) after omitting insignificant dynamics in the regressors. We are therefore interested in estimating the parameters in the following quadratic logarithmic Tuberculosis Kuznets curve model

$$lnH_{it} = \beta_1 + \beta_2\ ln(GDPPC)_{it} + \beta_3\ (ln(GDPPC))_{it}^2 + \boldsymbol{x}_{it}\boldsymbol{\beta}$$

$$+\gamma H_{it-1} + \alpha_i + \varphi_t + \varepsilon_{it}$$

In order for the GMM estimator to be consistent, $E\{\varepsilon_{it}\varepsilon_{jt}\} = 0 \; for \; i \neq j$. That is, the disturbances for country *i* in time *t* must not be correlated with the disturbance in country *j* in time *t*. Because countries may experience common shocks in any particular period, we include time dummies to remove universal time related shocks from the errors.

The logarithmic transformation of both sides of the model equation is motivated by the potential for heteroskedacity in the errors when using untransformed cross country GDP and morbidity / mortality data. We are particularly interested in the coefficients associated with second order polynomial on GDP. A positive value of β_2 coupled with a negative value on β_3 suggests an inverted U-shaped relationship between H_{it} and GDP per capita. In the logarithmic model, the turning point (TP) is given by

$$TP = exp\{-\beta_2/2\beta_3\},$$

that is, the level of income at which the tuberculosis Kuznets curve reaches its maximum and after which begins to turn down (under the assumption that $\beta_2 > 0$ and $\beta_3 < 0$).

In the case of the within groups fixed effects model, for every group the value of each variable in each time period is differenced from the average value of each variable for all time periods. The purpose of the differencing process is to eliminated the problem of unobserved heterogeneity in the case where $E\{\alpha_i\varepsilon_{it}\} \neq 0$. However, even when eliminating the country specific effects through first differencing, OLS estimation of fixed effects will yield inconsistent estimates when lagged dependent variables are included as regressors. The inclusion of a lagged dependent variable in the above model implies that ε_{it} is necessarily correlated with ε_{it-1} even if the errors do not exhibit autocorrelation. The problem for estimation via OLS is that the within-transformed lagged dependent variable is correlated with the within-transformed error. This means that OLS estimation of γ is inconsistent for finite T as N $\rightarrow \infty$. First differencing the model above yields

$$\Delta lnH_{it} = \beta_2 \, \Delta ln(GDPPC)_{it} + \beta_3 \, \Delta(ln(GDPPC))^2_{it} + \Delta \boldsymbol{x}_{it}\boldsymbol{\beta}$$

$$+\gamma \Delta H_{it-1} + \Delta\varphi_t + \Delta\varepsilon_{it}$$

This not only eliminates the fixed effects, α_i, but unlike mean-deviations fixed effects, lags of the regressors longer than 1 are orthogonal to the errors. Following Arellano and Bond (1991, 1996) we suppose that lagged values of *H* can be used as instruments for current differences. That is, lagged values of *H* are uncorrelated with the contemporaneous change in the error term. For example, in period t = 2, $E\{(\varepsilon_{i2} - \varepsilon_{i1})H_{i0}\} = 0$ is a moment condition. In t = 3, $\Delta\varepsilon_{it}$ might be uncorrelated with both H_{i0} and H_{i1} and so on. Thus there are potentially $1 + 2 + 3 + \ldots + T - 2$ moment conditions available for GMM estimation that exploit the orthogonality of contemporaneous changes in the error term with past values of H_{it} alone. The valid use of these moment conditions to derive the GMM estimator is dependent on the absence of autocorrelation in the disturbances, aside from negative first order correlation in the differences regression. That H_{i0} is uncorrelated with $\Delta\varepsilon_{i2}$ clearly requires that $E\{(\varepsilon_{i0}\varepsilon_{i2})\} = 0$ for the levels case.

The GMM estimator can potentially be made more efficient through the use of additional moment conditions made available by the presence of additional regressors. We consider population density (population per square kilometre, lnPDENSITY) as strictly exogenous. Thus, in the case of population density, $E\{(\boldsymbol{x}_{is}\Delta\varepsilon_{it})\} = 0$ for each s and t. GDP is considered 'predetermined', in the sense that present and past values are considered independent of the contemporaneous disturbance and hence $E\{(\boldsymbol{x}_{it}\varepsilon_{is})\} = 0$ holds only for for $s \geq t$. GDP per capita is obviously not exogenous, but to some extent, once the current period arrives, it has already been determined, with error feedback only affecting future changes. The moment conditions available for use in estimation are therefore $E\{x_{i,t-j}\,\Delta\varepsilon_{it})\} = 0, j = 1, \ldots, t-1$ in each period, t. We therefore exploit these additional $(1 + 2 + \ldots T - 1)$ moment conditions. Finally, the T year dummies, φ_t are also exploited as instruments, but only contribute one column to the instrument matrix and therefore represent an additional $T - 1$ moment conditions. The total number of moment conditions used as instruments in each regression is reported in table 3.2.

In section 3.4.2 we investigate the effects that additional variables exert on the tuberculosis Kuznets curve relationship including the time varying policy variable, DOCTORS. Inclusion of additional variables might cause the tuberculosis Kuznets

curve to flatten out or for the relationship to vanish entirely. Unfortunately, because GMM requires that the estimating equation be expressed in first differences, intercept dummies representing particular policies among countries in the sample cannot be incorporated. Time invariant country policies of interest (such as, for example, dummies indicating whether or not the country has an under 1 year BCG vaccination policy) drop out of the model. We, therefore, compare the estimated tuberculosis Kuznets curve in separate regressions of countries with "poor" policies to that estimated for countries with "good" policies. Differences in the shape and turning points of these curves are reported in tables 3.2 with results for the whole sample.

3.3.2 Random effects, fixed effects and GMM estimation of the unrestricted model

In addition to testing for the presence of the tuberculosis Kuznets curve, we also wish to measure the effects of the underlying variables of interest. The tuberculosis Kuznets curve is hypothesised to be the product of the underlying variables and it is hypothesised that the tuberculosis Kuznets curve relationship will vanish once the model includes more proximate explanatory variables. Additional regressors included in $\boldsymbol{x}$, therefore, are the average number of calories consumed per person (CALORIES), the proportion of the population living in urban areas (URBAN) and its square ($URBAN^2$), and the proportion of the total population engaged in manufacturing (MANU) for the regression on CASES. The motivation to include both URBAN and its square in the analysis is the same as for including GDP per capita and its square. When urbanisation is low, case / death rates are also expected to be low. As development proceeds, and as the level of urbanisation increases, so too does the tuberculosis case / death rate. At a high level of urbanisation, the case / death rate may fall as urban services mature and the economy enters developed status. For the regression on DEATHS we also include the number of physicians per 100000 population (DOCTORS) to capture the quality of health care. Hence, $\boldsymbol{x}$, now contains variables that directly influence the supply of, and demand for better health.

As discussed in the previous section, it is a requirement of the model that dynamic terms be involved and this invalidates the use of standard panel data methods. For the sake of comparison we do, however, include fixed and random effects estimates by

excluding the dynamic term. The random effects model is consistent under the assumption that the individual group effects are uncorrelated with the regressors. The random effects model assumes that the individual country effects can be subsumed into the error term. Unlike differencing methods, such as mean-deviation within-groups fixed effects and first differenced GMM, random effects does not eliminate time invariant variables of interest by construction. Hence in the random effects regression we include the level of insolation (SOL). If random effects are not rejected, then the random effects estimator is efficient. The assumption of random effects is that the country specific effects are uncorrelated with the regressors such that country specific effects can be considered a component of the error term. We perform a Hausman test to discover whether the random effects permit GLS estimates to be consistent by comparing estimated parameter values to the consistent fixed effects estimator. A significant difference suggests that individual α_i's are correlated with the regressors and that the random effects estimator is inconsistent. As with difference GMM, α_i drops out of the model as do time invariant regressors.

It is possible that DOCTORS is endogenous. For example, it is natural to hypothesise that an increase in physicians per capita captures improvements in healthcare which will lower mortality rates. But it could also be proposed that greater mortality might provoke countries to employ more doctors. Therefore

$$E\{[\varepsilon_{it} \mid x_{it}, x_{it+1}, \ldots, x_{it+k}]\} \neq 0$$

where *k* are the number of remaining periods after t $(t = T - k)$ and *x* is the number of doctors. This violated the assumption of strict exogeneity. In words, the number of deaths in the present is correlated with the number of doctors in the future. Therefore, only past values of *x* are valid instruments and orthogonal to the contemporaneous error when computing the GMM estimator.

3.4 Empirical Results for GMM estimation of the tuberculosis Kuznets curve

Our contribution in this chapter is to make use of GMM methods to test for the existence of the tuberculosis Kuznets curve that was hypothesised in chapter 2. In table

3.2 we report GMM estimates for tuberculosis cases and deaths. The assumption that the errors idiosyncratic disturbances ε_{it} are serially uncorrelated is important. The GMM model was decided upon because the full disturbance containing the fixed effects was assumed autocorrelated. But the moment conditions used in table 3.2 require in addition that the idiosyncratic disturbance terms are uncorrelated with each other. If they are correlated, lags of the dependent variable longer than that of the order of serial correlation should be employed. We therefore use the Arellano-Bond test applied to the residuals in differences. Because $\Delta\varepsilon_{it}$ and $\Delta\varepsilon_{it-1}$ are related via the shared ε_{it-1} it is expected that the first order serial correlations will be negative. Indeed this is the case, although in GMM (4) this correlation is not significant. Thus to check for first order correlation in the levels equation, we test for second order correlation in the differences equation. That is, to test for correlation between $\varepsilon_{i,t-1}$ in the residual in difference, $\Delta\varepsilon_{it}$ and $\varepsilon_{i,t-1}$ in $\Delta\varepsilon_{it-2}$. Neither test, reported in table 3.2, exceeds the critical value required to reject the null hypothesis of no serial correlation.

The Sargan statistic fails to reject the null hypotheses that the over-identifying restrictions are valid in both GMM (1) and GMM (4) at the 5 per cent significance, although in GMM (1) the null is rejected at the 10 per cent level. It can therefore be seen from the estimates presented in table 3.2 that a historical tuberculosis Kuznets curve is indicated for the whole sample of countries between 1963 and 1975. The main result for the regression on CASES is the positive coefficient on lnGDPPC and the negative coefficient on its square, both significant at the 1 per cent level. The results imply that at the minimum level of income of $422.17 in the sample (the income per capita in Botswana in 1963), the elasticity of tuberculosis morbidity with respect to income is 3.00. This means that a 1 per cent increase in income is associated with a 3 per cent increase in case rate, a non trivial relationship. As income rises the elasticity falls. The elasticity is equal to zero at the turning point level of income. In our sample this was calculated as $4674, the income level approximately equal to Hungary and Poland. As income increases to around $9000, approximately the income of Ireland and Greece in the sample, the elasticity falls to -0.79. At the upper end of the income distribution are the USA and Switzerland with incomes of approximately $15000 and an elasticity of tuberculosis morbidity with respect to income of approximately -1.42.

The elasticity of tuberculosis mortality with respect to death rates exhibits a similar pattern. The elasticities at $422.17, $9000 and $15000 are 1.60, -0.11 and -0.39

respectively. The turning point occurs at an income of $7072, similar to Portugal and Hong Kong in the sample. An intriguing difference therefore emerges between the shapes of the tuberculosis Kuznets curve for morbidity and for mortality. A peak in the tuberculosis morbidity Kuznets curve at a lower income level than for the mortality curve suggests that factors that lead to increases in morbidity during development are more easily overcome than factors that lead to an increase in mortality. Equivalently, the results suggest that factors that attenuate tuberculosis morbidity are attained at lower income levels than those that attenuate tuberculosis mortality. Figures 3.7 and 3.8 plot the inverted U-shape Kuznets relationship derived from GMM (1) and GMM(4) for morbidity and mortality respectively across the sample range of incomes.

The coefficient on the lagged morbidity variable in GMM (1) suggests that the adjustment rate of tuberculosis morbidity is high at for the whole sample 87 per cent per year (1 – 0.13). This means that 87% of the difference between the long run and the actual rate of morbidity are eliminated in a year. The quantity for the mortality regression 83 per cent indicating a similarly rapid adjustment process in GMM (4). The path to equilibrium rates of tuberculosis morbidity, holding all other regressors fixed is little more than a year.

Columns 2 and 3 of table 3.2 reports the observed tuberculosis morbidity Kuznets curve for those countries with good preventative policies and those with bad preventative policies respectively. The estimated turning point in countries with bad preventative policies is $2802.3 higher than it is in countries with good policies. Furthermore in countries with good policies the speed of adjustment to long run equilibrium is slower (a short run adjustment of 64 per cent per year against 92 per cent for countries with bad policies). More rapid adjustment towards the long run equilibrium level indicates that countries have less control over the epidemiology of mycobacterium tuberculosis. That is, adjustment proceeds relatively unimpeded for the sample of countries where vaccinations are not effectively administered. There is greater *rigidity* of adjustment in countries with better policies.

Columns 5 and 6 of table 3.2 reveal a similar pattern for the tuberculosis mortality Kuznets curve. The turning point for countries with bad policies is $4057.41 greater than that for countries with good policies. This implies that countries with poor policies would need to attain the income of France in 1969, whereas countries with good

policies would only need to attain the income of Czechoslovakia in 1972. Again in good policy countries adjustment is relatively slow but in bad policy countries the adjustment parameter is insignificantly different from zero.

The estimated turning points ought to be interpreted with caution. The value of the turning point is highly sensitive to the value of the coefficient on $(GDPPC)^2$. To make the point, if the turning points are recalculated in GMM (1) and GMM (4) using the 95 per cent confidence lower bound of the estimated coefficient on $(GDPPC)^2$ then the turning points are calculated as \$3.78 x 10^8 and \$1.81 x 10^{35} respectively. Upper bound turning points are calculated as \$215.68 and \$108.62 respectively. Obviously, the observation of the tuberculosis Kuznets curve is heavily dependent on precise estimates of its curvature. Despite this, turning points remain plausible across the specifications presented in the next section.

3.4.1 Empirical Results for the unrestricted model

Table 3.3 presents results for the unrestricted model regressions for both CASES and DEATHS as the dependent variables. We report the random effects (RE) and fixed effects (FE) results alongside our preferred dynamic Arellano - Bond GMM estimates and test whether additional regressors alter the tuberculosis Kuznets curve pattern thus far observed for both tuberculosis case and death rates. Again Arrelano – Bond tests for second order correlation are performed on the differenced dynamic models and reported in table 3.3 and again these tests fail to reject the null hypothesis of zero correlation. The Sargan test fails to reject the null hypothesis that the over-identifying restrictions are valid in both dynamic specifications.

In general inclusion of the variables omitted in GMM(1) and (4) removed some of the upward bias in the estimates reported in table 3.3 and this has had the effect of reducing the estimated turning points. In both GMM(9) and GMM(12) the tuberculosis Kuznets curve peaks at a lower level of income and so the decline in tuberculosis morbidity and mortality begins at a lower level of income. However, the tuberculosis Kuznets curve does not vanish when explanatory variables capturing the causal factors proposed in the historical literature are included in the regression analysis. This suggests either that there are omitted variables that ought to be included in the analysis or that income

exerts a direct effect upon morbidity and mortality rates or possibly that existing variable measures are inadequate proxies to capture the underlying variables of interest.

Because the relationship between GDP per capita and H_{it} is modelled as a second order polynomial, standard interpretation of the long run elasticities implied by estimation of GMM (9) and GMM (12) is altered. The long run elasticity is a product of not only the estimated parameters but is also dependent on the value of GDPPC. Hence the long run elasticity, is given by

$$\frac{\delta(lnH)^*}{\delta(lnGDPPC)^*} = \frac{(\beta_{2i} + 2\beta_{3i}(lnGDPPC)^*)}{1 - \gamma_i}$$

where starred term H^* is the value that H_{it} takes such that $H_{is} = H_{is-1} = H^*$ conditional on $|\gamma| < 1$ and GDPPC* is the value GDPPC takes when fixed at point *t.* The above equation, therefore, gives the impact on the long run equilibrium value of *H** for a change in *GDPPC** at *t* that remains fixed from *t* onwards. According to GMM (9) the long run elasticity of cases with respect to GDP per capita for a country with the income of Botswana is 2.26. That is, a one per cent increase in income is associated with an approximately 2.26 per cent increase in equilibrium tuberculosis morbidity *conditional on Botswana's income of $422 per capita.* As income rises so the long run elasticity falls to zero. In the case of Yugoslavia a one per cent increase in income is associated with zero per cent increase in the equilibrium tuberculosis morbidity rate at an income of approximately $2780, which, naturally coincides with the estimated turning point. After the turning point the long run elasticities turn negative, implying that at higher levels of income, increases in income will shift long run morbidity downwards. Obviously, the closer γ is to 1 the slower is the adjustment. GMM(12) estimates a value of $\gamma = 0.17$. A similar pattern is again observed for mortality. Long run elasticities for countries with incomes under the estimated turning point are positive and for countries with incomes above the turning point are negative.

We observe that URBAN and URBAN^2 are statistically significant in the regression on lnDEATHS and have the expected signs in both the regression on lnCASES and lnDEATHS. According to the estimates in GMM (12) the short run percentage change in mortality with respect to a unit change in the per cent living in urban areas for a hypothetical country in which URBAN = 0 (an exclusively rural country) is given by

$1 - exp\{0.09\} = 1.094$. That is for a 1 unit change in the per cent urbanised, evaluated a zero, and holding the other covariates at their (geometric) means we expect a 9.4 per cent increase in death rates. The estimated general expression for calculating the percentage change in death rates with respect to a unit change in the percentage urbanised is $1 - exp\{0.09 - 2 \times (5.82 \times 10^{-4}) \times URBAN\}$. Setting this expression to zero and solving for URBAN yields a turning point of 77 per cent urbanised. A country with an urbanisation rate close to 50 per cent would expect a one unit increase in the percentage of the population urbanised to result in a 3.2 percent increase in the death rate but a country with an urbanisation rate of 80 per cent would expect a 0.3 per cent fall in the death rate. This non-linear (inverted U-shaped) relationship is similar to that already presented for the tuberculosis Kuznets Curve for GDP per capita where the turning point occurs within the range of the data. A similar pattern is observed in GMM (9) for case rates although the estimates are not significant.[20]

We used the variable MANU as a proxy for the level of industrialisation in a country as distinct from urbanisation which was used as a proxy for overcrowding. To this end, we would expect that greater industrialisation, (proxied by the proportion of the population employed in manufacturing), to assert an independent effect on rates of tuberculosis morbidity and mortality. Because of the paucity of data available for the sample period, the number of complete observations in regressions including MANU is decreased. The coefficients in RE (7) , FE (8) and GMM (9) are all the expected sign but insignificant. Estimates in GMM (9) predict that a 0.1 unit increase[21] in MANU will result in a 25.9 per cent increase in cases, but the estimated standard errors are very large.

In GMM (9) and (12) calorie intake is negatively associated with tuberculosis morbidity and is significant at the 1 per cent level in GMM(12). The estimates in GMM (12) suggest that a one per cent increase in calories will result in a 1.21 per cent fall in the tuberculosis death rate.[22] In both RE(10) and FE(11) a higher intake of calories is again

20 We also experimented by regressing the annual growth rate of the urban population in place of URBAN and URBAN^2 on lnCASES and lnDEATHS to test whether countries experiencing higher rates of urbanisation fared worse. In unreported regressions using the benchmark models GMM (9) and GMM (12) the annual growth rate of the urban population was found to be statistically and economically insignificant with a reported p-value = 0.643 and 0.635 respectively.

21 The proportion of the population employed in manufacturing (MANU) is bounded between 0 and 1. A 0.1 unit increase represents a change in the percentage of the population employed in manufacturing from 20 per cent to 30 per cent for example.

22 The coefficient in table 3.3 on lnCALORIES in GMM (12)_is -1.22. The effect of a 1 per cent change in calories is calculated as $(1.01/1\hat{}\beta_{lnCALORIES}) - 1 = (1.01/1\hat{} - 1.22) - 1 \approx -0.0121\ or\ -$

associated with lower mortality. The estimated elasticity in GMM (9) of CASES with respect to CALORIES is -0.67 but is insignificant. Comparison of GMM (9) and GMM (12) suggests both that lower total calorie consumption is associated with higher rates of tuberculosis activation and that once active, those societies in which calories are more scarce suffer greater mortality. The long run elasticity of DEATHS with respect to calories predicted by GMM (12) is $-1.22/(1-0.17) = -1.47$ which is smaller than predicted effect in the static random effects model.

As discussed, a difficulty of the present study is in the reliability of our morbidity variable. Those patients who knew that their chances of treatment and cure were higher were incentivised to report tuberculosis illness to a physician, where both effective diagnostic and remedial facilities existed. In other words, in those countries that did not have the means to diagnose and cure patients, tuberculosis mortality was likely to have gone under-reported. The number of physicians could act as a proxy for the development of the health system and, therefore, be negatively related to mortality. When included as an explanatory variable in the unrestricted mortality regression it was insignificantly negative. The sign is as expected, but the insignificance might be a symptom of the correlation of reporting with the development of the country's health system – the consequence of which is an upward bias in the estimated coefficient. This, of course, is speculation. It could also be that DOCTORS is not a good proxy for the efficacy of country health systems or that health systems are largely irrelevant in determining historical tuberculosis death rates. The last possibility seems unlikely given the results in section 3.4.1

It could be the case that urbanisation and nutrition are endogenous variables in that they both explain and are explained by cases of tuberculosis. For example, poor nutrition could be hypothesised to be the result of tuberculosis infection in cases where a tuberculous patient is no longer able to work and thereby afford adequate nutrition. In addition, those with tuberculosis may be incentivised to move to cities in order to receive treatment. For both these examples the historical and contemporary literature is clear that, for the majority of cases, causality is operating in the opposite direction.

One of the variables of interest, SOL is time invariant. The random effects model allows

1.21 per cent.

for the inclusion of time invariant regressors. A Hausman test failed to reject the null that the GLS estimates were consistent for either the regression on lnCASES or lnDEATHS. GMM methods made feasible consistent estimation of parameters when lagged values of the dependent variable were included as regressors, but at the expense of time-invariant regressors of interest. RE(7) and RE (10) both exhibit the expected sign but the estimates are not significantly different from zero. Unfortunately, both of these models are likely misspecified because they do not include a lagged dependent variable as a regressor.

3.4.2 Empirical results for the unrestricted model including source of calories

Some authors have suggested a number of key nutrients that are vital for defending the body against tuberculosis morbidity. Carson (2010) uses historical panel data from the United States to argue the importance of vitamin D in the prevention of tuberculosis mortality. In an important review piece, Johnston has argued that tuberculosis is a problem amongst populations which lack animal proteins in their diets (Johnston, 2003, p. 337). We therefore asked whether specific diets rich in any particular food group are important for the prevention of tuberculosis. We considered the importance of four food groups; cereals (CEREAL); fruits, vegetables, pulses, nuts (FRUIT); meat, fish, milk and eggs (MEAT) and oils, fats and sugars (OILS) in explaining tuberculosis morbidity and mortality. Each category of foodstuff is measured by its calorie contribution and converted into natural logs. Results are reported in table 3.4 using the benchmark regressions GMM (9) for morbidity and GMM (12) for mortality. Table 3.4 omits reporting of coefficients and standard errors for the other independent variables.

We are particularly interested in the elasticities reported for MEAT because of the preoccupation in the literature concerning the importance of animal protein-calories. The estimated elasticity of the death rate with respect to MEAT is 0.28 and significant at the 0.1 per cent level. This surprising observation is coupled with an elasticity on fruit which is negative, -0.36 in the regression on lnDEATH. In the regression on lnCASES we do not observe significant results. Calories derived from oils, fats and sugars or calories derived from cereals exert neither a statistically significant nor economically significant effect on either case or death rates. The economic significance, however, of

fruit, vegetable, pulse and nut consumption is high. The results imply that an increase in fruit consumption of one per cent is associated with approximately a 0.36 per cent decline in the death rate.

3.5 Discussion

The following discussion contextualises the empirical findings within the body of existing research. Section 3.5.1 discusses the observation of the tuberculosis Kuznets curve. Section 3.5.2 discusses contribution of the above findings concerning the importance of nutrition. It is asked to what extent the findings are corroborated by the existing literature and in what ways the new findings contribute. Section 3.5.3 discusses the extent to which medical provision was an effective strategy to reduce countrywide mortality rates during a period in which effective curative treatments existed. Section 3.5.4 discusses the econometric limitations encountered when investigating whether insolation is an importance factor that explains tuberculosis morbidity and mortality using panel methods. Finally, section 3.5.5 discusses the extent to which our findings support the argument that urbanisation and cramped working and living condition provided the means by which the bacteria could spread and data limitations.

3.5.1 The tuberculosis Kuznets curve

Simon Kuznets (1955) originally hypothesised an inverted U-shaped relationship between income and inequality. Although now dismissed on empirical grounds (Fields, 2001) his eponymous curve was tested in other areas, especially in studies looking at the relationship between economic development and environmental degradation.[23] We hypothesised in chapter 2 that an historical tuberculosis Kuznets relationship did exist and have found evidence to suggest our hypothesis was correct. In chapter 2 we indicated that in terms of both morbidity and mortality, descriptive evidence suggested that the emerging economies of the time were following the rising portion of the tuberculosis Kuznets curve because of the deterioration in socio-economic conditions typically experienced during the early stages of development. The surprising finding in this chapter was that the tuberculosis Kuznets curve relationship persisted when controlling for the supposed underlying causal variables discussed below. In chapter 2

23 See Stern (2004) for an overview.

we discussed the literature that argued that income was the most important factor in explaining mortality decline and the literature that asserted that income growth alone is not sufficient to promote improvements in health. In the light of the results here, it is not clear whether either position is supported.

In chapter 2 we also indicated that some countries had better tuberculosis policies than others. We argued that countries with effective strategies for containing transmission, preventing infection via vaccination and curing the infected would fare better. After quantifying these differences and splitting the sample accordingly it was discovered that the turning point for countries with good policies was lower than those with bad policies. Countries that applied western medical science in local developing contexts could expect to come off the rising portion of the tuberculosis Kuznets curve far earlier than those with poor policies.

In chapter 2 we did not make a distinction between our empirical expectations for differences in the shape of the tuberculosis Kuznets curve for morbidity and mortality. We did recognise that the developed world enjoyed the “luxury” of being able to treat cases of tuberculosis in specialised institutions and with the assistance of chemotherapeutic drugs. For the developing world we argued that the direct costs of these facilities and drugs were prohibitive for some countries. We also found the direct costs of cure were far higher than those of prevention. Econometric evidence here now reveals the importance of these observations. That the turning point for tuberculosis mortality is higher than for morbidity would suggest that societies encounter greater difficulty in dealing with the sick than with escaping the development of active symptoms.

3.5.2 Nutrition

An explanation for the development of active symptoms is under-nutrition. Comstock has argued that the underweight are more likely to develop tuberculosis disease, once infected, than those of a normal weight (Comstock, 1980, p. 449). Our findings offer some support to Comstock's conclusion. In particular, we found that the elasticity of the total number of calories consumed with respect to tuberculosis mortality is strong. But

the negative and insignificant elasticities in GMM (9) and also in the static models present a mixed picture concerning the direct effect of calories on the prevention of disease activation.

Some authors have argued that vitamin D is important in preventing the activation of tuberculosis. Vitamin D can be obtained both in the diet and through exposure to ultraviolet solar radiation. Dietary vitamin D is most abundant in meat, fish, milk and eggs yet no significant negative correlation is observed between consumption of vitamin D containing foods and the case rate. Furthermore, a positive and economically significant relationship exists between tuberculosis mortality and consumption of this food group. This has important implications for policy. Vitamin D may be shown to have beneficial effects in reducing morbidity through its function in supporting immune function and these results do not rule that out. Studies investigating the role of vitamin D at the molecular and cellular level suggest that vitamin D deficiency can impair adaptive immune function (Denis, 1991). Case-control studies have also reported that patients with active symptoms are vitamin D deficient[24]. These investigations do not however establish the direction of causality. That is, does tuberculosis illness result from vitamin D deficiency or does tuberculosis illness cause vitamin D deficiency? (Wilkinson R. J. et al 2000, Crowle A. J., et al. 1987) Regardless of the direction of causality, it not clear that dietary sources of vitamin D are important in explaining variation in morbidity. This is not to say, however, that vitamin D, either supplemental or synthesised following skin exposure to solar radiation, is not prophylactic.

It should be noted that the protein deficiency hypothesis is not undermined by our results even though countries with greater consumption of foods with high protein content do not benefit from lower morbidity rates. (Smallman-Raynor and Cliff, 2004, p. 647). Recent findings in nutritional science offer an explanation. The WHO suggests that dietary protein can comprise as little as 4.5 per cent of total calories in order to meet the requirements for the majority of individuals. Building in a safety margin, the WHO says that 10 per cent of calories derived from protein is sufficient for human functionality. Virtually every lentil, bean, nut, seed and grain and most fruit and vegetables provide more than 10 per cent of calories from protein. Thus, if total calories are sufficient, individuals will be ingesting sufficient quantities of protein regardless of

24 See Cegielski P and D. N. McMurray, (2004) for a summary of case-control studies.

their consumption of animal based protein. In fact, it is almost impossible to design a calorically adequate diet that does not include sufficient protein, including all of the essential amino acids: "It is difficult to obtain a mixed vegetable diet which will produce an appreciable loss of body protein without resorting to high levels of sugar, jams, jellies, and other essentially protein-free foods." (Hegsted, M., cited in Register, U.D., et al, 1973). Johnson's hypothesis that a lack of animal protein is associated with tuberculosis morbidity and mortality is not supported by our results. Countries that increasing consumption of calories from meat, fish, milk and eggs do not reduce morbidity burdens and may increase mortality.

Another hypothesis is that vitamin D is important for preventing tuberculosis morbidity (and by extension, mortality) and this hypothesis is explored extensively by Carson (2010). Here it is argued that people living in 19th century USA living in states that had greater exposure to sunlight benefited from increased vitamin D production. This, it is argued, improved immunity to the disease and led to a reduction in tuberculosis death rates during the summer months and among those with outdoor occupations (Carson, 2010, pp. 69 - 70). Although vitamin D might be important for increasing specific immune resistance to tuberculosis *in vitro*, it's beneficial *in vivo* effect on adaptive immune response to *mycobacterium tuberculosis* has never been demonstrated in healthy human subjects. Complexity arises because mycobacteria, both the virulent tuberculosis and the Calmette-Guerin (bovis) form are rendered inviable by ultraviolet light[25] both in gelatin suspension, (Collins, 1971) and suspended in air (Xu et al., 2003). Hence increased insolation could lead to reductions in tuberculosis morbidity, and by extension mortality, by increasing vitamin D synthesis in populations living in equatorial latitudes, but equally, mycobacteria could be rendered less virulent. Carson does not accommodate for the possibility of the latter. In Carson's empirical work it is not known whether insolation is a proxy for improved vitamin D-mediated immune response a or whether sunlight kills the mycobacterium leading to fewer deaths from tuberculosis. The static random effects models, RE (7) and RE (10) in table 3.3 finds that insolation is inversely related to tuberculosis morbidity but not significantly so. Coupled with the finding that dietary sources of vitamin D show no relationship to case rates and may increase death rates, the evidence presented here suggests that morbidity

[25] There is also a large literature concerning the use of ultraviolet germicidal irradiation lamps for the control of tuberculosis transmission in the healthcare setting.

(and mortality) is reduced because increased insolation kills *mycobacteria tuberculosis*. Carson's findings for the case of the United States in the 19th century corroborate our results.

Modern nutrition science argues the importance of having a diet which is highly nutritious. It has long been known that malnourished individuals are at higher risk for infectious disease due to an inadequate immune response. The branch of the immune system that produces antibodies is depressed in malnutrition, specifically with a decreased number of circulating B-cells and antibody responses. Other mechanisms that kill infectious organisms are also depressed in malnutrition. The functions of cytokines, are altered in malnourished individuals. Optimal immune function is dependent on adequate consumption of vitamin A, B1, B2, B6, B12, C and E, minerals including iron, zinc, magnesium and selenium and also folic acid concentrations (Muir Bowers, 2002). Fruits, vegetables, pulses and nuts are rich sources of these micro-nutrients.

Vitamin A deficiency can interfere with how epithelial cells function, which is vital in maintaining tissue structure. The ability of certain immune cells to kill infectious organisms and the production of B-cells and T-cells are also dependent on vitamin A status. The immune-related roles of vitamin C include collagen synthesis, phagocyte oxidative burst activity, and the ability of B-cells and T-cells to work properly. Vitamin B12 supplementation has been shown to improve both T-cell counts and natural killer cell activity in people with significant vitamin B12 deficiency. Vitamin B12 and folate are both involved in the production of genetic material. Vitamin B6 deficiency appears to decrease T-cell responsiveness and natural killer cells' ability to kill infectious organisms (Chandra, 1992). Even a mild zinc deficiency has been shown to reduce thymic hormone production and activity, decrease the numbers of CD4+ cells, harm the function of T-cells, natural killer cells and neutrophils, increase cell death, impair the ability of cells to kill infectious organisms and interfere with cytokine production. Selenium is a significant cellular antioxidant. Vitamin B6 has been demonstrated to be particularly effective in procuring the production of antibodies and T-cell function (Encyclopedia of Immunology, Second Edition). Animal and human studies suggest that vitamin B6 deficiency affects both humoral and cell-mediated immune responses. Lymphocyte differentiation and maturation are altered by deficiency, delayed-type hypersensitivity responses are reduced, and antibody production may be indirectly impaired. Deficiency of the vitamin has been associated with immunological changes

observed in the elderly, persons infected with human immunodeficiency virus (HIV), and those with uremia or rheumatoid arthritis (Rail and Meydani, 1993).

Given the general importance of micronutrients in supporting immune function it is not surprising that we find that increases in consumption of calories derived from fruits, vegetables, pulses and nuts is associated with reduced tuberculosis mortality. Therefore, a diet that is micronutrient dense and adequately calorific may offer the best protection again mortality.[26]

3.5.3 Medical provision

Reductions in tuberculosis mortality are hypothesised to be dependent on medical factors. In the European sample (Bowden et al. 2014) , it was discovered, using pooled panel data, that greater provision of beds for the tuberculous was associated with higher rates of tuberculosis mortality. Those countries with a greater provision of beds, however, were likely those that had the greater tuberculosis problem. By contrast, indicators of the quality of health systems, such as more physicians per capita and longer average length of stay in hospital, are both associated with fewer deaths from tuberculosis.[27] Yearly data for medical variables, appropriate for dynamic panel modeling, are not available for the global sample, with the exception of physicians per 100000. In the present study DOCTORS is negatively associated with tuberculosis mortality but not signicantly so. In places where effective treatment was offered, there was an incentive for patients with tuberculosis to actively seek treatment. Countries able to support physicians in greater abundance were those more likely to host health systems that were better able to identify the infected and accurately record the cause of death which may explain upward bias in the estimate.

In view of probable measurement error in the dependent variables in the global sample we appeal to related work in Bowden et al. 2014 which utilises data from the European sample which is probably more accurately measured. For these developed countries,

26 In a meta-analysis of studies that investigated the relationship between BMI and the relative risk of developing tuberculosis disease with respect to the reference category, Lönnroth et al (2008) aggregated the findings to argue that the relationship was log-linear. As BMI increases, the relative risk of developing tuberculosis disease falls, but at a decreasing rate, using findings from 8 studies selected on the basis of their methodological approach.

27 See Table 5 in Bowden, Jalles, Pereira and Sadler, 2014.

healthcare institutions were important in procuring decline in mortality rates in the post-war era where effective curative treatment was available. Firstly, this contrasts to the pre-war case for Britain. Evidence presented in Chapter 1 suggested that medical interventions were relatively unimportant in explaining fatality or mortality rates when compared to other socio-economic factors. Secondly, it was noted in chapter 2 that institutional factors that explain the steady decline in tuberculosis mortality and morbidity in developed countries were not suitable for deployment in developing countries. Developed countries largely eliminated tuberculosis as a public health concern in the mid to late twentieth century. They took advantage of the unique opportunity that the combination of established healthcare institutions, relatively high income and the means with which to administer effective treatment presented. Thirdly, we acknowledge that for the developing world, the root of the problem of poor institutions may lie in a 'reversal of fortunes'. Those societies that were more urbanised in the year 1500 (a proxy for wealth in the middle ages) had lower GDP per capita in 1995 (Acemoglu, 2002)[28]. As a result, the institutional constraints that emerged following the incentives for colonists to set up extractive institutions have limited the capacity of now 'developing' countries to deal effectively with the tuberculosis problem – whether through reduced income or through the qualitative limitations of the inherited institutional framework or both (Acemoglu, 2001).

3.5.4 Insolation

Countries which receive greater solar radiation or insolation were hypothesised to have lower case rates either because inhabitants had greater vitamin D production or because *mycobacterium tuberculosis* was more likely to be rendered inviable. The problem encountered, however, was that in order to estimate a model that was not misspecified, time invariant variables of interest, including insolation, were omitted by construction. The random effects model allowed for the inclusion of time invariant regressors, but it was dropped in favour of the fixed effects model because a Hausman test rejected the null that the GLS estimates were consistent. The fixed effects model had the benefit of eliminating the problem of omitted time invariant variable bias – a significant problem in a sample where countries are units - but at the cost of excluding insolation as a variable in the model.

28 See Figure I. Log GDP per capita (PPP) in 1995 against Urbanization rate in 1500

An alternative strategy is to estimate a model using Hausman-Taylor instrumental variables method for panel data. Two significant problems, however, would arise using this method. One issue is that dynamic effects cannot be included in the Hausman-Taylor model because the Hausman-Taylor instrumental variables approach still relies on the assumption that the expectation of the covariance of errors between periods is zero. Inclusion of a dynamic component violates this assumption by construction. The second issue is choosing suitable exogenous time variant and time invariant regressors to act as instruments for those variables that are endogenous. From the discussion thus far it is not clear that any of the variables, except insolation, can truly be considered to be uncorrelated with the disturbance. With too few variables suitable as instruments and the potential for misspecification, Hausman Taylor estimation is not an appropriate strategy. GMM methods therefore, avoid some of the issues regarding misspecification, but unfortunately do not provide any information on the importance of insolation. Though likely misspecified, regression specifications RE (7) and RE (10) found that when country specific effects were assumed to be random, insolation had no statistically significant effect on H_{it} but was inversely related to case rates. Given that insolation data are time invariant but are hypothesised to be important in explaining variations in tuberculosis morbidity this methodology is incomplete. Annualised observations, available from 1980, offer the possibility that studies investigating more recent data can incorporate insolation into panel regression analysis.

3.5.5 Urbanisation and industrialisation

Exhaustion affects the capacity of the human immune system to respond to the pathogenicity of *mycobacteria tuberculosis*, *bovis* and *africanum*. These organisms can remain quiescent in intracellular spaces in the pulmonary system known as tubercular lesions. Phagocytes released by the human immune system often die when they come into contact with the bacilli and enter a state of caseous necrosis. Granuloma follows in immunocompetent patients to contain the infection. The tuberculous bacilli are rarely eliminated by this process alone and can remain viable in the proteinaceous dead cell mass for several decades. A pathogenic steady state is established when the immune response is sufficient to contain, but not eliminate the infection. Continued strength of

the immune system is therefore required for the prevention of the development of active symptoms. Exhaustion associated with newly industrialising economies, as well as overcrowding and sustained exposure to the bacillus can compromise or weaken immune response. In the case where caseous necrosis expands into the bronchus, a lung cavity is formed. Coughing follows which allows the transmission of the disease. Importantly, transmission is not possible when active symptoms are not present.

Therefore, urbanisation and cramped working implied by industrialisation provided the means by which mycobacterium tuberculosis could spread. The relationship between industrialisation and H_{it}, using our proxy variable, MANU, was hypothesised to be linear. The independent effect of industrialisation was not found to be significant, but its inclusion in the regression significantly increased the model's explanatory power and it was positively related to case rates. By contrast, the relationship between urbanisation and H_{it} was hypothesised to be a non-linear inverted U-shaped relationship. In the long run higher levels of urbanisation imply greater economic development, more developed health systems and wealthier citizens. In the short-run, urbanisation implies greater stress on urban services, increased population density in the home and in the workplace and longer working hours. We observed an inverted U-shaped relationship between urbanisation and the burden of tuberculosis, controlling for other influential and related factors such as GDP per capita. This effect is expected in the early stages of development as health systems fail to keep pace with growing urban populations, especially in the NICs. We also experimented with the annual growth rate of the urban population as an independent variable to test the hypothesis that faster growth rates of the urban population create a situation in which tuberculosis abatement measures cannot keep pace. In unreported regressions we did not observe this effect.

Future research investigating the period after 1980 will be able to utilise hours worked in manufacturing as a proxy for exhaustion. Hours worked in manufacturing could be related to tuberculosis morbidity through a number of channels. The first is that work is exhausting, and that exhaustion reduces the capacity of the immune system to function. The second is that the tuberculosis bacillus is protected from ultraviolet radiation inside manufacturing units. The third is that the length of exposure of individuals to the bacillus is increased in cramped working and living environments. Although panel data covering our period and countries of interest do not exist, Bowden, Pereira, Jalles and Sadler (2014) discovered that for a cross-section of European countries, those that had

longer working hours also had higher case rates ($p<0.001$). It was also found that the greater the percentage of the workforce employed in manufacturing, the greater number of cases reported. Corroborative evidence presented here supports the hypothesis in chapter 2 that tuberculosis is a greater problem where populations are more urbanised and industrialised.[29] Further research is required to find more appropriate substitute measures of working conditions for global analysis in the period 1950 - 1980.

3.6 Conclusions

This chapter is based on the creation of a new global panel dataset. Using that dataset, we explored a number of economic mechanisms which procure activation of, and death from, tuberculosis for a cross-section of developed, newly industrialising and developing countries. We draw out a number of important subtleties in the nutrition literature and make precise the relative importance of the competing and complementary hypotheses.

Firstly we discovered the existence of both a tuberculosis morbidity and a tuberculosis mortality Kuznets curve. As income rises from an initially low level, so too does the burden of tuberculosis. The tuberculosis case rate and death rate reach a predicted maximum at $4673 and $7072 respectively and decline thereafter. We also observed that for countries with poor policies, both these turning points are much higher. Our interest was not only in the indirect observation of a Kuznet's type relation between income per capita and the burden of tuberculosis. We also wanted to test whether controlling for the socio-economic factors that the historical literature argues underlie cross country differences in tuberculosis rates caused the tuberculosis Kuznets relation to vanish. Our results indicate that this is not the case and we offered some reasons for this.

An increase in the number of calories consumed has a strong negative relationship with tuberculosis mortality. An increase in calories derived from fruits, vegetables, pulses and nuts also has a negative relationship with tuberculosis mortality, but the estimated effect is approximately a third of that of an increase in calorie consumption. Consumption of meat, fish, eggs and dairy is positively associated with tuberculosis death rates, a result which superficially may appear contrary to conventional wisdom.

29 See tables 5 and 6 (Bowden et al., 2014).

The protein deficiency hypothesis, however, is not undermined by these results even though foods with high protein content are not related to morbidity rates and are positively associated with death rates. As discussed, where total calories are sufficient, individuals ingest sufficient quantities of protein regardless of their consumption of animal protein.

This research helps to explain empirically observed patterns of tuberculosis morbidity and emphasises the importance of having a diet which contains is both adequate calories and comprises nutrient rich foods. Recent review papers have suggested that higher BMI is consistently related to lower relative risk of acquiring active tuberculosis disease. Whatever economic or environmental circumstances are present, it is clear that maintaining a good nutritional status and adequate micronutrient stores in the body are essential for an effective immune response to opportunistic infections, including tuberculosis. More investigation needs to be directed into the mechanisms by which resistance is conferred by a high calorie diet. Chapter 4 explores whether the existing scientific literature that has investigated the relationship between nutrition and tuberculosis morbidity, offers any insight into this mechanism.

Urbanisation was hypothesised to be both good and bad for the control, prevention and cure of tuberculosis. In the long run, higher levels of urbanisation imply greater economic development, more developed health systems and wealthier citizens. In the short run, faster urbanisation implies greater stress on urban services, increased population density in the home and in the workplace and longer working hours. We observed an inverted U-shaped relationship between the proportion of the population urbanised in a country and the burden of tuberculosis cases and deaths. Results from from the GMM regression on DEATHS suggest that after a society is approximately 77 per cent urban, death rates start to fall. We also investigated the effect of the annual growth rate in unreported regressions to test the idea that in rapidly urbanising countries urban services were overwhelmed, leading to environments in which mycobacteria could thrive. We were unable to reject the null hypothesis that there was no relationship.

The distinction was made between urbanisation and industrialisation. We used the proportion of the population employed in manufacturing as a proxy for industrialisation to test whether it had an independent effect separate from the closely related phenomenon of urbanisation. This independent effect might exist if factory conditions

facilitate the spread of the disease, through overcrowded working conditions, or activate the disease through workplace exhaustion. Although we did observe a positive coefficient, we could not reject the null hypothesis that the coefficient was zero.

Given the limitations of available medical data amenable to the present methodology, our findings are not complete. Similar investigations in Bowden et al. (2014) reveal, however, that in Europe, where diagnoses were more accurate and where the sick were more likely to come forward for treatment, that the quality of healthcare did determine mortality rates. In the present study, the finding that greater numbers of doctors are not negatively associated with death rates could be as a result of better reporting in countries with more developed healthcare systems. The reader is directed to Bowden et al. (2014) for a more thorough evaluation of the relative important of healthcare systems in Europe in the post-war era.

3.6 Appendix

Table 3.1 **Average calories consumed and annual rate of growth of urban population for selected countries, 1967.**

	Calories consumed	Rate of growth of urban population		Calories consumed	Rate of growth of urban population
Africa					
Angola	2036	5.73	Rwanda	2211	6.01
Botswana	2075	19	South Africa	2653	2.39
Cameroon	2077	6.21	Swaziland	2112	11.09
Chad	2230	8.92	Upper Volta	1834	3.68
Congo (Brazzaville)	2110	4.92	**Americas**		
Benin	1990	8.18	Bahamas	2497	5.23
Ghana	2264	4.19	Barbados	2805	0.47
Kenya	2224	7.05	Brazil	2380	4.77
Libya	2132	12.39	Canada	2895	2.56
Mail	2008	2.81	Colombia	1902	4.34
Mauritania	2043	10.77	Costa Rica	2231	4.58
Mauritius	2361	4.45	Cuba	2364	2.21
Niger	1956	8.45	Dominican Republic	1916	5.94
Nigeria	2092	4.74	El Salvador	1849	3.18

Table 3.1 continued …

Americas cont…	**Calories consumed**	**Rate of growth of urban population**		**Calories consumed**	**Rate of growth of urban population**
Guatemala	2044	3.55	Netherlands	3102	1.43
Haiti	1959	4.41	Norway	2990	3.45
Jamaica	2331	3.18	Poland	3379	1.56
Mexico	2581	4.64	Spain	2730	2.55
Panama	2252	4.35	Sweden	2807	1.66
Paraguay	2441	3.18	Switzerland	3285	2.4
Surinam	2182	2.3	**Asia**		
United States	2975	1.56	Iran	2092	4.93
Uruguay	2713	1.26	Iraq	2196	5.43
Europe			Israel	2956	5.12
Austria	3197	0.5	Japan	2649	3.29
Bulgaria	3480	3.35	Jordan	3318	6.93
Denmark	3083	1.54	Laos	2086	5.32
Finland	3066	3.34	Macau	2003	4.82
Hungary	3241	1.1	Philippines	1833	3.85
Iceland	2976	1.54	**Oceania**		
Ireland	3420	1.76	Australia	3159	1.69
Italy	3191	1.52	New Zealand	2982	2.33

Table 3.2 **GMM estimation of the tuberculosis Kuznets curve**

Dependent Variable		**lnCASES**			**lnDEATHS**	
	All countries	**Good policy**	**Bad policy**	**All countries**	**Good policy**	**Bad policy**
Model	GMM(1)	GMM (2)	GMM (3)	GMM (4)	GMM (5)	GMM (6)
lnGDPPC	10.50***	10.14*	8.31*	4.99*	4.23*	4.84
	[3.01]	[4.40]	[3.95]	[2.15]	[1.85]	[3.76]
$(\text{lnGDPPC})^2$	-0.62***	-0.61*	-0.47	-0.28*	-0.24*	-0.26
	[0.18]	[0.25]	[0.24]	[0.13]	[0.10]	[0.23]
lnPDENSITY	-1.54	-1.81	-1.71	0.63	-2.46***	-0.09
	[0.96]	[0.94]	[1.39]	[0.62]	[0.60]	[0.91]
lnCASES_{t-1}	0.13**	0.36***	0.08	-	-	-
	[0.05]	[0.07]	[0.05]	-	-	-
lnDEATHS_{t-1}	-	-	-	0.17*	0.45*	-0.02
	-	-	-	[0.08]	[0.06]	0.11
Test for 1st order (within group) autocorrelation	-3.03**	-1.41	-2.26*	-1.13	0.78	-0.77
Test for 2nd order (within group) autocorrelation	1.24	1.05	1.41	0.52	-0.85	-1.06
Turning point ($)	4673.94	4066.66	6868.96	7071.81	6811.94	10869.35
Wald test	67.69***	130.59***	22.98	431.15***	1138.63***	123.55***
Sargan test	220.80	108.22	174.21	142.08	165.4**	86.73
Number of instruments	211	178	185	202	137	151
n	612	261	351	369	163	206

Notes: ***, ** and * indicate significance at the 0.1 percent, 1 percent and 5 per cent level respectively. Standard errors in parentheses. Yearly time dummies not reported.
In GMM (1-6) the following instruments were used; any lags of the dependent variable higher than 2 with the contemporaneous error term, lags of lnGDPPC and $(\text{lnGDPPC})^2$ greater than 1 with the contemporaneous error term. The first difference of the remaining exogenous variables (including the time dummies) were used as standard instruments where available. Let $\boldsymbol{\varepsilon_i}$ be a vector of the first differenced residuals for country i and let the instrument set be a matrix, Z. In matrix form the available moments are given by

$$Z_i = \begin{pmatrix} H_{i63} & G_{i64} & 0 & 0 & 0 & \cdots & 0 & \vdots & \Delta x'_{i65} & 1965 \\ 0 & 0 & H_{i63-64} & G_{i64-65} & 0 & \cdots & 0 & \vdots & \Delta x'_{i66} & 1966 \\ \vdots & & & & \ddots & & & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & H_{i63-73} & G_{i64-74} & \vdots & \Delta x'_{i75} & 1975 \end{pmatrix}$$

where x_{it} is the vector of exogenous variables and G is the abbreviation of GDPPC. Then the moments

used in GMM (1) to GMM (6) for all countries are given by $E[\boldsymbol{Z}'\boldsymbol{\varepsilon}] = \mathbf{0}$. Here the empricial moment conditions are $plim\left(\frac{1}{n}\sum_{i=1}^{n}\boldsymbol{Z}'\hat{\boldsymbol{\varepsilon}}\right) = \mathbf{0}$. The notation H_{i63-73} indicates the range of years for which the variable can serve as an instrument, that is $[H_{i63}, H_{i64}, H_{i65}, H_{i66}H_{i67}H_{i68}H_{i69}H_{i70}H_{i71}H_{i72}H_{i73}]$ in 1975. For countries with fewer than 11 observations available some of the instruments are removed. If a country has instruments available continuously from 1963 to 1966 but not in 1967, then the row of Z corresponding to 1967 as well as subsequent rows are set to zero. Two observations per country are lost in taking first differences and constructing lags.

Critical value for test for 1st and 2nd order serial correlation distributed as N(0,1); Two tail critical value = ± 1.96 at 5 per cent significance level.

Critical value of Wald test for overall significance of the explanatory variables $\chi^2_{0.05,\,15} = 24.996$.

Critical values of the Sargan test of over-identifying restrictions $\chi^2_{0.05,\,195} = 228.58$ in GMM (1), $\chi^2_{0.05,\,162} = 192.70$ in GMM (2), $\chi^2_{0.05,\,169} = 200.33$ in GMM(3), $\chi^2_{0.05,\,186} = 218.82$ in GMM(4) $\chi^2_{0.05,\,121} = 147.67$ in GMM (5) and $\chi^2_{0.05,\,135} = 163.12$ in GMM(6).

Table 3.3 **Random effects, fixed effects and Arellano-Bond GMM estimation of the unrestricted model for morbidity and mortality**

Dependent Variable	**lnCASES**			**lnDEATHS**		
Model	RE(7)	FE (8)	GMM (9)	RE (10)	FE (11)	GMM (12)
lnGDPPC	6.16***	6.53***	9.22**	9.65***	10.00***	4.85*
	[1.40]	[1.76]	[3.01]	[1.56]	[1.93]	[1.99]
$(lnGDPPC)^2$	-0.39***	-0.39***	-0.58**	-0.57***	-0.58***	-0.28*
	[0.09]	[0.12]	[0.19]	[0.09]	[0.11]	[0.11]
lnPDENSITY	3.72×10^{-3}	0.25	-0.74	8.74×10^{-3}	0.13	-0.62
	[0.12]	[0.83]	[1.37]	[0.14]	[0.48]	[0.56]
lnCALORIES	-0.47	-0.42	-0.67	-1.04*	-1.19*	-1.22**
	[0.64]	[0.69]	[0.85]	[0.47]	[0.50]	[0.44]
MANU	2.87	4.14	2.64	-0.52	-1.14	-0.68
	[3.16]	[3.56]	[5.05]	[1.89]	[2.05]	[2.07]
URBAN	3.06×10^{-3}	-0.03	0.05	0.03*	0.04*	0.09***
	[0.02]	[0.04]	[0.06]	[0.02]	[0.02]	[0.03]
$URBAN^2$	-3.77×10^{-5}	4.50×10^{-4}	-5.61×10^{-5}	3.38×10^{-4}*	3.93×10^{-4}*	-5.82×10^{-4}*
	[2.14×10^{-4}]	[3.18×10^{-4}]	[5.6×10^{-4}]	[1.62×10^{-4}]	[1.85×10^{-4}]	[2.41×10^{-4}]
lnSOL	-0.09	-	-	-0.52	-	-
	[0.85]	-	-	[0.92]	-	-
DOCTORS	-	-	-	-1.80×10^{-3}	-1.96×10^{-3}	-1.88×10^{-3}
	-	-	-	[1.19×10^{-3}]	[1.27×10^{-3}]	[1.16×10^{-3}]
$lnCASES_{t-1}$	-	-	0.03	-	-	-
	-	-	[0.05]	-	-	-
$lnDEATHS_{t-1}$	-	-	-	-	-	0.17*
	-	-	-	-	-	[0.08]
Test for 1st order (within group) autocorrelation	-	-	-1.87	-	-	-2.73**
Test for 2nd order (within group) autocorrelation	-	-	1.16	-	-	0.46
Turning point ($)	2658.34	4208.95	2779.76	4543.87	5445.02	5108.65

Table 3.3 continued …

Dependent Variable	**lnCASES**			**lnDEATHS**		
Model	RE(7)	FE (8)	GMM (9)	RE (10)	FE (11)	GMM (12)
Wald test (RE/GMM) / F-Test (FE)	72.65***	3.79***	60.58***	524.40***	25.61***	828.63***
Sargan test	-	-	166.18	-	-	159.8
Hausman	6.27	-	-	4.21	-	-
Breush-Pagan LM test for RE	961.66***	-	-	652.92***	-	-
Number of instruments	-	-	187	-	-	178
n	423	423	301	341	341	214

Notes: : ***, ** and * indicate significance at the 0.1 percent, 1 percent and 5 per cent level respectively. Standard errors in parentheses. Yearly time dummies not reported. The available instrument set $\boldsymbol{Z_i}$ detailed in the note to table 3.2 is modified here so that $\Delta \boldsymbol{x_{it}}$ now contains lnCALORIES, MANU, URBAN and URBAN^2 in GMM(9). For GMM (12) the vector of differenced variables additionally contains DOCTORS.

The data are unbalanced, therefore the number of available instruments varies depending on which variables are included in the analysis. The number of complete observations on country i in period t are fewer because data on manufacturing are only available in a subset of countries in the sample.

Critical value for test for 1st and 2nd order serial correlation distributed as N(0,1); Two tail critical value = ± 1.96 at 5 per cent significance level.

Critical value of Wald test for overall significance of the explanatory variables and Hausman test RE(7): $\chi^2_{0.05,\,20} = 31.41$, GMM(9) and RE(10): $\chi^2_{0.05,\,19} = 30.14$ and GMM (12) $\chi^2_{0.05,\,19} = 28.87$.

Critical value of Sargan test of over-identifying restrictions in GMM (9): ,GMM(12): $\chi^2_{0.05,\,158} = 188.33$.

Critical value of the Breush-Pagan LM test $\chi^2_{0.05,\,1} = 3.84$.

Critical value of the F-test FE(8) $\text{F-stat}_{0.05,\,19,\,363} = 1.62$,FE(11) $\text{F-stat}_{0.05,\,20,\,280} = 1.61$.

Table 3.4 **Arellano-Bond GMM estimation of the effect of individual food groups on tuberculosis morbidity and mortality**

Dependent Variable	lnCASES				lnDEATHS			
Model	GMM (13)	GMM (14)	GMM (15)	GMM (16)	GMM (17)	GMM (18)	GMM (19)	GMM (20)
FRUITS	-0.15	-	-	-	-0.36***	-	-	-
	[0.19]	-	-	-	[0.06]	-	-	-
CEREALS	-	0.36	-	-	-	0.10	-	-
	-	[0.25]	-	-	-	[0.08]	-	-
MEAT	-	-	-0.13	-	-	-	0.28***	-
	-	-	[0.15]	-	-	-	[0.05]	-
OILS	-	-	-	-0.08	-	-	-	0.01
	-	-	-	[0.17]	-	-	-	[0.06]
Wald test	50.43***	52.49***	50.86***	49.76***	1063.78***	908.18***	1046.06***	899.11***
Sargan test	144.61	145.56	146.57	145.57	142.55	144.74	150.2	140.77
Number of instruments	175	175	175	175	164	164	163	163
n	242	242	242	242	185	185	185	185

Notes: GMM (13) – (16) estimated using the benchmark model $lnH_{it} = \beta_1 + \beta_2 ln(GDPPC)_{it} + \beta 3(ln(GDPPC))^2_{it} + \boldsymbol{x}_{it}'\beta + \gamma H_{it-1} + \alpha_i + \varphi_t + \varepsilon_{it}$. where H_{it} is the log of the case rate (lnCASES) and $\boldsymbol{x}_{it}$ is a vector of explanatory variables containing lnPDENSITY, MANU, URBAN and URBAN^2. In GMM (17) – (20) $\boldsymbol{x}_{it}$ contains DOCTORS in addition.
Critican value Sargan test of over-identifying restrictions in GMM (13) – (16): $\chi^2_{0.05,\,154} = 183.96$, GMM (17) and (18) $\chi^2_{0.05,\,142} = 170.81$, GMM (19) and (20) $\chi^2_{0.05,\,141} = 169.71$.

Table 3.5 ILO data availability

Country	(BA) (Employees) (ISIC – Rev. 2)	(BA) (All persons engaged) (ISIC- Rev. 2)	(CA) (Employees) (ISIC – Rev. 2)	(CA) (All persons engaged) (ISIC- Rev. 2)	(DA) (Employees) (NSIC)	(DA) (Employees) (ISIC- Rev. 2)	(DA) (All persons engaged (NSIC)	(DA) (All persons engaged) (ISIC – Rev. 2)	(E) (Employees) (ISIC- Rev. 2)	(E) (All persons engaged) (ISIC – Rev. 2)	(FA) (Employees) (NSIC)	(FA) (Employees) (ISIC- Rev.2)	(FA) (Insured persons) (ISIC 1)	(FA) (Insured persons) (ISIC Rev. 2)	(FA) (All persons engaged) (ISIC – Rev.2)	(FD) (Employees) (NSIC)
Angola									1983							
Australia						1969										
Austria												1969 *	1972			
Bahamas														1976		
Barbados			1977													
Belgium									1970			1970				
Bermuda				1978												
Botswana						1972										
Brazil	1981 *							1970								
British Honduras											1973	1977				
Brunei						1972										
Bulgaria			1969													
Cameroon						1969			1973 *							
Canada	1969					1969										
Chad																
Colombia						1975 *										
Congo (Brazza)																
Costa Rica	1981 *															
Cuba																1971
Cyprus								1969								
Czechoslovakia			1976	1969												
Dahomey										1977 **						
Denmark						1970										
Dominica																

Table 3.5 continued …

Country	(BA) (Employees) (ISIC – Rev. 2)	(BA) (All persons engaged) (ISIC- Rev. 2)	(CA) (Employees) (ISIC – Rev. 2)	(CA) (All persons engaged) (ISIC- Rev. 2)	(DA) (Employees) (NSIC)	(DA) (Employees) (ISIC- Rev. 2)	(DA) (All persons engaged (NSIC)	(DA) (All persons engaged) (ISIC – Rev. 2)	(E) (Employees) (ISIC- Rev. 2)	(E) (All persons engaged) (ISIC – Rev. 2)	(FA) (Employees) (NSIC)	(FA) (Employees) (ISIC- Rev.2)	(FA) (Insured persons) (ISIC 1)	(FA) (Insured persons) (ISIC Rev. 2)	(FA) (All persons engaged) (ISIC – Rev.2)	(FD) (Employees) (NSIC)
Dominican Rep.					1970											
El Salvador						1977										
Fed. Rep. Germany	1976		1969						1981							
Finland	1969		1970													
France									1971							
Gambia			1973													
Ghana			1969													
Gibraltar			1972													
Greece	1981															
Guatemala						1974						1974				
Guyana																
Haiti																
Honduras																
Hong Kong						1969										
Hungary			1970													
Iceland														1969		
Iraq						1982		1974								
Iran																
Israel												1972				
Ireland								1973								
Italy	1977	1969 *														
Jamaica																
Japan	1969					1978										

Table 3.5 continued …

Country	(BA) (Employees) (ISIC – Rev. 2)	(BA) (All persons engaged) (ISIC- Rev. 2)	(CA) (Employees) (ISIC – Rev. 2)	(CA) (All persons engaged) (ISIC- Rev. 2)	(DA) (Employees) (NSIC)	(DA) (Employees) (ISIC- Rev. 2)	(DA) (All persons engaged (NSIC)	(DA) (All persons engaged) (ISIC – Rev. 2)	(E) (Employees) (ISIC- Rev. 2)	(E) (All persons engaged) (ISIC – Rev. 2)	(FA) (Employees) (NSIC)	(FA) (Employees) (ISIC- Rev.2)	(FA) (Insured persons) (ISIC 1)	(FA) (Insured persons) (ISIC Rev. 2)	(FA) (All persons engaged) (ISIC – Rev.2)	(FD) (Employees) (NSIC)
Jordan								1972								
Kenya			1972													
Kuwait						1985										
Laos																
Lebanon																
Libya																
Luxembourg												1978 *				
Macau						1984										
Madagascar																
Mali																
Malta																
Mauritania																
Mauritius			1970													
Mexico												1976				
Netherlands									1975							
New Zealand						1969				1971						
Nicaragua												1970				
Niger												1974				
Nigeria					1969											
Norway	1972															
Panama	1969															
Paraguay	1983															
Peru						1974										

Table 3.5 continued …

Country	(BA) (Employees) (ISIC – Rev. 2)	(BA) (All persons engaged) (ISIC- Rev. 2)	(CA) (Employees) (ISIC – Rev. 2)	(CA) (All persons engaged) (ISIC- Rev. 2)	(DA) (Employees) (NSIC)	(DA) (Employees) (ISIC- Rev. 2)	(DA) (All persons engaged (NSIC)	(DA) (All persons engaged) (ISIC – Rev. 2)	(E) (Employees) (ISIC- Rev. 2)	(E) (All persons engaged) (ISIC – Rev. 2)	(FA) (Employees) (NSIC)	(FA) (Employees) (ISIC- Rev.2)	(FA) (Insured persons) (ISIC 1)	(FA) (Insured persons) (ISIC Rev. 2)	(FA) (All persons engaged) (ISIC – Rev.2)	(FD) (Employees) (NSIC)
Philippines	1977					1971										
Poland						1969										
Portugal	1974				1971	1971 *										
Puerto Rico						1969										
Qatar						1983										
Romania						1980										
Rwanda																
Senegal						1971 *		1971								
South Africa						1970										
Southern Rhodesia						1971										
Spain	1969															
Surinam									1973							
Swaziland			1970													
Sweden					1969		1970									
Switzerland																
Syria																
Turkey						1972						1969				
United Kingdom									1975							
United States						1969										
Upper Volta																
Uruguay	1974					1982										
Venezuela						1972										
Yugoslavia							1969									

Table 3.5 continued …

Notes: *Data not disaggregated by sector. ** Manufacturing not included in returns.
Key: (BA) – Labour force survey; (CA) – Labour – related establishment census; (DA) Labour – related establishment survey; (E) – Official Estimates; (FA) – Insurance records; (FD) – Administrative reports. The number in each box is the year in which data on employment by sector becomes available.

Table 3.6 Good policies and bad policies by country

	Policies		
Country	*Proportion vaccinated ≥ 0.9 ?*	*Long course treatment available?*	*≥ 5 technicians per 100000 population?*
Angola	Y	N/A	N
Australia	Y	Y	Y
Austria	Y	Y	Y
Bahamas	N	N	Y
Barbados	N	N	Y
Belgium	N	Y	Y
Bermuda	N	N	N/A
Botswana	N	N	N/A
Brazil	N/A	N	Y
British Honduras	N	N	Y
Brunei	N	N	Y
Bulgaria	Y	N	Y
Cameroon	N/A	N	N
Canada	N	Y	Y
Chad	N	N	N
Colombia	Y	Y	N
Congo (Brazzaville)	N	N	N
Costa Rica	N	Y	Y
Cuba	Y	Y	Y
Cyprus	N	Y	N/A
Czechoslovakia	Y	Y	Y
Dahomey	N	Y	N
Denmark	Y	Y	Y
Dominica	N	N	Y
Dominican Republic	N	N	N
El Salvador	N	N	N
Federal Republic of Germany	Y	Y	Y
Finland	Y	Y	Y
France	Y	Y	Y
Gambia	N	N	N
Ghana	N	N	N
Gibraltar	N	N	Y
Greece	N	Y	N/A
Guatemala	N	N	N
Guyana	N	N	Y
Haiti	N	N	N
Honduras	N	Y	Y
Hong Kong	Y	N	N

Table 3.6 continued…

	Policies		
Country	*Proportion vaccinated ≥ 0.9 ?*	*Long course treatment available?*	*≥ 5 technicians per 100000 population?*
Hungary	Y	Y	Y
Iceland	Y	Y	Y
Iran	N	N	N
Iraq	N	N	Y
Ireland	Y	Y	Y
Israel	Y	Y	Y
Italy	N	Y	Y
Jamaica	N	N	Y
Japan	Y	Y	Y
Jordan	N	N	N
Kenya	Y	N	N
Kuwait	N	N	Y
Laos	N	N	N
Lebanon	N	N	Y
Libya	N	N	Y
Luxembourg	Y	Y	Y
Macau	N	N/A	N
Madagascar	N/A	Y	N
Mali	N	N/A	N
Malta	N	N/A	Y
Mauritania	N	N/A	N
Mauritius	N	Y	Y
Mexico	N	N	N
Netherlands	N	Y	Y
New Zealand	Y	Y	Y
Nicaragua	N	N	N
Niger	N	N	N
Nigeria	N	N/A	N
Norway	Y	Y	Y
Panama	Y	Y	Y
Paraguay	N	N/A	Y
Peru	N	Y	Y
Philippines	Y	N/A	N
Poland	Y	Y	Y
Portugal	N	N/A	Y
Puerto Rico	N	Y	Y
Qatar	Y	Y	Y
Romania	Y	Y	Y

Table 3.6 continued…

	Policies		
Country	*Proportion vaccinated ≥ 0.9 ?*	*Long course treatment available?*	*≥ 5 technicians per 100000 population?*
Rwanda	N	N	N
Senegal	N	N/A	N
South Africa	Y	Y	Y
Southern Rhodesia	N	N	N
Spain	N	N/A	Y
Surinam	N/A	N	N
Swaziland	N	N/A	N
Sweden	Y	Y	Y
Switzerland	Y	Y	Y
Syria	N	Y	N
Turkey	Y	N	N
United Kingdom	N	N	N
United States	Y	Y	Y
Upper Volta	N	N/A	N
Uruguay	Y	N/A	N
Venezuela	N	N/A	Y
Yugoslavia	Y	Y	Y

Notes: Brazil, Madagascar and Surinam do not report the number of recorded vaccinations of children under 1 year old for any period (1963 - 1975). Cameroon reports under 1 year vaccinations, but not under 1 year population.

Figure 3.1 **Respiratory tuberculosis case rates in selected Developing Countries**

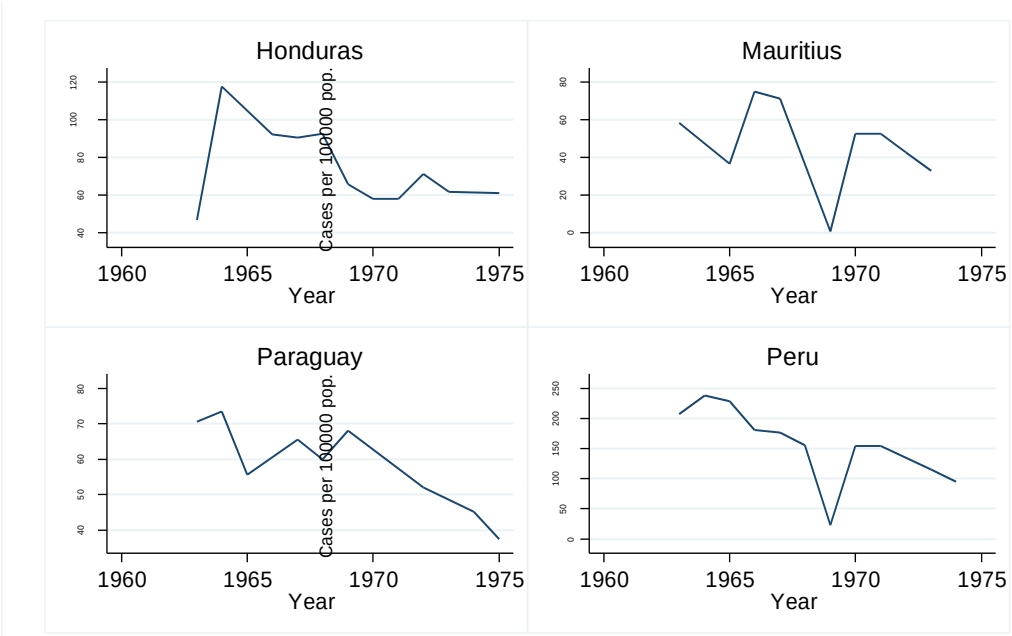


Figure 3.2 **Respiratory tuberculosis case rates in selected Newly Industrialised Countries**

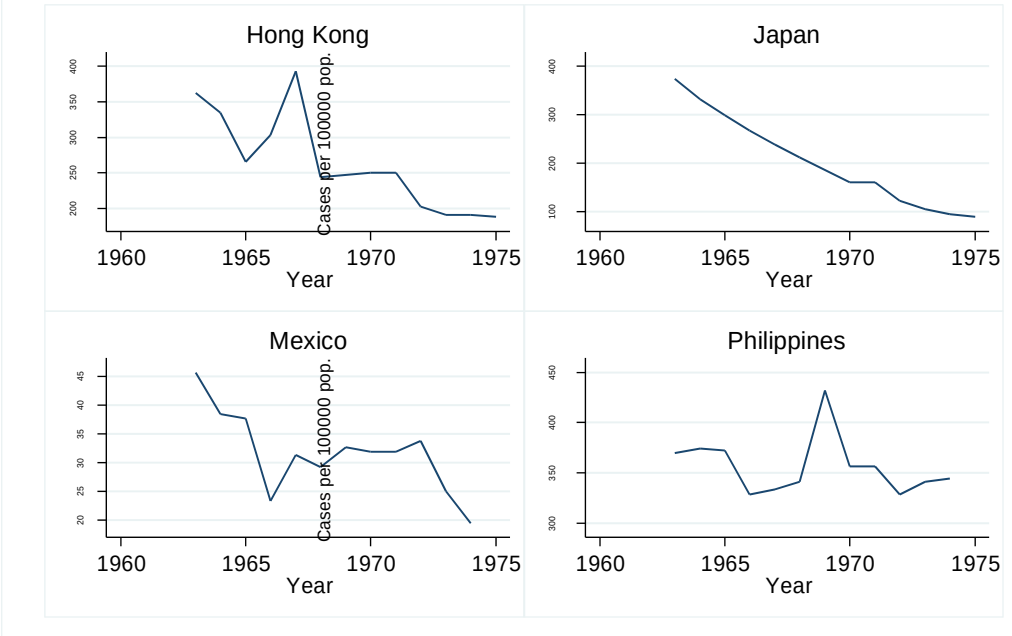


Figure 3.3 **Respiratory tuberculosis case rates in selected Developed Countries**

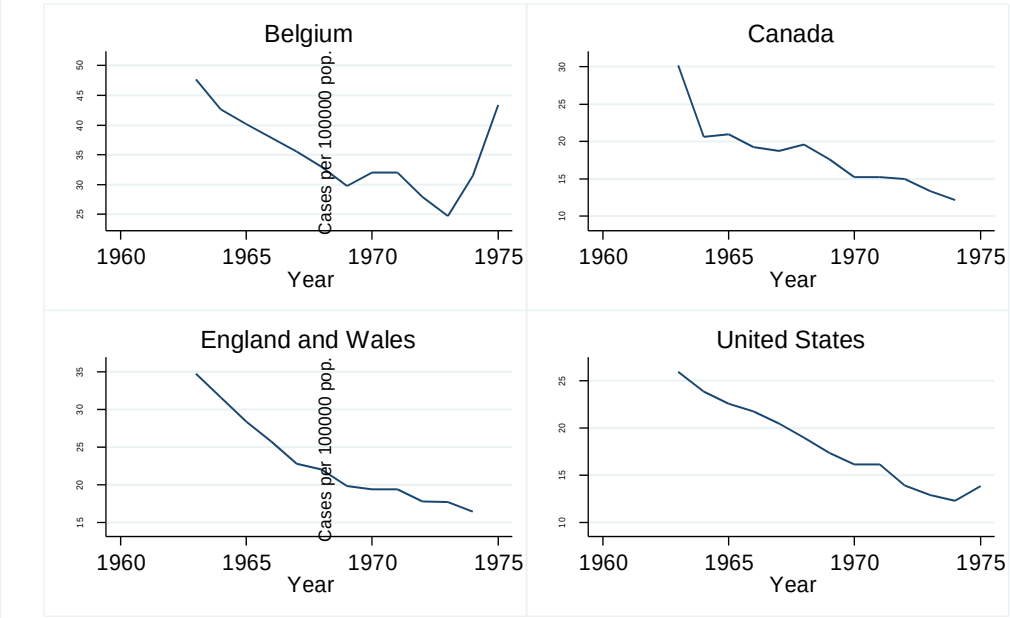


Figure 3.4 **Respiratory tuberculosis death rates in seleced Developing Countries**

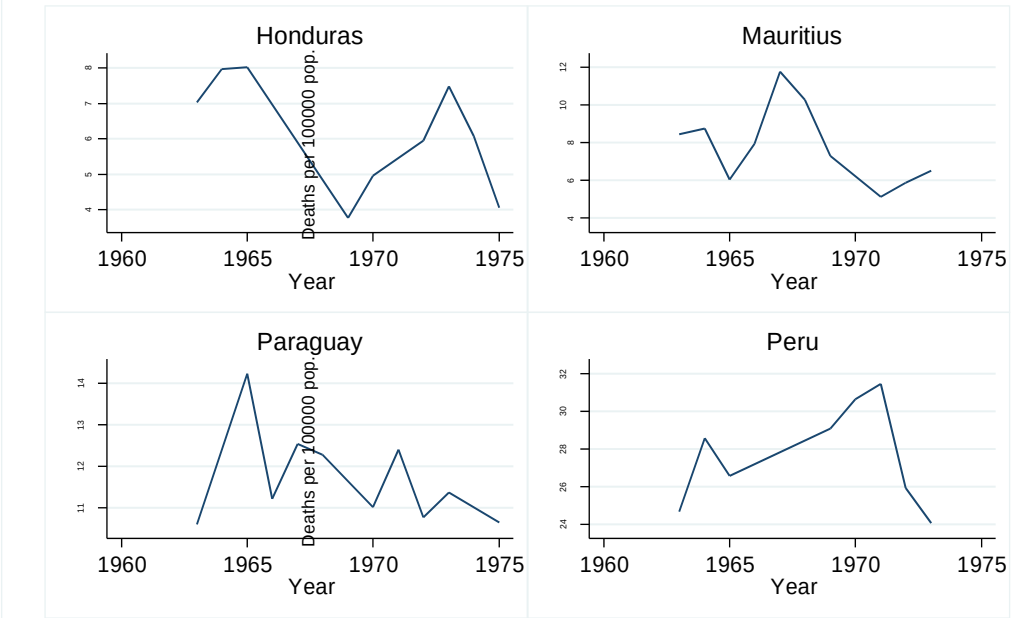


Figure 3.5 **Respiratory tuberculosis death rates in selected Newly Industrialising Countries**

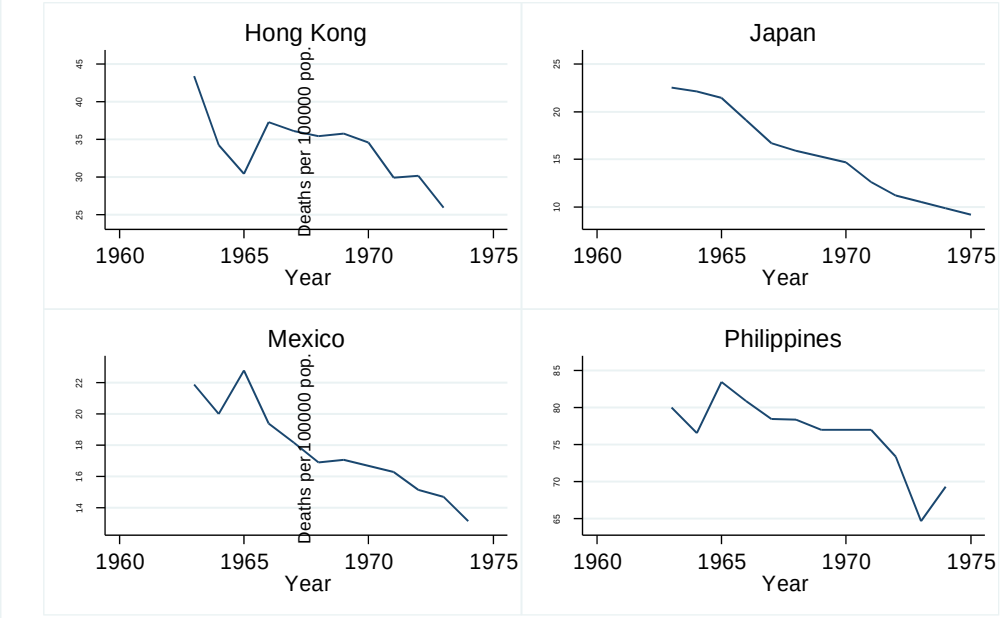


Figure 3.6 **Respiratory tuberculosis death rates in selected Developed Countries**

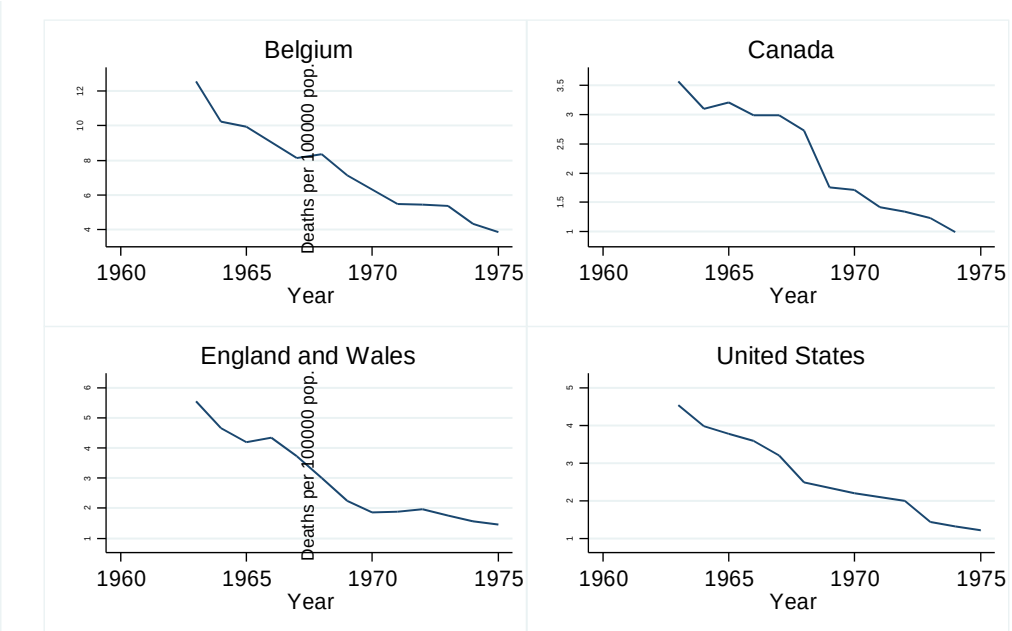


Figure 3.7 Tuberculosis Morbidity Kuznets curve

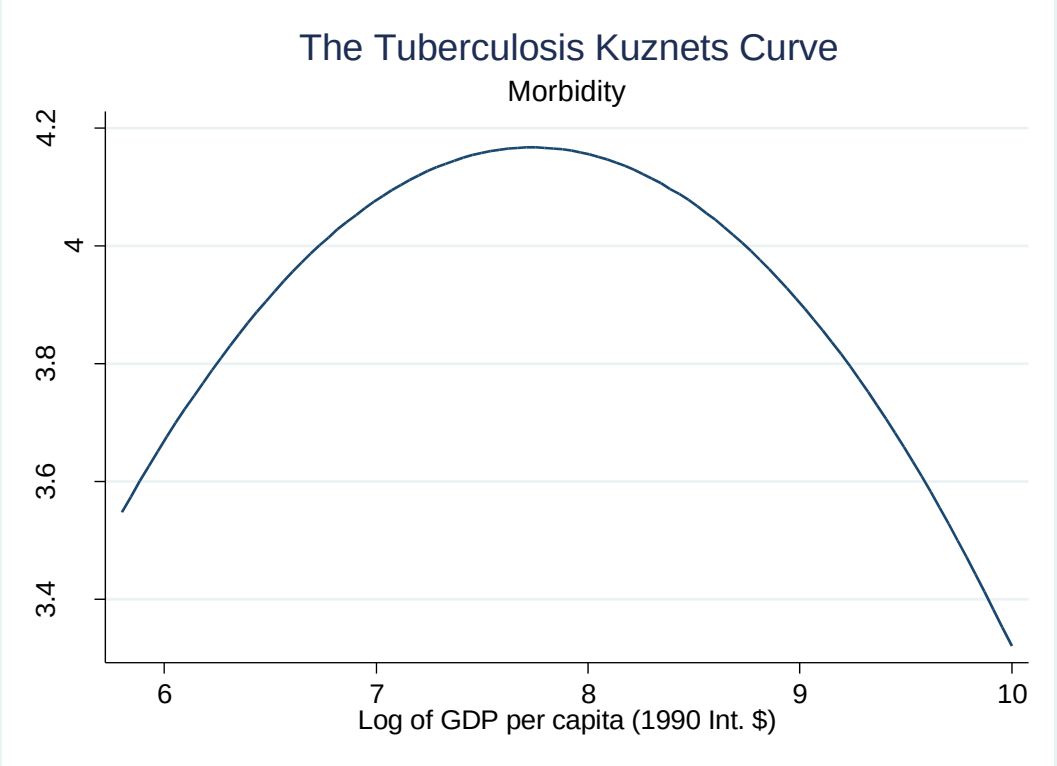
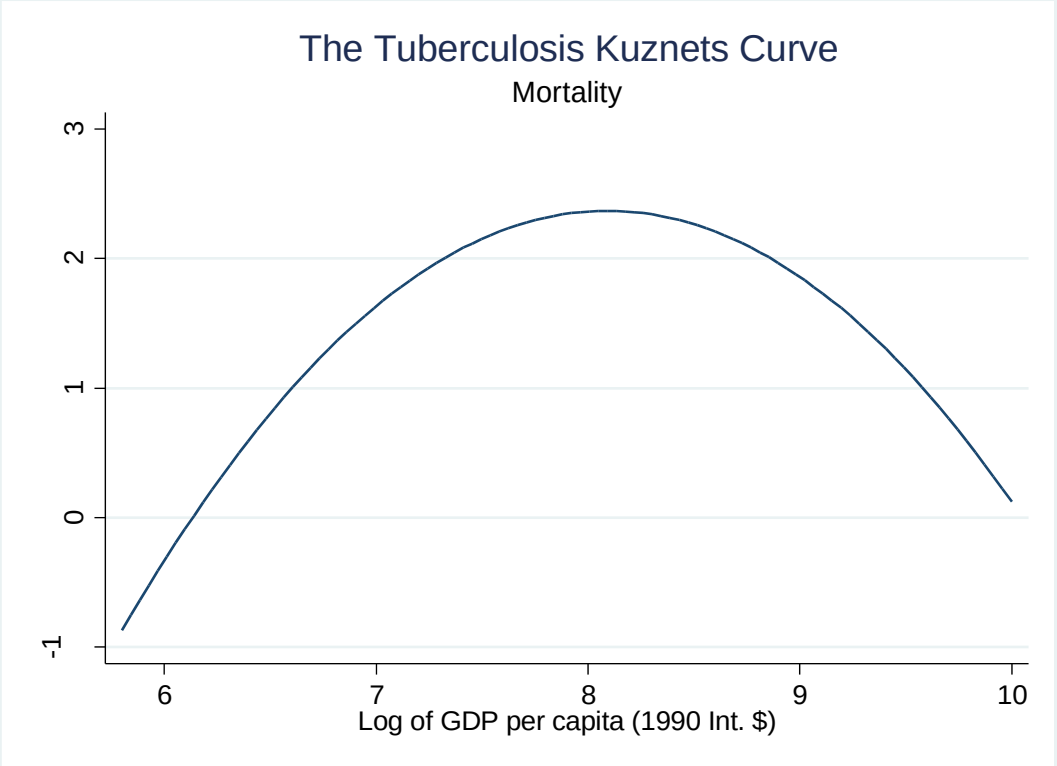


Figure 3.8 Tuberculosis Mortality Kuznets curve



4. Nutrition, standards of living and the future of tuberculosis prevention

Alex Sadler

4.1 Introduction

This thesis has investigated two distinct periods relating to the medical history of tuberculosis (TB). The first is one in which TB disease could not be cured using antimicrobial drugs and control of the disease relied on prevention and methods to reduce transmission. The second period is one in which antimicrobial drugs became available and where the possibility that TB could be cured and eliminated existed. This was a distinct, but short, era. The inexorable decline of both TB morbidity and mortality in developed Western Countries led to a loss of public health interest, but in developing and emerging contexts the problem remained. Complacency toward TB control and elimination during the period after 1950 meant that there was a missed opportunity to eliminate the disease before *mycobacteria tuberculosis* adapted to antimicrobial drugs and HIV-TB interacted in patients in the 1980s and 1990s.

Resistance of micro-organisms to antimicrobial medicine is now an issue of mainstream scientific research and increasingly one of global health significance. The World Health Organisation (WHO) has identified some worrisome consequences, both present and future, from increasing antimicrobial resistance (AMR) (WHO, 2001, pp. 11-13). The possibility exists, to the extent that new antimicrobial therapies are delayed, that there will be periods of time in the future in which no effective antibiotic interventions are available for some patients. A return to the pre-antibiotic era is not certain but increasing AMR to first and second line therapies is observed across many pathogens including staphylococcus aureus, streptococcus, pseudomonas aeruginosa, clostridium difficile, salmonella, E. coli, acinetobacter baumannii and for the purposes of this chapter, *mycobacterium tuberculosis*. In addition, new antibacterial agents approved by the Food and Drug administration declined between 1983 and 2003, from 16 (1983-87) to 7 (1998-2002) (Alanis, 2005, p. 702, Fig. 1).

The classification of TB as multidrug-resistant (MDR) and extensively-drug-resistant (XDR) is a reflection of new problems emerging in destroying TB pathogens in active cases. Not only is prevalence of MDR-TB rising, but treating MDR-TB is generating AMR to second line drugs, a selection pressure which favours increased prevalence of XDR-TB. Countries which have been using second line drugs for ten years or less including Thailand (7 years), Philippines (9 years) and Peru (10 years) had the lowest rates of resistance to second line drugs, whereas those with the longest histories of use, including Russia and South Korea (over 20 years) had the highest rates (Dalton, 2012, pp. 9- 11). Such a trend correlates well with AMR in other species.

Chapters 1 and 3 investigated the importance and effects of nutrition in cases of TB, both in terms of the approach of local councils in the pre-antibiotic era and in terms of the relationship of specific food groups to TB morbidity in a cross-section of developing, emerging and developed countries. This chapter seeks to utilise advances in the field of TB and nutrition to identify whether, in this prospective world of widespread MDR and XDR-TB, nutrition can mitigate the impact of the disease if antimicrobial medicines cannot. Section 4.2 investigates the current state of AMR in relation to TB to ask whether the threat of a post-antibiotic era is real. Section 4.3 reviews existing non-medical socio-economic strategies to control TB and asks if these are effective. Section 4.4 reviews the scientific literature which relates TB to nutrition. The purpose of this is to discover whether nutritional interventions are viable public health options. Section 4.5 concludes.

4.2 MDR-TB and XDR-TB

Drug resistant strains of TB pose an increasing threat to conventional medical methods to treat TB disease. Soon after the discovery of streptomycin in 1944 (Espinal, 2003, p.44) and its first use in clinical treatment in 1948, (Johnson, 2007, p.1) drug resistant TB strains emerged. In the decades that followed increasing prevalence of MDR-TB and XDR-TB was observed in both new and existing cases of the disease. First line anti-tuberculosis drugs included Isoniazid and Rifampicin. These drugs were chosen on the basis of their low cost, efficacy, ease of use in Direct Observed Therapy- Short Course (DOTS), and few side effects. Strains of *mycobacterium tuberculosis* resistant to these first line drugs are now termed MDR-TB. This section details the emergence and

consolidation of drug resistant TB strains through time. The purpose of this section is to argue that drug resistant TB is a growing problem which shows little sign of abating and that the possibility exists that antimicrobial drugs will fail to treat increasing numbers of TB patients.

Studies in 1960s attempted to measure the prevalence of drug resistant strains resistant to at least one drug. Miller, investigated sputum specimens from 1292 newly diagnosed patients from 125 randomly selected chest clinics in the Second National Survey, 1963, in Great Britain. Of the 894 cultures that were *mycobacterium tuberculosis* positive and 2 that were *mycobacterium bovis* positive, 3.0% of cultures were resistant to streptomycin and 1.7% to isoniazid. Resistance to PAS was lower at 0.8% (Miller, 1966). In addition strains resistant to both streptomycin and isoniazid had already emerged in Great Britain by 1963. Other developed world studies were conducted in Canada, Australia, France and United States. In Canada drug resistance was highly regional. The study carried out by the Department of the Laboratories of the Human Health Association collected 2064 samples appropriate for study. Drug resistance to either streptomycin, PAS or isoniazid was observed to be between 3.1% and 5.1% for all Canadian provinces, except Quebec. Here resistance was found in 16.4% of cultures, similar to the highest contemporary rates in Eastern Europe (Armstrong, 1966, p. 421, Table III). Across Canada, resistance to two drugs was discovered in 0.9% of cultures and resistance to three drugs in 0.3% (Armstrong, 1966, p. 422, Table VI). Similar rates were found in the incidence of resistance to at least one drug in Australia (3.8%), France (9.8%) and United States in previously untreated patients (Howells, 1968; Canetti, 1972). It is not surprising that early evidence exists for AMR in *mycobacteria tuberculosis* for a small proportion of patients since some in the population will have genetic mutations that confer resistance. The worry is that strains infecting these patients will be selected for as non-AMR strains are eliminated by antimicrobial drugs.

More recently, this has become a problem in the context of dismantled or poorly organised health systems. In developed economies declining rates of TB led to a reduction in the level of health service provision to tackle the disease. For example, TB case rates rebounded in the United States between 1983 and 1992 in the context of a trend of overall decline thus leading to the suggestion that ‘control services must be maintained even in the face of decreasing case rates’ (Institute of Medicine, 2000, p. 29; Figures 2-4). The WHO and the International Union against Tuberculosis and Lung

Disease (IUATLD) launched the Global Project on Anti-Tuberculosis Drug Resistance Surveillance in 1994 with the purpose of standardising global reporting methods and estimating prevalence of MDR-TB. An effort was made to distinguish between those patients with acquired and primary drug resistance. Acquired drug resistance was defined in those patients who had had at least one month on either isoniazid or rifampicin at some point in the past. Primary drug resistance was defined in those patients for which no evidence could be found of prior anti-tuberculosis treatment (Pablos-Méndez, 1998, p. 1642). Areas in Africa with health systems that did not effectively administer DOTS during the 1990s, including countries such as Benin, Kenya, Lesotho and Zimbabwe, had comparatively few MDR-TB positive cultures owing, perhaps, to the late introduction of first line drugs. Areas in the former Soviet Union with a longer history of anti-tuberculosis drug use but disorganised, non-standardised health systems, following the collapse of the USSR, had higher prevalence rates. General TB notification rates in Russia, Latvia, Lithuania, Ukraine and Estonia roughly doubled between 1990 and 2000 (Raviglione, 2003, p.9, Figure 4). In a sample of 248 patients in Ivanovo Oblast, Russia, 28% of samples were primary-resistant to at least one drug. In Latvia, of 347 patients, drug susceptibility was observed in only 66% of samples (Pablos-Méndez, 1998, p. 1645, Table 3). As with primary-, acquired-resistance rates were correspondingly high in the former Soviet Union (Pablos-Méndez, 1998, p. 1645, Table 4). Hence, MDR-TB strains have become more prevalent in certain parts of the world as a result of the failure of health services in some countries.

XDR-TB, following the conference of the WHO XDR-TB Task Force in Geneva (October 2006), sought a definition of XDR-TB that would be more amenable to testing. Pre-existing definitions, which required scrutiny of the efficacy of many different lines of drugs, were difficult to perform in developing economy contexts. Thus XDR-TB is defined as any strain that has the resistance of MDR-TB, but in addition is resistant to any fluoroquinolone and at least one of the three injectable drugs; capreomycin, kanamycin and amikacin (WHO, 2006). Because XDR-TB has only been defined recently, few comprehensive prevalence reports exist. As of 2004, 22 countries had confirmed cases of XDR-TB (Migliori, 2007a, p. 424). By 2013, a WHO update confirmed that XDR-TB has been detected in 84 countries worldwide (WHO, 2013). Therefore, the evidence suggests that XDR-TB is a growing problem.

Gandhi (2006) assessed the prevalence and consequences of MDR- and XDR-TB in the

rural area of KwaZulu Natal (KZN), South Africa. The primary outcome of interest were the number of cases and the prevalence of MDR and XDR-TB. Three different groups were tested for various sputum culture resistance. The largest group (group 3) comprised 1428 tested individuals in the district hospital between June 2005 and March 2006. Within this group 13% had MDR-TB and 2% had XDR-TB (Gandhi, 2006, p.3). The secondary outcomes for patients with XDR-TB were mortality, proportion with previous hospital admission, HIV-coinfection and genotype of isolates. Of the 53 patients with XDR-TB, all that were tested were co-infected with HIV and all had died within 210 days following the collection of sputum. Despite evidence that XDR-TB emerged in hospital settings, the study found that 55% of the XDR-TB patients whose hospital medical records were reviewed, (n=47), did not contain evidence of previous treatment and 67% of records, (n=42), indicated no admission to hospital for any cause. The evidence suggests, therefore, that KZN strain XDR-TB can be acquired in the community (Gandhi, 2006, p. 3, Table 2). Although not all XDR-TB patients are co-infected with HIV, the study found that of those whose records revealed HIV status (n=44), 100% of XDR-TB patients were HIV-coinfected.

Some reviewers have argued that effective controls measures are available to prevent the spread of new drug resistant strains of TB in hospital settings. Measures, such as long course use of second line drugs are sufficient, if implemented effectively, will contain the threat posed by AMR strains in developing Asian, Eastern European and African countries (Raviglione, 2003, p.9). New evidence from Italy casts doubt on this congenial perspective. Strains of "XXDR" TB were identified in two female patients under the age of 50 that were resistant to all known drugs *in vivo*, and culture resistant to all drugs except dapsone, clarithromycin and thiacetazone for which laboratory testing was unavailable (Migliori, 2007b). Additionally, doubt is again cast by the recent finding that AMR *mycobacterium tuberculosis* complex strains may not necessarily retain their lower bacterial fitness compared to non-AMR strains. A subset of the T85 (Beijing) strain had mutations in their rpoB gene which conferred resistance to rifampicin, with no *in vitro* fitness cost (Gagneux, 2006, p. 1944).

A global pillar of the current anti-TB public health effort is the Direct Observed Therapy, Short Course (DOTS) strategy implemented by the WHO when TB was declared a global health emergency in 1993. The five components of the strategy included political commitment to support TB treatment, the detection of active TB via

sputum microscopy, direct observation of short course therapy using first line drugs, guaranteeing a supply of medicine to the sick and gathering information relating to national tuberculosis programme (NTP) efficacy. The belief, upon implementation of this strategy, was that the failure to reduce the number of global TB cases during the 1990s lay not in the limitations of science but in the political will and resources of national governments (Brewer, 2004, p. 822).

Despite initial support, the efficacy of DOTS has been placed into doubt. Studies have concluded that TB case rates may be expected to continue to rise in the context of HIV and TB co-epidemics in Botswana, despite implementation of DOTS strategy (Kenyon, 1999). Vietnam is the only country in the group of 22 high burden TB countries which has successfully met the detection and cure rates targets set by the DOTS strategy. Yet, in the 15-24 years prime income producing age group, case notification rates rose in four of six provinces and declined less than average in two between 1996 and 2003 (Huong, 2007, p. 96). Finally, the case of Russia provides a compelling example of how socio-economic factors can disrupt DOTS strategy and reverse progress towards elimination. Although Russia has not adopted DOTS, its own strategy is very similar. Patients are observed during treatment, a regular supply of drugs is available and effort is made to isolate infected individuals. Despite weaknesses in the Russian system during the Soviet era, economic issues resulted in increasing rates of TB mortality across Russia and in specific areas, such as the Kemerovo Region (Siberia). As a result of the failure of the Russian centralised economy and a reliance on barter, an inability to pay health worker salaries and rapid decline in drug production, distribution and sale, followed. In this context the mortality rate in Western Siberia increased from approximately 10 per 100000 to 26 per 100000 between 1990 and 1997 (TO, F. S. C., 2000, p. S161, Fig. 2).

These findings, coupled with the finding that Beijing strain *mycobacterium tuberculosis* has spread out of Eastern Europe and into Georgia (Niemann, 2010), and the KZN strain can now be acquired in the community, offers compelling evidence that conventional medicine will be unable to cure ever larger numbers of TB patients into the future. At present the problem is geographically localised, but underdeveloped health care systems in high burden countries are an opportunity for MDR- and XDR-TB to gain traction. This section has argued that a worrisome trend has emerged wherein drug resistant strains are now more geographically dispersed and more prevalent in sub-populations.

Mycobacteria, in addition, are acquiring resistance to increasing numbers of antimicrobial drugs, a trend which does not show signs of stopping.

4.3 Socio-economic strategies to control TB

These trends in medical control and TB epidemiology have motivated the search for, and implementation of, new strategies that recognise the socio-economic-epidemiological interaction in TB control. New alternative methods to control TB do not proceed along medical lines, but instead focus on the ways in which socio-economic risk factors for TB disease can be managed and reduced. Current ideas on how to mitigate TB risk by using socio-economic methods, excluding nutritional methods, are critiqued in this section. This section then motivates the use of nutrition as the primary option for a socio-economic intervention. In the next section, the scientific basis for considering nutritional intervention is evaluated.

Chapter 2 considered the developing setting. Here it was argued that solutions applied in the 'developed world' were not easily transplanted into a developing country setting. Thus, we highlighted that in these countries key cost and logistical considerations apply which do not in developed settings. These considerations are relevant when considering socio-economic policy in present day high burden countries. Historically, socio-economic interventions were made in a developed setting. In chapter 1, using evidence from Department of Health audits, we found that local governments were willing to offer nutritional assistance in some areas in the interwar period. We also found that in many ways local governments pre-empted many of the recently proposed socio-economic strategies to control TB such as better housing, sanitation and examination of contacts.

Others have discovered the importance of socio-economic risk factors in a similar context. In an examination of the children of parents who had TB at the Papworth Institution in Britain (1918 - 1943), Bhargava (2012) compared case rates of those children who were 'village-born' with those that were born outside of the village setting. Only 1 child of 24 that went on to eventually develop TB was village born. Those who lived in the village benefited from spacious dwellings, open air and good nutrition (by contemporary standards). The findings support the hypothesis that creating a social

environment, favourable to resisting TB activation, reduced the incidence of TB disease in children who had always lived in a favourable environment (Bhargava 2012). This corroborates evidence presented in chapter 1 which found that counties and towns in interwar Britain with better living conditions often had fewer cases and fewer TB deaths.

Given that social determinants have been identified, and medical interventions may be insufficient, the drive has been to turn evidence into action by proposing practical methods to improve food security, housing and environmental conditions and financial, cultural and geographic barriers to health care access (Hargreaves, 2011, p.3). As discussed in chapter 2, TB not only has consequences of immediate poverty, but can also perpetuate cycles of poverty over the long term. One suggestion has been to make conditional transfer payments to poor families. Examples already exist in Latin America and Asia where such interventions have been made in situations where a family member is afflicted by AIDS. Assisting vulnerable families can help prevent a loss of subsistence, a loss of enrolment of children in schools and the sale of livestock for consumption, whilst also enabling micro investments and improving women's outcomes (Adato, 2009, p.2). Since these problems arise in TB cases in the developing world and in the combination of AIDS and TB infection, similar approaches are warranted to protect livelihoods. Hargreaves et al (2011) suggest training to support human capital development, microfinance initiatives to complement social protection from the non-governmental sector in addition to conditional transfer payments. Such activities could, it is argued, lead to mitigation of material constraints of the tuberculous, reduce barriers to diagnosis and treatment, offer an educational opportunity to patients and their communities and be conditional on adherence to treatment regimens to improve compliance (Hargreaves, 2011, pp. 5-6).

As discussed in chapters 1, 2 and 3, the transmission of TB is facilitated more readily in urban areas and in the context of societies that are rapidly urbanising. Poor ventilation and overcrowding in the home and the workplace, as well as in public and hospital settings facilitate the spread of disease via sustained exposure. Solutions offered are generally interventionist on the grounds that, given the severity of the TB problem, patients in developing countries cannot wait for economic growth and development to produce urban environments which are less conducive to TB transmission. General solutions such as upgrading urban slums and improving housing quality are proposed

which appear to have attractive knock-on effects and interactions. Not only would urban regeneration directly address the physical transmission problem but also be an opportunity to create health seeking behaviours in individuals (Hargreaves, 2011, p. 7). Management of the urban environment to control TB has been a consideration of policy makers since at least the 1929 audit of health services in interwar Britain discussed in chapter 1. However, implementing urban regeneration to the extent that a significant proportion of the population benefits often takes as long as waiting for economic growth to solve the problem of poor urban environments. Projects such as electrification, sanitation, irrigation and bridge construction, taken together are not separate from, but constitute, economic development regardless of their ancillary effects on TB control.

A promising new approach looks at ways in which TB specific interventions can be used to procure better living and working environments. Barriers to seeking TB care include stigma, isolation and depression. A lack of TB care results in poverty, sickness and often death. In a study which investigated TB afflicted households in Lima, it was found that those with TB were more likely to be depressed ($P<0.0001$) and isolated ($P<0.0001$) (Rocha, 2011, Table 1). The Innovative Socio-Economic Interventions Against TB (ISIAT) project suggests that simple psychosocial interventions, coupled with micro-enterprise and credit, vocational training and food and cash transfers can result in poverty mitigation and income generation. Poverty reduction increases the resources available to then improve environmental conditions and reduce TB susceptibility (Rocha, 2011, Fig. 1).

There is a long history of association between the onset of TB symptoms and malnutrition. Other risk factors have been associated with the onset of active TB including immunosuppression (HIV) and diabetes (Cegielski, 2004; Jeon 2008). In patients without these comorbidities, anti-TB nutrition is an exciting prophylactic measure. Large scale interventions described above are politically and economically difficult to coordinate. Structural interventions designed to provide improved living and working conditions are long-term strategies. The prospect of providing prophylactic nutrition is tantalising as a socio-economic intervention. It has the potential to be cost effective and to produce immediate results. The next section explores whether the science supports the notion that specific nutrients or general nutrition can enhance resistance to the onset of TB disease. The scientific literature has explored the relationship between specific nutrients and immunology, as well as nutritional status and

onset of TB disease. Here, the purported biological mechanisms through which nutrition can improve cell mediated immunity are evaluated using evidence from human and animal studies.

4.4 Nutritional approaches to prevent latent TB from becoming active

4.4.1 TB pathogenesis and nutrition

Mycobacteria have developed mechanisms through which they are able to resist destruction by the host immune system. In human respiratory infection, mycobacteria typically inhabit an area of the lung called the Gron focus. In response the host will release cytokines, such as tumour necrosis factor (TNF-α) and interferon gamma (IFN-γ) to attract immune cells to the site of infection. Macrophages engulf (phagocytosis) mycobacterium and attempt to deliver them to lysosomes for destruction. However, some mycobacteria are able to resist delivery and surviving bacteria can establish colonies within the phagosome of the macrophage (Houben, 2006, p.1). In most cases when pathogens undergo phagocytosis, the lysosomes of the macrophage fuse with the phagosome and nitrous acid is released which digests the foreign body. Once a colony is established, mycobacteria multiply and the alveolar macrophage can lose its cellular integrity and disintegrate (lysis) (Clark-Curtiss, 2003, p. 519). Mycobacteria are then released into the alveolar space and again provoke a non-specific immune response. Specific immune cells respond to antigen presentation on the surfaces of lysed macrophages and assist in the formation of granulomas within the lung tissue. When the host specific immune system functions correctly lymphocytes help form granulomas to contain mycobacteria. Functional macrophages and lymphocytes enter the periphery of the granulomas to replace non-functional macrophages. However, necrosed non-functional macrophages remain within the granulomas and mycobacteria are able to remain dormant in this environment, either within the phagosome or in the extracellular caseous necrosis. Recently it has been suggested that mycobacteria induce macrophages to increase in lipid content, entering a ‘foamy’ state. This provides nutrients for the mycobacteria to persist (Peyron, 2008).

Some hosts however are unable to control infection because too few specific immune cells or functional macrophages can be recruited. The result is the destruction of lung cells in the surrounding tissues and the growth of tubercular lesions in the lung, followed by the onset of symptoms. Nutritional status may explain why some people can resist TB disease, and some cannot. Many studies implicate the role of specific nutrients in facilitating immune function in humans (Chandra, 1992; Ibs, 2003; etc.). Diets low in protein, calories, vitamin A, pyridoxine, biotin and zinc can result in thymic atrophy (McMurray, 1984) .T-cell maturation, necessary for effective granuloma formation, could therefore be impaired. T-cells also require the release of cytokines including interleukin-2 (IL-2) for growth, proliferation and differentiation into effector T-cells. Other cytokines, such as IFN-γ can also be impaired in nutritionally deprived hosts (McMurray, 1990). T-cells use IFN-γ to upregulate macrophages so that mycobacteria are unable to prevent phagolysosome maturation. In non-specific immunity, malnutrition, particularly protein-calorie malnutrition, has been implicated in reduced mobilisation of immune cells into tissue lesions, impaired phagocytosis and reduced production of macrophage cytokines, IL-1 and tumor TNF-α (Dai, 1998, p.110). Studies have sought to emulate some of the *in vitro* observations *in vivo*. However, the investigation is fraught with methodological difficulties.

4.4.2 In vivo studies

In case-control studies untreated, but active, TB patients are compared to a control group, which is usually matched for a number of sample characteristics such as age, gender, HIV status, weight and so on. The purpose of these studies is to reveal differences in nutritional status between TB active patients and the control group. An early study investigated markers of malnutrition among 30 TB patients with TB aged 19-61, who were admitted to Northwick Park Hospital, Harrow. The nutritional measurements of the tuberculous were compared to a matched control group. The investigator found that when compared to the control group, the TB group had reduced body mass index (BMI) ($P=0.001$), tricep skinfold thickness ($P=0.002$), serum albumin ($P<0.0001$), iron ($P<0.0001$), arm muscle circumference ($P=0.002$) and iron binding capacity ($P<0.001$) (Onwubalili, 1988). Karyadi et al also used a case-control study design to investigate the nutritional status of patients with active TB and compared them to healthy controls in Jakarta, Indonesia. The study compared the BMI, skinfold thickness of the triceps, biceps, subscapular and suprailiac, mid-upper arm

circumference, proportion of fat, serum albumin, blood haemoglobin, plasma retinol, plasma zinc and plasma zinc protoporphyrin of 41 outpatients aged 15-55 with untreated pulmonary TB to 41 healthy matched controls. It was found that when compared to healthy controls, all the anthropometric markers and micro-nutrient status indicators were lower in the TB group, with the exception of plasma zinc protoporphyrin. The addition of serum zinc and vitamin A (retinol) levels as markers of TB infection indicate that active TB is associated with lower micro-nutrient status (Karyadi, 2000). Other studies have corroborated these findings when comparing patients with lepromatous leprosy co-infected with pulmonary tuberculosis (Saha, 1989) and whilst observing the levels of IL-1 (interleukin-1) and TNF (tumour necrosis factor) in moderately and severely malnourished TB patients (Tsukaguchi et al., 1989). The innovation in Tsukaguchi et al. is the attempt to correlate IL-1 and TNF with differing levels of nutritional depletion. They find that when nutritional depletion is mild to moderate, IL-1 and TNF levels are higher than in controls, but when depletion is severe they are lower. This provides evidence for a possible mechanism linking nutrition with activation of pulmonary TB.

These case-control studies suffer from a design problem in the present context if the intent of the investigation is to establish that nutritional depletion increases the risk of TB activation. These studies must necessarily make the conclusion that nutritional and anthropometric status of TB patients is poor when compared with healthy controls. No statement of the direction of causality can be made. Although nutritional depletion can compromise cell mediated immunity, active TB can also lead to weight loss and a change in nutritional status once in the disease state (Cegielski, 2004; Lönnroth, 2010). Thus case-control studies which compare untreated TB patients to healthy controls cannot determine whether active TB causes or is caused by nutritional depletion, or both.

As a result, the study of the chronology of nutritional status and TB activation presents a methodological challenge. In a randomised controlled trial investigators could potentially infect a group of volunteers with TB, randomise them into a placebo-control group and a treatment group, and then test the efficacy with which different dietary treatments affected TB outcomes in a factorial or parallel-group design. Nutritional and anthropometric status could be observed at baseline and then repeated measurements taken over time, before and after any onset of disease. Obviously this is impractical for

reasons of ethics and self-preservation. A solution is available, but at a cost. A cohort study could follow a sample of individuals over time and record their dietary habits and other TB related risk factors. Comparison of the subgroup of the cohort that goes on to develop active disease with those that do not might reveal differences in dietary habits that are correlated with disease. Typical TB prevalence rates in a randomly selected population are likely low. This means that any investigative study must necessarily follow a large number of subjects so that results are obtained with sufficient statistical power. Some sub-populations have higher prevalence, including migrant Gujarati Indians and Muslims living in London (Wilkinson, 2000, pp 618-619). Potentially, these sub-populations could be followed, but genetic predispositions impair generalisations of findings to the wider population.

Cohort studies that investigate the relationship between nutritional status and TB disease exist but they are few. Hemilia et al. investigated whether dietary intake of vitamin C affected the incidence of tuberculosis in a cohort of 26975 Finnish male smokers between the ages of 50 and 69 years. A follow-up was conducted which identified 167 tuberculosis cases from the hospital discharge register. Data for the study were collated from the Alpha-Tocopherol, Beta-Carotene Cancer Prevention (ATBC) study which was conducted in order to examine the effects of vitamins A and E on the incidence of cancer. (Hemila, 1999, pp.632-633) In order to use these data for the purposes of establishing a relationship between TB disease and dietary intake of vitamin C, the investigators took advantage of baseline measurements made available by the ATBC study. Daily dietary intake of vitamin C was measured using the National Public Health Institute and corrected for vitamin C losses made during preparation. The study also took advantage of data available at baseline for potential confounders. Controls for age, BMI, marital status, education, residential neighbourhood and daily cigarette and alcohol use were included. The authors used a proportional hazard regression model in which they tried to discover which variables increased relative risk of TB disease over time. After controlling for confounders vitamin C intake was not significantly related to TB incidence. However, consumption of fruits, vegetables and berries, which were poor sources of vitamin C, were significantly correlated with lower TB incidence in those whose diets were rich in vitamin C. The authors speculate that this effect is observed because of other beneficial compounds in these foods (Hemila, 1999, pp.635-636) and make the surprising finding that vitamin C poor fruits and vegetables have the greatest

effect in reducing incidence. The investigators also found that a higher BMI at baseline was protective against TB disease. Those males with a BMI of >27 (kg/m^2) had a relative risk of 0.29 compared with those whose BMI was <23. This result complements the consistent finding of case-control studies that lower BMI is correlated with cases of TB.

It should be noted that these are particularly generalised results that do not indicate that there is any specific immune mechanism that reduces tuberculosis risk. The study does suggest that vitamin C, despite its purported benefits in bolstering immune response to other pathogens (Hemila 1996; Hemila 1997a; Hemila 1997b), is not effective in reducing the risk of TB disease. Fruit, vegetable and berry intake are associated with lower risk but the specific mechanism is unknown. Many recent studies in the nutrition literature have emphasised that the synergy of various nutrients is important, but often investigators prefer to isolate specific nutrients to discover associated effects (Messina, 2001; Jacobs, 2003: Jacobs, 2007). Because the body is a complex system, its many dynamic interactions with food are difficult to model. Isolating vitamin C intake may be important for purposes of managing this complexity, but if vitamin C is only effective in synergy with other nutrients, then this study provides strong supporting evidence. In a major review of the relationship between nutrition and tuberculosis, Cegielski (2004) criticise Hemila (1999) for using the discharge registry to detect cases of tuberculosis. This, it is argued, selects for patients that were sick enough with TB to seek hospital admission (Cegielski, 2004, p.290). Under the assumption that more advanced TB is related to hospital admission, (which may not always be the case), these results offer at the very least the conclusion that higher intake of fruits, vegetables and berries, in the context of a diet rich in vitamin C, are protective against being hospitalized with TB.

Some studies have avoided the problems associated with tracking constituent food intake in cohorts over time and instead focused on physiological markers of nutrition status. Leung et al identify BMI, haemoglobin A_{1c} and cholesterol level as potential risk factors for the development of active cases of TB among a cohort of 42116 elderly patients in Hong Kong, China (in addition to other socio-medical controls). The cohort was measured at baseline between Jan 1 2000 and 31 Dec 2000 by the Elderly Health Service. Individuals were identified using their identity cards and then matched to either the date of tuberculosis notification, date of death, or Dec 31 2005 during the follow-up,

whichever came first.[30] Unfortunately the relative risks of different haemoglobin A_{1c} and cholesterol level quantiles are not reported. After controlling for age and gender, it was found that those that did go on to develop active TB had lower BMI (P<0.001) than those that did not. This result is consistent with Hemila (1999). The investigators, however, disaggregated cases of pulmonary and extrapulmonary TB and calculated the corresponding hazard ratios for different BMI ranges. A BMI ≥30 in the cohort, before exclusion of individuals with comorbidities, had an associated hazard ratio of 0.33 (0.17-0.65, P<0.001) compared to the reference group with BMI 18.5 to <23 (Leung, 2007, p. 1301, Table 4). Those with extrapulmonary TB, however, saw no statistically significant differences across the BMI range (P=0.92). Once all confounders were controlled a BMI ≥30 had a hazard ratio of 0.35 (0.18-0.69, P<0.001), whereas a BMI <18.5 had a hazard ratio of 2.19 (1.64-2.92) compared to the reference category. Again the risk of contracting extrapulmonary TB bore no statistically significant relationship to BMI. The interesting result that low BMI is a risk factor for pulmonary but not extrapulmonary TB disease suggests that the mechanism by which BMI confers protection is specific to pulmonary immune function.

Unfortunately, no other cohort studies investigate the relationship between specific nutrients and the risk of developing active TB. Animal studies, however, can provide *in vivo* evidence of nutritional treatment in preventing the onset of symptoms, in a randomised, controlled trial. The obvious weakness of this approach is the degree to which results can be generalised to humans. Animal models involving guinea pigs have been explored in a small literature. McMurray et al have investigated the role that protein deficiency plays in increasing their susceptibility to TB disease in a factorial randomised and controlled trial. The animals were divided into four groups. Two control groups, one in which guinea pigs were vaccinated and one in which they were not, received a feed mixture of cornstarch and ovalbumin with the recommended proportion of protein. Two treatment groups, one in which guinea pigs were vaccinated and one in which they were not, received a low protein diet, but with the same number of calories (McMurray, 1985, pp. 555-556). Vaccinations were given to half of the guinea pigs in

[30] Confounding factors controlled for include age, gender, smoking, alcohol use, marital status, education level, housing, work-status, means-test public assistance, diabetes mellitus, cholesterol level, cardiovascular disease, hypertension, COPD and/or asthma, malignancy, recent weight loss of 5% or more within 6 months, hospital admission within 12 months, and activities of daily living scores (Leung, 2007, p. 1301, Table 5).

the treatment and control groups at the commencement of the dietary regimen. After six weeks on the control and treatment diets, guinea pigs were exposed to virulent *mycobacterium tuberculosis* in an exposure chamber. One, two and three weeks after exposure, guinea pigs were killed. Lung, spleen, bronchotracheal lymph nodes and serum albumin were harvested. These tissues were then tested for concentrations of viable *mycobacterium tuberculosis* per gram of wet tissue and compared between groups at the given time interval on a $\log_{10}$ scale.[31] The results indicated that the protein deficient animals had greater numbers of viable *mycobacterium tuberculosis* living in their lung tissue than those on a normal diet, when autopsied either one, two or three weeks after infection. In addition, unvaccinated animals on the low protein diet had greater numbers of viable bacilli in lung tissue than vaccinated, although the difference small. Vaccinated guinea pigs on normal diets fared much better than vaccinated guinea pigs on a protein deficient diet (McMurray, 1985, p.557, Fig. 2). Thus, the authors argue that the failure of the BCG vaccine in human trials might be related to the lack of protein consumption in developing world populations (McMurray, 1985, p.558).

The study contained three notable weaknesses. Firstly, very few guinea pigs were killed in each time interval (between 4 and 6) leading to the strong possibility of type I error. Secondly, unlike humans, guinea pigs have innate susceptibility to tuberculosis (Gupta, 2005, p. 278). Hence, pathogenesis and the extent of clinical manifestations is divergent between the two species. Thirdly, the method of necropsy is a basic monitoring technique little changed from the time of Koch (Young, 2009, p. 2013) and more advanced methods have become available that will allow for direct monitoring of disease morphology and pathogenesis.

Other animal models have investigated the role of zinc and vitamin D deficiency. Zinc deficiency has been implicated in reduced thymic function *in vitro*. Hence, circulating T lymphocytes, required to form granulomas in response to mycobacterial infection, are reduced (Dai, 1998, p. 114). McMurray et al investigated the effects of dietary deficiencies of zinc and protein alone, and the combination of both compared to fully fed animals on the number of viable *mycobacterium bovis* at the site of BCG vaccination. They discovered that when protein was high, restricting zinc had no effect on mean viable *mycobacterium bovis* compared to unrestricted. The same result was

[31] The purpose of the $\log_{10}$ scale was to account for differences in organ size and sampling procedures (McMurray, 1985, p.556).

obtained when protein was low. However, when inguinal (groin) lymph nodes were examined, a marked difference between low and high zinc guinea pigs was observed. Those on zinc deficient diets had significantly greater viable BCG than those that were not ($P<0.05$) (McMurray, 1983, p.759, Fig. 4). In addition, lymphocyte blastogenesis was severely impaired in animals on low zinc, low protein diets (McMurray, 1983, p.760, Fig. 5), impairing adaptive immune response. This animal model study suggests that zinc and protein are both important in explaining variation in immune response of animals subcutaneously injected with non-virulent BCG vaccine. However, the study cannot be used to suggest that zinc supplementation reduces the risk of virulent TB activation in guinea pigs exposed to bacilli via the natural bronchial route.

Thus in an augmented study of similar design, investigators asked whether guinea pigs fed an isocaloric diet, identical in every nutrient except protein, and subjected to aerosol infection five weeks after vaccination, varied in tuberculin sensitivity. Half of the animals on the low protein diet were switched to normal protein levels following infection. The results indicated that protein deficient guinea pigs had low sensitivity to tuberculin, a loss of BCG protection against virulent TB and increased numbers of viable mycobacteria in the lungs and spleen. Animals re-fed after infection had similar outcomes to vaccinated controls (McMurray, 1986). Again, however, animals were not observed during pathogenesis and their clinical symptoms were not tracked through time. Guinea pigs were euthanised within four weeks in all three studies discussed and hence chronic onset of symptoms was not observed. Thus animal models provide a methodological advantage not available in human studies, but as yet, methods have not been employed to extend animal models to mimic human pathogenesis.

The role of vitamin D in cell-mediated immunity has also been investigated using guinea pigs. Vitamin D deficiency can impair T-cell function and lack of metabolically active vitamin D ($1,25(OH)_2D_3$) can impair containment of intracellular replication of mycobacteria in human macrophages (Denis, 1991). Guinea pigs were fed five experimental diets which were identical, except in their vitamin D concentration. Vitamin D concentration was 0%, 25%, 50%, 75%, 100% and 200% of the recommended daily intake for guinea pigs across the six diets. The aim was to test both the effect of deficiency and supplementation. Guinea pigs were tuberculin tested one day before euthanasia. Blood samples were taken from the heart and the spleens and lungs removed from animals upon death. In terms of tuberculin sensitivity, those

animals both on the 200% diet and those on <100% exhibited diminished response on both 5 and 100 tuberculin unit doses. Those on the 100% diet had the greatest response, as measured by the diameter of hardened tissue at the site of the tuberculin test. However, when the numbers of viable mycobacteria were measured from samples of the spleen and lung, no significant differences were found between the dietary groups. Differences were observed between those animals which had been vaccinated and those which had not 3 and 6 weeks after infection via aerosol. Again the efficacy of BCG in retarding the proliferation of mycobacteria in lung and spleen tissue, when the dose is small, was demonstrated (Hernandez-Frontera, 1993). The authors assumed that the proliferation of mycobacteria at 3 and 6 weeks was a reliable model of human TB pathogenesis. The severity of clinical symptoms in guinea pigs that experience chronic onset of TB disease correlates to quantities of viable mycobacteria in relevant tissue. Again, as before this study is limited by the lack of ongoing observation of the animals throughout the disease state. In addition, guinea pigs were fed on their vitamin D diets for only six weeks before they were killed, introducing the possibility that the dietary interventions were not sufficiently long for differences in tuberculin sensitivity to result. However, significant differences in vitamin D plasma levels were observed between the 100% diet and the deficient diets but not the supplemented diet.

In Bowden et al (2014) and in chapter 3 we found that increased intake in oils, fats and sugars was associated with lower TB morbidity. Based on this we speculated as to whether increased adiposity conferred resistance to TB. Chronic wasting has long been associated with TB, but it was not until recently that Wieland et al (2005) established a novel link between malnutrition, circulating leptin, and TB disease in a mouse model. Leptin is a protein that is produced in adipose tissue in mammals that regulates appetite. Mice containing the ob/ob genotype have decreased circulating leptin levels and, if fed *ad libitum*, become obese. In these mice the genomic mutation results in non-functional leptin and lack of signalling of satiety. Low levels of leptin are also associated with reduced adaptive immune function. Reduced spleen and thymus (important for T-cell pathogen adaptation) mass, signs of immunodeficiency consistent with wasting can, therefore, be modelled using ob/ob mice. Ob/ob and wild type mice were exposed to *mycobacteria tuberculosis* and monitored for 28 weeks. The investigators found that ob/ob mice had greater mortality compared with controls, although the result was not significant (P=0.1). The immune responses were initially varied between wild type and ob/ob mice. Wild type mice exhibited well-formed granulomas in their lungs on autopsy

after two weeks. Ob/ob mice on the other hand had impaired adaptive immune response. Instead lung tissue was inhabited by a greater number of immune cells from the innate immune system designed to respond to areas of inflammation within the body. Consistent with thymic atrophy, T-cell activity was also reduced in leptin deficient mice as well as reduced levels of the cytokine, IFN-γ required for the recruitment of immune cells to the site of infection (Wieland, 2005, pp. 1401-1406) The finding that BMI has a consistent inverse log linear relationship with the relative risk of developing active TB thus finds a possible mechanism through the activity of leptin on immune status. One may speculate that, given leptin is present in greater quantities in those with higher BMI, adaptive immune response is strengthened in these individuals and granulomas are better able to contain *mycobacteria tuberculosis* at the site of infection. Unfortunately, the expression of the disease in mice differs to that in humans and as a result, the degree to which these results can be generalised to humans is more restricted than in guinea pig models (Young, 2009, p. 2012, Table 1).

4.5 Conclusions

A lack of investigation into the particular relationship between protein consumption and TB disease in the scientific literature, and a lack of integration of nutritional factors into a generalized approach which recognises TB as an economic as well as an ecological problem alludes to the importance of discoveries made in chapters 1 and 3. Though both chapters investigate a non-individual level cross-section they offer an indication of the importance of standards of living as well as nutrition in real world histories of TB disease. In particular, chapter 1 finds that current innovation in policy reflects standards of living concerns in interwar Britain. Moreover, chapter 3, which directly investigated the effects of different dietary components on TB morbidity for a global cross section of countries, found that calorie consumption was the strongest dietary predictor of morbidity, not protein calories. Lönnroth et al (2008) confirm the importance of greater BMI in reducing risk of developing TB in a meta review of cohort studies linking BMI to TB incidence. Though we do not measure BMI, calories per person may be considered a proxy in population level data and so historical trends find recent corroboration. In fact, our discovery that greater caloric intake of fats, oils and sugars was historically related to lower morbidity in populations is reflected in the recent discovery that greater adiposity might improve immune function.

One of the most consistent findings in scientific literature concerning the relationship between nutrition and activation of human TB disease is that BMI is a strong predictor of risk. Increases in BMI, already under way globally in developing regions, will likely confer some resistance in general to TB disease. However, given the risks of metabolic syndrome posed by increases in BMI, this is not an attractive solution. Micro-nutrient interventions could offer low cost supplements to high risk groups and be effectively stored and distributed in developed, emerging and developing contexts. Unfortunately, micro-nutrient interventions appear not to have any distinct effect, either in animal models or in human studies. An obvious limitation of the science so far has been the lack of human cohort studies which investigate the relative risks of developing TB for a range of different micro-nutrient statuses. These could be used to rule out the efficacy of cost-effective micro-nutrient supplementation. In addition, investigators could ask whether alternatives exist to increasing BMI. In chapter 3, we investigated whether any particular food group conferred benefits when measured in absolute calories or as a percentage of calories consumed. To confirm our findings a subset of a cohort who eat a diet rich in specific whole foods could be compared to controls that do not. It could then be asked whether the synergy of nutrients in the food matrix confers resistance if isolated micro-nutrients do not. There is some indirect support for this notion in Hemila (1999). We also speculated as to whether there was an identification problem within the literature which confounds the effects of vitamin D production with insolation as a mechanism for protection against TB. If insolation produces both vitamin D in human hosts but also creates an inhospitable environment for mycobacteria then it is unknown whether increased vitamin D production or increased insolation is protecting individuals against active disease or both. That vitamin D does not affect quantities of mycobacteria in guinea pig organs suggests our suspicions are well founded.

The last time mycobacteria could operate uninhibited by antimicrobial agents in human bodies policy makers were not cognisant of the economic-epidemiological interactions at work in TB pathogenesis. Now, there exists the potential to mitigate TB disease risk by looking at one of the most important factors predisposing individuals to a disease state: nutrition. Time will tell if DOTS will be an effective strategy to contain the global threat of AMR mycobacterial strains. If it should prove ineffective, our best line of defence against TB could be in the form of targeted nutrition.

5. Background health-related risks and smoking choices in developing and emerging market countries

Alex Sadler

5.1 Introduction

Chapters 1 to 4 investigated the extent to which environments that are risky to health determined morbidity and mortality from tuberculosis. It was found that the more densely people lived, both in terms of general urban living density and in the household, the greater the morbidity and mortality burden both in Britain and in a global sample of countries. Chapter 2 used a qualitative approach to examine the features of living and working environments that facilitated the spread of infection in a developing setting. A recurring theme was that individuals in the developing and emerging world are exposed to living conditions that are risky to health. To a great extent, the conditions under which peoples were infected and then developed active symptoms of the disease were the result of where they lived and when they lived. In terms of the former, our research has shown that in industrialising urbanised environments where living and working conditions were poor and nutrition was undermined, so the risks of acquiring infection were higher. In terms of the latter, an individual was hostage to not only the progress made in reducing infection and curing the disease but also to the commitments of medical agencies and their governments to dealing with the problem. To some extent then the risks of infection and of developing active symptoms were a reflection of factors outside the control of the individual. In this final chapter we wish to concentrate on a case study where the risks are within the control of the individual.

Often in economic decision making it is not appropriate to assume that individuals only face one type of risk and that these risks do not influence their decision making. Health choices are a particular example. An activity that is risky to health such as smoking has a risk directly associated with it, but the decision to smoke is made in the context of other health risks present in the environment. These environmental risks are statistically independent of the risk of smoking, but they may affect the decision to smoke. The theoretical literature, based on Pratt's (1964) characterisation of locally and globally risk averse utility functions, argues that in the presence of background risks, decision makers

will strengthen risk aversion. Gollier and Pratt (1996) established (following some standard restrictions on the utility function) that risk vulnerability is necessary and sufficient for an increase in background risk to generate more risk aversion (Gollier and Pratt, 1996, p. 1121).[32] Eeckhoudt, Gollier and Schlesinger (1996) established the necessary and sufficient conditions to guarantee that any first or second order stochastic deteriorations in background risk induce more risk aversion. Gollier and Pratt's results hold for many forms of risk averse utilities but Eechhoudt, Gollier and Schlesinger require that individual utility functions are decreasing absolute risk averse (DARA). Implicit in the theoretical work is that utility is defined over wealth and not something else. Empirical work follows the theoretical literature by defining utility over wealth and has investigated the role of background risks when risk choices are related to wealth. For example households choose the share of risky assets in their portfolio based on uninsurable income risk and future borrowing constraints (Guiso, 1996).[33] Thus, the evidence suggests that under a few restrictions on utility, that anticipated background risk to wealth (in the form of subjective income uncertainty) reduces the willingness of investors to hold risky assets (Kimball, 1993). Thus, in terms of wealth, the limited empirical work is supportive of the theory. Empirical work, however, has not yet considered the case in which individuals or households are exposed to uninsurable environmental risks to their health. Neither has there been an investigation into whether background risks to health affect the willingness to consume consumption goods that are risky to health. The willingness to consume a good that is risky to health in the presence of background risks to health is analogous to the willingness to hold risky assets in the presence of background risks to wealth. Both contain a choice about the extent to which an individual endowment is subject to risk and both can be measured in terms of utility.

The purpose of this chapter is to investigate the effect of independent background risks to health on the willingness of individuals surveyed in the 2002 World Health Survey (WHS) to participate in a chosen, but independent risk to their health; tobacco consumption. Herein it is proposed that consumption goods risky to health enter

[32] This result applies to the family of absolute and relatively risk averse utility function. Risk vulnerability is defined as when individuals behave in a more risk averse way with respect to another independent risk when an unfair background risk is added to their wealth (Gollier and Pratt, 1996, p.1109).

[33] It has been suggested that their omission in the theory of risk aversion may be responsible for the so-called 'equity premium puzzle' (Weil, 1992).

positively into the individual's utility function, but carry with them some health related risk. In effect the utility they generate is the certainty equivalent 'in-kind.' The WHS data offer a unique opportunity to study real world risk taking behaviours in the context of a richly defined background risk environment across individuals from different countries. It is worth noting that although background risk can be manipulated at will in the experimental setting (Lee, 2008) a full gamut of background risks is available in the WHS. In fact the WHS data may provide more complete evidence for the effects of background risk on risky consumption. These data are valuable not only because they contain variation in the severity of background risk for various environmental categories, but also because they offer a wide range of discrete risk categories, some of which may affect risk aversion at the margin to a greater extent than others. Because simulating risks to health rather than wealth in the experimental setting is subject to ethical constraints and infeasible the WHS data allow field investigation otherwise unavailable to experimental economists.

Section 5.2.1 reports key concepts and results in the theoretical literature. Section 5.2.2 offers discussion of the extant literature that has investigated the empirical basis for the theoretical literature. Section 5.3 discusses how appropriate are the WHS data for discovering the effect of background risks to health on consumption that is risky to health. The format of the data pertaining to health risks is discussed in detail. Section 5.3 also discusses the use of primary sources to supplement the WHS. Section 5.4 discusses the risk factors of the WHS households across sub-samples . Section 5.5 presents the empirical methodology. Section 5.6 discusses the results. Section 5.7 concludes.

Before proceeding, it is worth noting that the present study is primarily concerned with establishing the relationship between background health risks and consumption that is risky to health. It is not concerned with identifying specific channels through which this may operate. Individuals may have utility functions in which the marginal disutility from each lost dollar of earnings is far higher than the marginal disutility resulting from a fall in healthiness. In this case, the primary reason for a reduction in foreground risk-taking behaviour would appear to be income motivated. Since healthiness is entered into the income generating function this may be a competing channel through which risk-taking behaviour is modified.

5.2 Background

5.2.1 Theoretical Literature

The purpose of this section is to present the theoretical motivation for empirical work studying the effect of risks to wealth on risk aversion. It is a truism that risks in the real world do not occur in isolation. In fact individuals are often subject to a multiplicity of risks (or lotteries) which all can be presumed to interact with their decision making. Pratt (1964) investigated the case of one lottery, and articulated the conditions for indifference between the acceptance of a single gamble or receipt of the certainty equivalent. Building on this, microeconomic theory now analyses and predicts behaviour in the presence of multiple risks. The purpose of an investigation of 'properness' is to discern whether or not an individual who faces two undesirable monetary gambles will, if forced to take one, still find the other gamble unattractive. If he does find the other gamble unattractive he has achieved proper risk aversion (Pratt and Zeckhauser 1987, p.143).

Fixed proper and proper risk aversion imply DARA. Suppose that a decision maker faces three independent risks, $\tilde{w}$, $\tilde{x}$ and $\tilde{y}$ with non-random quantities denoted w, x and y where w is wealth and x and y are two arbitrary sources of unfair risk to wealth. Then the decision maker is fixed-wealth proper if $w + \tilde{y}$ is preferred to $w + \tilde{x} + \tilde{y}$. That is, he prefers a situation in which he is not exposed to an additional unfair lottery. More generally if wealth is random too, $\tilde{w} + \tilde{y}$ is preferred to $\tilde{w} + \tilde{x} + \tilde{y}$.

An individual is standard risk averse if any undesirable risk increases the decision maker's sensitivity to independent risks when there is non-random reduction in wealth. Suppose that x aggravates a reduction in wealth such that

$$E[u(w + \tilde{x}) - u(w - \epsilon + \tilde{x})] \geq u(w) - u(w - \epsilon)$$

(Kimball, 1993, p. 592). Aggravation is the situation in which the presence of a risk x ensures that the difference in the expectation following a reduction in wealth, ϵ, is greater than the difference in utility generated by that same reduction in wealth. If ϵ is

made arbitrarily small then the differential result is $Eu'(w + \tilde{x}) \geq u'(w)$. Expected marginal utility is higher in the presence of reductions in wealth. Thus standard risk aversion measures the effect of risks that are expected to increase marginal utility.

Suppose that a decision maker is exposed to a pure risk scenario; that is, a situation in which a mean zero risk is added to his background wealth. Properness has to do with the effect of introducing an unfair gamble, whereas risk vulnerability defines all those utility functions for which an increase in risk makes the decision maker reject risk. Hence properness is a special case of risk vulnerability in the sense that an unfair gamble is never complementary to another unfair gamble or alternatively, that individuals treat independent risks as substitutes.

Risk vulnerability does not require that risks are actuarially unfair and but instead proposes the weakest possible condition on another independent risk; that it has a mean equal to (or less than) zero and non-zero variance. Risk vulnerability can be defined for $Ey \geq 0$ as

$$-Eu''(w + \tilde{y}) \,/\, Eu'(w + \tilde{y}) \geq u''(w)/u'(w)$$

for all w so long as utility conforms to the regularity conditions (Gollier and Pratt, 1996, p.1112). Equation 9 says that when a background risk y is incorporated into expectations, the Arrow-Pratt measure of risk aversion is higher than it would be in the absence of that risk. Local risk vulnerability could, of course apply along some but not all regions of the utility function. This definition pertains to decision makers who are globally risk vulnerable. This definition is satisfying because it generalises the concepts of standardness and properness by showing that the addition of independent risks induces concave transformations in the utility functions of individuals exhibiting DARA (Gollier and Pratt, 1996, p. 1113). These theoretical results have motivated empirical investigation to which we now turn.

5.2.2 Empirical Literature

This section introduces the existing literature that has investigated the effect of independent background risks to wealth on risk aversion. Despite relatively complete

treatment in the theoretical literature, empirical studies investigating this phenomenon are few. Guiso, Jappelli and Terlizzese (1996) suggested portfolio decisions might not be affected exclusively by interest rate uncertainty. They explored whether independent risks, such as uncertain income, might affect the willingness of decision makers to expose themselves to risk using data from the 1989 Bank of Italy Survey of Household Income and Wealth (SHIW). One of the problems that was encountered was finding a suitable variable to measure subjective income uncertainty and another was the lack of information regarding borrowing constraints (Guiso, 1996, p.158). Subjective income uncertainty, it is argued, is reduced in the aggregate, since individual households are hypothesised to be highly idiosyncratic. In other words, the majority of decisions concerning risk are correlated with household specific characteristics. Borrowing constraints too have some relationship with various socio-economic groups and hence, those who are able to borrow with relatively little restraint on liquidity will have different characteristics to those that face far higher constraints. Risky assets are defined as the sum of long-term government bonds, corporate bonds, investment fund units and equities. Safe assets are the remainder. The demand for risky assets is then hypothesised to be related to perceptions of income risk under a number of broad and narrow definitions of risky assets subject to controls. Subjective evaluations of income risk are hypothesised to be related to expectations of inflation and expectations on the growth of wage and pension income. The main finding is that background risk in the form of risk to wages depresses demand for risky assets on both narrow and broad measures. They also find some support for the notion that background risks to health induces a contraction of demand for narrowly defined risky assets using a proxy variable for the number of days that the head of the household was ill. A further finding is that borrowing constraints encourage people to keep their wealth in a more liquid form (Guiso, 1996, p.169). Here, evidence has been presented that portfolio choices are affected by background risk, where background risk takes the form of income risk.

Hochguertel (1998) and Heaton (2000) offer broadly supportive conclusions. In the former it is discovered that Dutch households who report riskier income streams hold safer investment portfolios. The latter builds on Guiso (1996) because it includes the possibility that individuals also hold assets in illiquid form, such as in business capital. It is discovered that when households hold significant business assets, they hold a smaller proportion of their assets in liquid stocks. If business capital is risky, (which it surely is), then entrepreneurial individuals will prefer less exposure to stock market risk.

It is similarly the case when households hold a greater proportion of their employer's stocks. The results in Gollier and Pratt (1996) and Eeckhoudt, Gollier and Schlesinger (1996) are further supported by a recent paper in which Guiso and Paiella (2008) use more recent data, again from the SHIW. Previously their work had omitted consideration of decision maker's endowments when individuals were considering whether to purchase risky assets. Under expected utility, the theory has suggested that risk aversion need not be constant over the domain of endowments. In fact, many observations (and indeed common sense) have suggested that at as total endowment increases, risk aversion diminishes. Using more recent data, Guiso and Paiella (2008) take into consideration the level of endowment and present evidence that supports the notion that decision makers are DARA. This is an important empirical finding because DARA is a necessary and sufficient condition for increases in background risk to increase risk aversion (Eeckhoudt et al. 1996). Hence for first order stochastically dominated deteriorations in background risk, DARA is sufficient to guarantee increased concavity of the utility function and therefore it can be reasonably hypothesised that increases in background risk will increase local foreground risk aversion.

Studies of the nature of risk aversion in the presence of background risk are not limited to studies of cross-sectional field data. Testing for the effects of increased concavity of the utility function of subjects in the laboratory has been pursued most recently by Lusk and Coble (2008) and Lee (2008). Lusk and Coble find that adding abstract mean preserving background risk to an elicitation procedure using artificial monetary outcomes generates more risk aversion. The effect however, is not large but the scale of the effect should be interpreted with caution. The hypothetical monetary rewards are problematic because when used, they tend to reduce to the coefficient of risk aversion vis-a-vis when pay-offs are paid in actual cash (Holt and Laury, 2002). That individuals in the laboratory setting react to background risks to wealth by increasing risk aversion corroborates the findings of non-experimental studies.

Experimental research, in contrast to empirical studies, does not rely on the accuracy of reported income risk (Lee, 2008, p. 19). Lee argues that background risk in the laboratory setting can be calibrated to remove this subjectivity. An important question thus arises; does subjectivity matter in the reporting of background risk? Arguably, the concavity of the utility function is not based on the objective properties of the world, but on the subjective perceptions of each individual. The problem with using subjective

estimates of income risk does not arise from its subjectivity, since this is all the information the individual has to inform their utility, but from the mismatch between subjective perceptions and reporting.

Lee sets up an experiment in which the probability of a participant realising a potential gain is positively related to the amount of the winnings they are willing to give up. They may choose to win with a higher percentage chance, but this entails giving up some quantity of their gain. In this chance improving decision model each participant can then give up the amount of potential winnings that maximises their expected utility. Background risks to initial wealth becomes the treatment variable to ask, again, the question of whether or not individuals become more risk averse in the presence of new and statistically independent risks. In addition, Lee tests whether constant absolute risk aversion applies as wealth is varied. Keeping only the risk averse individuals in the analysis, (for analytical tractability), Lee finds that they are willing to give up more potential gains under background risk. And amongst risk averse individuals that pattern is in favour of DARA, even for moderate stakes.

Empirical studies have not investigated whether the same general conclusions can be obtained when wealth is replaced with health; that is, do increases in background risks to health make individuals behave in a more risk averse way? In this chapter we investigate whether or not decision makers reduce their consumption of tobacco or abstain the greater are the background risks to their health.

5.3 Data

The empirical analysis pursued in this research uses the 2002 WHS. These data contain detailed information on consumption choices risky to health and background risks to health as well as detailed information on household expenditure and demographic characteristics of households. These data can be used to answer the research question; does the presence of independent background risks to health reduce the willingness of respondents (in the aggregate) to engage in consumption that is risky to their health? In addition, we exploit the fact that households are nested within countries by investigating not only exposure to background risks at the household level, but also at the country level. WHS data are therefore supplemented by data from the Uppsala Conflict Data

Program (UCDP, 2013a; UCDP 2013b) and the WHO Statistical Information Service (WHOSIS) for country-specific risks such as armed conflict, aggregated prime-aged adult mortality and public health expenditure. The additional country-level explanatory variables are used to complete the more general background risk environment to which households are subject.

The WHS is a stratified random sample of households collated by WHO in 2002–2004 in partnership with 70 countries to generate information on the health of adult populations and health systems. Of the 70 countries surveyed, 53 report data from the Long version Individual Questionnaire. This questionnaire asks individuals to report risk factors. Of these 53, 52 fully report environmental risk factors.[34] There are 17 developed countries in the survey, from WHO European Region and WHO Western Pacific Region that do not report environmental risk factors. With the exception of Spain, the remaining 52 countries are developing, emerging market or transition economies. Table 5.6 in appendix B provides details of the countries provided in the original 2002 WHS survey, those with data on environmental risks, number of households sampled in each country and their setting. The data regarding both risk taking behaviour and background risks are unusually rich and collected from each household in the form of a written (or spoken) questionnaire by a representative of the household. Sampling methodology is standardised between countries, sample clusters and sample strata where possible. Single stage random sampling is employed if feasible though most sites carry out multi-stage cluster sampling. Although these techniques are commendable the reliability of these survey data are dependent on the truthful responses of participants. The WHS recognises that the mode in which the question is posed may affect respondent choices as well as the presence of other household members in the interview. Despite measures to prevent this many of the questions are of a personal nature. Therefore the WHS is a rich and under-explored source of information about the health risks individuals are willing to take and the health risks to which they are exposed.

The measurement of risk in WHS substantively differs in an important way from the measurement of risk used in the 1989 Bank of Italy Survey of Household income and

[34] Turkey reports Q4000 – Q4038 but not Q4040 – Q4052. There are 17 countries that do not report background risk factors. These countries include Australia, Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Israel, Italy, Luxembourg, Netherlands, Norway, Portugal, Sweden and United Kingdom.

Wealth (SHIW). In the SHIW respondents were asked to assign probability weights, summing to 100, over given intervals of inflation and income in the year ahead. In Guiso (1996) investigators utilised these expectations of income risk and inflation to construct a subjective measure of the variance of real income. Income variance and inflation variance were regressors in a model which sought to explain the proportion of household financial assets held in risky assets. In the current setting, many household included in the WHS are exposed to unavoidable and unfair risks. Here, the assumption is made that households form decisions about current avoidable activities that are risky to health based on current unavoidable health risks that they report in their environment. This is an important difference. In Guiso (1996) the risk measure is a subjective expectation. Here, however, the risk measure is objective and qualitative.

Firstly the WHS contains information on respondent's tobacco consumption habits. Respondents were asked if they currently smoked any tobacco products. They could answer that a) they did daily, b) they did but not daily, and c) they did not smoke at all. For those that did smoke, they were asked for how many years they had been smoking daily and how many products of each type (manufactured cigarettes, hand-rolled cigarettes, pipefuls of tobacco, other) were smoked each day.

Secondly, the WHS contains data pertaining to background risk. Environmental background risk factors on a household level are of a qualitative nature. WHS reports them, in some cases, as a binary variable and in other cases there are a range of possible qualitative responses of varying riskiness. Questions concerning housing quality included; 'what type of floor does your dwelling have?' (either hard floor or earth floor) and 'what type of wall does your dwelling have?' (either cement, mud brick, thatch and other, plastic sheet, metal sheet or other). Questions concerning water security and safety included; 'what is the main source of drinking water for members of this household?' (either piped water through house connection or yard, public standpipe, protected tube well or bore hole, protected dug well or spring, unprotected dug well or spring, rainwater into tank or cistern, water taken directly from pond-water or stream and tanker truck or vendor), 'How long does it take to get there, get water and come back?' (either less than 5 minutes, between 5 and 30 minutes, between 30 and 60 minutes, between 60 and 90 minutes and more than 90 minutes) and 'Are there at least 20 litres of water per person (about one bucket) available per day (for drinking, cooking, personal hygiene etc.) in the household?' (either yes or no). Questions concerning

sanitation included; 'What type of toilet facilities does your household use?' (either flush to piped sewage, flush to septic tank, pour flush latrine, covered dry latrine, uncovered dry latrine, bucket latrine, no facilities or other) and 'How far is the facility from your dwelling?' (either within property / yard used by a single household, within property / yard used by multiple household, outside property / yard in private or outside property / yard shared). Finally, questions concerning cooking and heating included, 'What type of fuel does your household mainly use for cooking?' (either gas, electricity, kerosene, coal, charcoal, wood, agriculture/crop, animal dung, shrub/grass and other), 'What type of cooking stove is used in your house?' (either open fire stove without chimney or hood, open fire or stove with chimney or hood, closed stove with chimney or other), 'Where is cooking usually done?' (either in a room used for living or sleeping, in a separate room used as a kitchen, in a separate building used as a kitchen or outdoors), 'Do you heat your house when it is cold?' (either yes or no), 'What type of fuel does your household mainly use for heating?' (either gas, electricity, kerosene, coal, charcoal, wood, agriculture/crop, animal dung, shrub/grass and other) and 'What type of heating stove is used in your house?' (either open fire or stove without chimney or hood, open fire or stove with chimney or hood, closed stove with chimney or other).

The WHS also asked whether the respondent had access to healthcare the last time they needed it. Access to healthcare was assessed via the question; 'The last time you (your child) needed healthcare, did you get healthcare?' where respondents can either answer yes or no. If no, then respondents are asked a number of yes or no questions about why they did not get healthcare. Whether an individual had access to healthcare or not could also influence their willingness to take health risks. If they have access to healthcare they may believe, to some extent, that the risks that they take have the potential to be insurable.

Though the WHS uses micro-level data, households sampled from different countries have exposure to country-level risks as well. As well as using a binary variable for each country-specific intercept, we also consider country-level variables that could potentially capture the background risk of households. Thus we supplement WHS data with UDCP and WHOSIS data for country-level background risks. We derive from the UDCP data whether or not there was an armed conflict on the territory in which the surveyed households reside between 1990 and 2001 and if so, the number of associate battle-related deaths. Given that risk of death from conflict is an independent risk, this

may induce individuals reduce their overall exposure to risky consumption if they are risk averse. From WHOSIS the adult mortality rate for both genders (probability of dying between 15 and 60 years per 1000 population) is derived. Household respondent's subjective interpretation of their risk environment might be informed by the prevailing prime-aged mortality rates in their country of origin. WHOSIS also provides per capita government expenditure on health care (PPP int. $). The importance of this explanatory variable is discussed below.

Data indicating whether households were in territory in which conflict had taken place between 1990 and 2001 was derived from the Uppsala Conflict Data Program (UCDP) / Department of Peace and Conflict Research Institute in Oslo (PRIO) Armed Conflict Dataset. The binary variable takes a value of 1 if either extra-systemic, interstate, internal or internationalised internal conflict occur within the territory that surveyed households reside.[35] In addition, the binary variable is interacted with the calculated average annual UCDP 'best estimate' for battle-related deaths in the conflict. Battle-related deaths are those deaths which are directly related to combat over the contested area. This includes deaths from traditional fighting, guerilla activities, bombardment of military and civilian sites, urban warfare of both military personnel and civilians. Battle-related deaths are distinguishable from war-related deaths, which include indirect deaths due to starvation, criminality or deliberate attacks against civilians only (UCDP, 2005). If a conflict began before 1990 but continued into the 1990s, deaths in previous years are included in the calculation of the average annual number of deaths for that particular conflict.[36] Battle-related deaths from concurrent conflicts are added to the annual total.

The natural log of per capita government expenditure on health was derived from WHO Statistical Information System (WHOSIS) database for the year 2001, the year before the WHO began collecting WHS data in 2002. The purpose of this variable is to capture the extent of the public health safety net. Data on estimated health expenditure were

[35] An extrasystemic conflict is defined as a conflict between a state and a non-state group outside the territory of the state. Interstate conflict is defined as a conflict between two or more governments. Internal conflict is defined as a conflict between a government and a non-government party and internationalised internal conflict is a conflict which spills over into the territory of another state.

[36] UCDP defines conflict as “a contested incompatibility that concerns government and/or territory where the use of armed force between two parties, of which at least one is the government of a state, results in at least 25 battle-related deaths.”

collected by WHO from several sources. Where it was not possible to obtain data on expenditure for local government, other ministries besides health, and extra budgetary items, some figures may be underestimated. The adult mortality rate is defined by WHOSIS as the probability of dying between 15 and 60 years per 1000 population in a given country for the year 2000.[37] This year was chosen because it is the most recent year for which data were collected prior to the beginning of the WHS. Given the lack of reliable mortality data for low and middle income countries, especially among adults and the elderly, WHO applies modelling techniques, based on data from other populations, to estimate life tables. The extent to which respondents are covered by public health provision is important if it is perceived by the individual to represent an insurance against behaviour that is risky to health. If individuals have the expectation that the potential costs of their decision to smoke may be partially covered then they may be more inclined to participate in the activity or participate more intensely even if they are risk averse.

A set of controls are available for each household in order to control for household heterogeneity. These include gender, age, age squared, year of formal education, marital status, job type and household expenditure. Controlling for variation in respondent characteristics is important because of the potential effect of tastes on risk preference (Dohmen, 2005).

Finally, household expenditure is an important predictor of the quantity of tobacco products a household can consume whilst remaining within budget. However, expenditure measures are problematic in WHS. Expenditure is reported at the household level in individual country currencies in 2002-4 prices and includes payment for goods and services in both cash and in kind. For comparative analysis the values are converted into 2005 international US dollars from local currently units using the World Bank, International Comparison Program Database. That different countries in the WHS were sampled during different years is taken into account. Purchasing power parity, private consumption (LCU per international $) conversion factor is the number of units of a country's currency required to buy the same amounts of goods and services in the domestic market as U. S. dollars would buy in the United States. The conversion factor is for private consumption; that is, household final consumption expenditure (World

[37] See http://apps.who.int/whosis/data/.

Bank, 2013).[38] Table 5.1 provides comprehensive information on each of the variables.

5.4 The risk factors of households surveyed in WHS

The way in which data on tobacco consumption were collected in WHS is problematic if the purpose is to use the data to construct a left-censored dependent variable. If respondents smoked daily, the number of tobacco products consumed each day is recorded. If respondents smoked, but not daily, the number of tobacco products consumed is not recorded and so these observations are erroneously censored at zero. Finally, those that did not smoke are recorded at zero. One option would be to estimate a tobit model where known smokers are considered non-smokers because nothing is known about the quantity smoked. This approach would bias estimated coefficients by increasing the number of corner observations in the sample above those in the population. The alternative is to drop observations and risk inconsistent estimates. One possibility is that among those that did smoke, but not daily, the distribution of the number of products smoked is similar to that of the remaining two groups. Another possibility is that those that did not smoke daily were likely to smoke less than those that smoked daily but not so few as to be indistinguishable from those that did not smoke at all. Because only 6.7% of respondents replied that they smoked, but not daily, the observations are dropped from the analysis. Full survey sample characteristics and selected sample characteristics are reported in table 5.2. In addition, observable sample characteristics are reported for the sample for which observations are complete for tobacco.

Sample selection bias arises if non-responses about environmental risks are not randomly distributed throughout the population. Column (1) of table 5.2 reports the proportion of the total sample exposed to specific risks. Column (3) reports the sample that excludes those that smoked, but not daily, and for which complete observations are available for a regression on TOBACCO. This sample has a slightly higher risk profile than the total sample. In this sample 17% of households access water via protected well or borehole whereas in the total sample it is 13%. Similarly, a higher proportion of

[38] See http://data.worldbank.org/indicator/PA.NUS.PRVT.PP for description.

households in the regression sample have earth floors, mud brick walls, no toilet facilities and cook using either agricultural crops, animal dung, shrubs or grass. In general these differences are small. Larger differences between selected and total samples occur for household expenditure and number of children under the age of 5. For example, expenditure in the tobacco regression sample is 38% of the total sample. Discarding households that report above PPP 2005 $1 million in monthly household expenditure produces a mean expenditure of $432.47. Total sample figures are primarily distorted by an Ecuadorian household reporting monthly expenditure in excess of PPP $100 million in the WHS.

The number of children under 5 increases by 0.06 children from 0.18 per household in the total sample and by 0.08 to 0.26 in the tobacco sample. In addition, in regression samples, respondents have completed 5 to 6 months less formal education. These differences are largely attributable to survey design. For example, Mexico was surveyed under WHS but very few respondents gave a response concerning how far toilet facilities were from their dwelling and so these observations are incomplete. Because of its large population, Mexican households were extensively sampled. The omission of many Mexican households in the regression samples produces sample means of the number of children under 5 in the household, years of education and monthly household expenditure that are significantly different to those in the total sample.

5.5 Empirical Methodology

The demand for health risks is modelled as a two stage decision process. First, individuals must decide whether to consume tobacco at all. If they do, then they decide in what quantity they wish to consume these goods. The latent variable describing the choice to consume goods risky to health is not observed and hence many individuals choose the corner solution by consuming zero quantity. The standard tobit model can address the problem of biased estimates in the presence of a truncated dependent variable. However, the decision to smoke at all is likely a different decision process to choosing how much to consume. The standard tobit model imposes that the marginal probability of consuming some quantity of either good is some positive multiple of the estimating parameter. It also imposes that the expectation of the quantity consumed, if the quantity consumed is positive, is also a positive multiple. Thus in both the

participation probability and the intensity equation the estimated parameter of any variable must have the same sign. One can imagine, for example, that background risks reduce the probability that individuals will choose to smoke for example, but that once individuals do begin to smoke daily, addiction or fatalism cause a reversal in attitudes towards risk. Alternatively, some individuals may choose to abstain no matter how small their background risk exposure. The standard tobit model would, in such a circumstance, suffer from misspecification.

Inspection of a frequency plot of the quantity tobacco consumed suggests that the corner solution model may not be appropriate (See figures 5.1 and 5.2). Not only is there a relatively large cluster of observations at zero,[39] but relatively few observations are clustered near to zero. The relative frequency of observations clustered at zero is also large compared to frequencies observed in the positive part of the distribution for both variables. The data, therefore do not take the form of a distribution that is truncated at zero. Forcing a standard tobit model on these data will therefore likely distort the estimator.[40] This suggests that the double hurdle model (sometimes referred to as Cragg's model (1971)) is appropriate.[41] In this model, the decision to consume goods that are risky to health can be determined by a different subset of the variables to those that determine the quantity consumed. The first "hurdle" needs to be crossed if the individual is to be a potential smoker at all. Socio-demographic characteristics of individual respondents as well as background risk factors are hypothesised to be correlated to the decision to abstain. Given that respondent is a smoker, the "second hurdle" is the extent to which current demographic and risk circumstances influence the quantity smoked. The double hurdle model for individual i contains two equations:

$$d_i^* = \boldsymbol{x_{1i}}'\gamma + \varepsilon_i$$

$$y_i^* = \boldsymbol{x_{2i}}'\beta + u_i$$

and

[39] 107908 of 125154 of respondents reported that they do not smoke.
[40] See Greene 2012 pp. 852 – 857.
[41] The double hurdle model also assumes conditional independence of the latent variable's distribution. That is, given the data, the distribution of the quantity of tobacco consumed should be independent of the decision to consume tobacco given the data. See figures 5.1 and 5.2.

$$(\varepsilon_i, u_i) \sim bivariate\ normal\ (\rho = 0)$$

$\boldsymbol{x_1}$ is a vector of covariates which determine the decision to smoke, d_i^* and $\boldsymbol{x_2}$ determine the quantity to be smoked once the decision to smoke has been made, y_i^*. An asterisk indicates a latent variable. The variance of ε_i is normalised to 1 and both errors ε_i and u_i are assumed independent. Thus the first hurdle observation rule is given as

$$d_i = 1\ if\ d_i^* > 0$$

$$d_i = 0\ if\ d_i^* \leq 0$$

The individual is observed to smoke if the latent variable d_i^* is greater than zero. The second hurdle observation rule is given by

$$y_i^* = 0\ if\ d_i = 0\ and\ y_i^* = y_i\ if\ d_i = 1$$

That is, the quantity consumed will be zero if the decision is to abstain from smoking, and the quantity consumed will be equal to the latent variable if the individual chooses not to abstain. The nature of respondents who do not smoke may differ in an important way. Some non-smokers fall into the category of those people that would never smoke regardless of their circumstances because of their idiosyncratic preferences. Others fall into the category of those people who would smoke, but because of their circumstances choose a consumption level of $y_i^* = 0$. If their situation was more favourable, for example if they had greater income, then they would choose some quantity, $y_i^* > 0$. As discussed inspection of the frequency distribution of TOBACCO suggests that both of these groups are represented in the sample since there are a large number of observations at zero, but also a relatively large number limiting observations near zero.

Tables 5.3 and 5.4 present the results of the censored regression model, probit, tobit and

double hurdle estimation for TOBACCO. Columns (1), (2) and (3) of table 5.3 give estimated coefficient values and standard errors of the censored regression model, the probit model and the tobit model respectively. Column (4) in table 5.3 gives the tobit coefficients divided by the estimated standard error (σ). Columns (5) and (6) in table 5.4 give the estimated coefficients on the participation likelihood and the intensity of participation using the standard double hurdle model and (7) and (8) report results for the Box Cox transformed double hurdle model.

The log-likelihood for the standard double hurdle model is given by

$$logL = \sum_{0} \ln[1 - \Phi(\boldsymbol{x_{1i}}'\gamma) \cdot \Phi(\boldsymbol{x_{2i}}'\beta)] + \sum_{+} ln[\Phi(\boldsymbol{x_{1i}}'\gamma) \cdot (1/\sigma) \cdot \phi((y_i - \boldsymbol{x_{2i}}'\beta)/\sigma)]$$

The tobit model is nested in the double hurdle model and is equivalent to the double hurdle model if $\boldsymbol{\gamma} = \boldsymbol{\beta}/\sigma$ and $\boldsymbol{x_1} = \boldsymbol{x_2}$ where $\boldsymbol{\gamma}$ is the vector of coefficients to be estimated in the participation equation and $\boldsymbol{\beta}$ is the vector of coefficients to be estimated for the intensity equation.[42] Here, however, some variables that determine the decision to smoke are considered not to affect the quantity smoked. GENDER, MARRIAGE, EDUCATION, CHILD and *JOB* are included in the participation equation but not the intensity equation. EXPENDITURE is included in the intensity equation, but not the participation equation. Column (3) in table 5.3 reports the tobit estimates for which all variables are included. A test for whether the double hurdle model is appropriate is available. The tobit model is the sum of the log-likelihoods of the truncated and probit regression models. The purpose of estimating the probit and the truncated regression models separately is so that it can be discovered if the participation and intensity

[42] Note that $f(w, y\,|\boldsymbol{x_1}, \boldsymbol{x_2}) = [1 - \Phi(\boldsymbol{x_1'}\gamma)]^{1(w=0)}$ · $[\Phi(\boldsymbol{x_1}'\gamma)(2\pi)^{-(1/2)}\,\sigma^{-1}exp\{-(y - \boldsymbol{x_2}'\beta)^2/2\sigma^2\}\Phi(\boldsymbol{x_2}'\beta/\sigma)]^{1(w=1)}$ where w is an indicator variable equal to 1 if y is positive and 0 otherwise. This expression integrates both the probit expression for the probability that y will be larger than zero and the truncated regression model of the conditional density for values of y greater than zero. The double hurdle model is a generalisation of the tobit because it allows for the contribution of observation i to the likelihood function to be conditioned by different variable vectors in the participation and intensity equations and for each associated coefficient to be estimated independently.

decisions are separate decision processes. If the sum of their log-likelihoods differ significantly from the log-likelihood of the tobit model, then the tobit model does not account for differences in the decision to participate from how much to consume and is misspecified. The likelihood ratio (LR) test computes a test statistic as $\Gamma = -2[lnL_T - (lnL_P + lnL_{TR})]$ distributed as $\chi 2$ where the subscript T denotes the tobit model, P the probit model, and TR the truncated regression model (Greene, 2012, p. 853). The test statistic is distributed as $\chi 2$ (98). The null is rejected if $\Gamma > \chi 2$ (98) critical value 1% value. The test statistic is 9790.42 which is greater than the critical value of $\chi 2$ (98) $\approx$ 133.48 at the 1% level. Therefore the assumption of the tobit model that the parameters are constant across both the decision to smoke and how much to smoke is not validated. Column 4 gives the tobit model divided by the estimated standard errors. If the tobit model is correct then dividing by the standard errors should return the probit coefficients. Casual observation of the coefficients reveals that tobit/σ roughly returns the probit coefficients but their statistical similarity is rejected by the more formal LR test.

Just as the tobit model depends on the normality of the errors, the double hurdle model requires that the errors in the participation and the intensity equation are jointly normal under the assumption that both the decision to smoke and the decision of how much to smoke are made jointly. A departure normally distributed errors implies maximum likelihood estimates are no longer consistent. A Lagrange multiplier (LM) test for the normality of the errors for data that is left-censored at zero is available after tobit estimation (Drukker, 2002). The null hypothesis is that the error term is homoskedastic and normally distributed. The LM value is 1530.6 compared to the (scaled) critical value at the 1% level of 209.89 implies rejection of the null.[43] Figure 5.2 reveals that the dependent variable exhibits positive skew with a long tail which also suggests non-normality. Given the rejection of the null in the LM test that $\lambda = 1$ a Box – Cox the double hurdle model is estimated using the Box-Cox transformed likelihood function in which λ is a separate parameter that requires estimation. The log likelihood function of the Box Cox double hurdle model is given by

[43] Testing for normality of the errors after tobit estimation is achieved by taking a Box – Cox transformation of the dependent variable $y^T = (y^{\lambda-1})/\lambda$., where under the null hypothesis $\lambda = 1$. Critical values were obtained via parametric bootstrap with 499 replications.

$$logL = \sum_{0} \ln\left[1 - \Phi(\boldsymbol{x}'_{1i}\gamma) \cdot \Phi\left(\boldsymbol{x}'_{2i}\beta + \frac{1}{\lambda}\right)\right] + \sum_{+} \ln\left[\Phi(\boldsymbol{x}'_{1i}\gamma) y_i^{\lambda-1} \cdot \left(\frac{1}{\sigma}\right) \cdot \phi\left(\frac{y_i^T - \boldsymbol{x}'_{2i}\beta}{\sigma}\right)\right]$$

where λ is the parameter that is chosen by the Box Cox transform so that the resulting skewness of the new variable, y^T, is zero and $y_i^{\lambda-1}$ is a required Jacobian term. The Box Cox transformed variable is $y^T = (y^{\lambda-1})/\lambda$.[44]

There are a number of reasons to control for the effect of age on the decision to participate in consuming goods risky to health. One reason may be that the young are more likely to take health risks in the present because the consequences are most distant in time. Conversely, the old may have less incentive to modify their health behaviours because their time horizons are much shorter. The young may also be less educated about health risks, a process that requires gathering information over time (Hsieh, 1998). A second-order polynomial is used to model the effect of age. Also included are a number of demographic controls (marital status, gender, education, number of children under 5 and job type) which are used as proxies for taste heterogeneity. Household expenditure is used as a proxy for wealth. Years of education may also be an important determinant of abstention.

Medical expenses that cannot be insured may also affect the demand for goods risky to health. Since households are grouped by country in WHS, this information is used to investigate if there are country-specific effects on risk taking. The level of public health spending in the country of residence may act as a form of insurance against the associated risks and incentivise risk taking. Not only is the level of public health spending important but the ease with which it can be accessed. Some WHS participants reported that they were unable to access healthcare in the past. In practice these individuals take health, regardless of public health expenditure, that might be uninsurable in their location. Therefore a dummy for whether or not individuals received healthcare last time they tried to get it is included. Finally, the prevailing mortality rates in the country of origin may influence individual decision making in

[44] The Box Cox double hurdle model is estimated using programming for STATA v.6 suggested by Moffatt (2005) appendix A which has been updated in the present study to be applicable to coding rules in STATA v.11.

either direction. CONFLICTDEATH proxies perceptions concerning the probability of dying as a consequence of conflict. Perceptions concerning the likelihood of dying before old age are proxied by the adult mortality rate. Increases in these country specific background risks may incentivise, in the average, individuals residing in those locations to reduce their demand for risky consumption goods. Equally, it could be that higher rates of mortality in the adult population or a higher risk of dying in conflict could produce fatalistic decision making and result in increased risk taking where futures are uncertain.

5.6 Results

Box Cox double hurdle models are suitable for situations where individuals are heterogeneous in their decision making process. There are some individuals that do not wish to smoke at all. In their case the decision to participate dominates the decision of how much to consume. But there are also individuals in the sample who may wish to smoke, but for whatever reason (perhaps budget), they do not and therefore choose zero consumption as a corner solution. It has been argued that once individuals have decided to smoke the question is simply one of how much to consume (Garcia and Labeaga, 1996). Figure 5.2, however, indicates that there is still a large number of individuals near to the corner solution, (despite the very large number of observations *at* zero), indicating that tobit censoring remains relevant for some individuals in the distribution. The implication is that both types of individuals are represented in the sample and that a model is required that accommodates both.

The hypothesis was that households with greater exposure to unavoidable health risks would choose to bear fewer foreground health risks. The results obtained in the Box Cox double hurdle regression suggest that behaviour does not follow the predicted pattern. Compared to those with cement, brick, stone or wood walls, those with thatch or plastic walls are more likely to participate in tobacco consumption in (7). Living in a dwelling with poor quality walls correlates to a higher likelihood of participation but a lower quantity consumed for plastic and thatch. Greater health risk from having an earthen floor also does not significantly reduce the likelihood of consuming tobacco products in (7).

A pattern emerges when investigating the role of the source of water and on the type of toilet facilities on participation behaviour. For *WATER* variables that are slightly riskier than "piped water through house connection or yard" the effect on the likelihood of participation is moderately positive and largely insignificant. However, as the riskiness of the water source increases from a protected well to the rain and finally to a pond, the individual is increasingly likely to consume tobacco products. For *TOILET* variables a similar pattern is observed in (7). Those with toilet facilities slightly more risky that the reference category of a flush toilet connected by pipe to a sewage system react to this risk by reducing tobacco consumption. Those with a toilet that flushes to a septic tank are significantly less likely to participate and the coefficient on pour flush latrine and covered latrine is also negative but not significant. As riskiness increases so the coefficients become positive. Those with uncovered latrines are significantly more likely to consume tobacco products. The WHS considers the location of toilet facilities to be an environmental risk factor. Again a similar pattern emerges. When the risk is only slightly above that of safest category; a toilet within the dwelling, the individual is less likely to participate. However, when the toilet is no longer private either inside or outside the dwelling, and especially when shared outside, individuals are more likely to smoke.

Few of the remaining environmental risk factors, including the location of the kitchen, the type of stove used for cooking and heating, the type of fuel used for cooking and heating significantly affect the likelihood that an individual will participate in risky consumption or attenuate the quantity consumed. Whether or not an individual has access to at least 20 litres of water per day (WATER20) does not significantly influence the participation decision in (7) but it does affect the intensity of consumption. If water was available then individuals in the sample smoked more. That individuals with access to at least 20 litres of water smoked more may not be because they have a higher tolerance of foreground risk but rather that access to sufficient water is a proxy for wealth. If tobacco is a normal good then one would expect a proxy for wealth to be positively correlated with the quantity consumed. This objection could equally apply to other variables that are related to household wealth. There is no consistent pattern, however, between other risk factors associated with wealth and the quantity consumed. In (8), for example, those who use a pour flush latrine consume less than those will a flush toilet to piped sewerage but those with a toilet outside the dwelling consume more.

In general, environmental risks influence the participation decision and not the consumption decision. Relative to demographic and socio-economic controls background risk factors are not strong determinants of either the participation or the consumption decision in (7) and (8). As discussed, these are qualitative and unordered categorical variables (except the time it takes to go, collect water and return) which means that individual household environmental risks must be accounted for using binary variables in order for correct empirical specification. Environmental health risks may not individually affect decision making in an economically significant way whereas broader wealth risks might (Guiso et al. 2008). One option would be to reduce the number of binary variables used to describe the categorical groupings of the WHS questionnaire. For example, households that do not have a flush toilet to piped sewerage are the reference category and all other types of toilet facility is the other category. This method might produce economically significant results but at the expense of ignoring trends within the risk categories that allude to non-DARA utility functions among those exposed to the highest risks.

Environmental risks are also correlated which might reduce their individual statistical significance despite their overall significance in regressions in tables 5.3 and 5.4.[45] As a result the benchmark model reported in (7) and (8) in table 5.4 is reproduced for various categories of the environmental risk data. Columns (9) and (10)in table 5.5 report results using variables in *FLOOR* and *WALL*, columns (11) and (12) for *WATER*, columns (13) and (14) for *TOILET* and *TOILETLOCATE*, columns (15) and (16) for *COOKING*, *STOVECOOK* and *KITCHEN* and columns (17) and (18) for *HEATING* and *STOVEHEAT*. Adopting this approach reveals a clear pattern. The presence of environmental risk increases the likelihood of participation but reduces the quantity consumed if individuals decide to participate. Again, where qualitative measures of risk are greater so individuals tend to be more likely to participate compared lowest risk reference groups. These results do not confirm the hypothesis that individuals exposed to background risk will reduce their foreground risk taking.

Whether or not a household had access to healthcare last time they tried is an indication of whether a survey participant believed that their health was insurable. If health risk is

[45] A matrix of tetrachoric correlations estimated by MLE and their p-values revealed significant relationships between environmental binary variables. For example, the tetrachoric correlation between having a mud floor and using agricultural waste to cook is 0.82 (std. error 0.002). The table, because of its size, is not reported.

not insurable then it may induce people to reduce demand for consumption goods risky to their health. The evidence in (7) suggests that if people did not have access to healthcare last time they tried to get it (ACCESS) then they are less likely to consume tobacco products at all. Individuals reacted to health uncertainty in the participation decision, but the statistical evidence does not suggest that health uncertainty provokes individuals to curtail their consumption once the decision to participate has been made. This observation is also made in the restricted model. A related variable, the log of the per capita $ PPP 2001, (PUBLICHEALTH) spent at the country level, also conveys a similar effect. Individuals that live in countries with higher per capita healthcare spending are not only more likely to participate, but also consume more according to (7) - (18). Potentially, this variable could be capturing a wealth effect. Otherwise, it may be that it represents the reduction in health-related costs associated with illness and therefore, the extent to which health risks are insured.

That surveyed household data are divided into countries provides an opportunity to investigate country level variables that are a source of health risk. Annual deaths from conflict between 1990 and 2001 ranged from 12206 in Ethiopia to 0 in Brazil, Burkina Faso, China, Côte d'Ivoire, Czech Republic, Estonia, Ghana, Kazakhstan, Kenya, Malawi, Malaysia, Mali, Mauritania, Mauritius, Morocco, Namibia, Paraguay, Slovakia, South Africa, Swaziland, Tunisia, UAE, Ukraine and Vietnam as defined by UDCP (see table 5.1). The coefficient on CONFLICTDEATH suggests that where there is conflict in the present or where there has been conflict in the past individuals are less likely to participate and if they do participate will consume less. Similarly, if there is a high rate of adult mortality between the ages of 15 and 60 (ADULTMORT) in any given country, survey respondents are less likely to participate.

Given the poor quality of wage data in WHS (often because of refusal to answer the interviewer's question or inconsistencies between reported weekly, monthly and yearly wages) expenditure data is a natural proxy. It is surprising, however, that household expenditure bears no relationship to the quantity consumed in either the benchmark model in (7) and (8) or the restricted models in table 5.5. Expenditure data were difficult to construct for the sample (see table 5.1) and also depended on individuals accurately reporting their level of expenditure over the period defined by the interviewer. Omitting variables from (7) – (18) which are ostensibly related to income does not alter the

significance of the coefficient on expenditure in the consumption equation.[46] Monthly expenditures may not be a suitable proxy for income especially if reported solely for the month prior to the survey interview. For these reasons, results should be interpreted with caution.

5.7 Conclusion

The theoretical literature has offered understanding of consumer decisions in the presence of independent risks to income, rate of return risk and portfolio choice under some assumptions regarding the form of the utility function. In this chapter, tobacco consumption was a proxy for avoidable health risk and idiosyncratic household environments, as well as country level risk factors were proxies for unavoidable background risk to health. Chapter 5 has offered evidence, however, using the 2002 World Health Survey to suggest that some individuals are less likely to abstain from tobacco consumption the *greater* the more unfavourable is the environmental "background" risks to health. It is found that when individuals are presented with unavoidable background risks that they are often more likely to smoke, and as environmental risks become more risky compared to the reference category, that likelihood increases further. Once the decision to smoke has been made, the presence of background risks tends to attenuate the quantity consumed.

By contrast evidence suggests that in the presence of greater public health provision, individuals are also more likely to smoke. Thus the implied reduction in background risk to either their wealth or their health motivates an increased willingness to smoke. Within the WHS sample the evidence suggests that individuals are not DARA with respect to their health. However, that individuals abstain more and consume less when their wealth is subject to greater risk in the absence of public health it is consistent with evidence from other empirical studies. That individuals consume fewer tobacco products the lower the public health provision, but more in the presence of environmental risks, suggest DARA may only hold over wealth and not health.

[46] Regressions were run omitting job-type and environmental risks individually and together. These regressions were run on the whole regression sample and also on a sample omitting extreme positive outliers in the household expenditure data (99 observations). None of the reported coefficients on EXPENDITURE were significant at the 5% level.

Consumers that reduce their consumption may be doing so in response to background risks to wealth, not health. Whether this phenomenon for health is an effect of the discount on future health states or and effect of risk-loving is unknown (Anderson and Mellor, 2008).

It is apparent from the above analysis that risk attitudes towards health vary from those towards wealth. Any characterisation of the decision to smoke in the context of background risks is complicated by the addictive qualities of the good itself, the fatalistic nature of decision making when environmental risks are high (Bernard et al., 2011) and the fact that health risks are often sources of utility in the short run but sources of unfair risk long run. However, the analysis is simplified by the fact that health is a non-transferable asset. Taking these factors into consideration in a theoretical approach that considers risk aversion over health is needed to understand why, on the surface, risk taking in these two realms is so divergent.

5.8 Appendix A

Table 5.1 Variable name, type, definition, related survey question and notes

	Variable type	*Definition*	*Survey Question and Notes*
Dependent Variable			
TOBACCO	Continuous if >0, 0 otherwise.	The number of tobacco products smoked daily.	Do you currently smoke any tobacco products such as cigarettes, cigars, or pipes? 1. Daily 2. Yes, but not daily 3. No, not at all. If daily, then how many manufactured cigarettes, hand-rolled, cigarettes, pipefuls of tobacco or other smoked each day? Note: Number of tobacco products smoked calculated as the sum of daily individual product consumption.
Independent Variables			
Household Environmental Risks			
FLOOR	Binary	1 if household has an earth floor, 0 otherwise.	What type of floor does your dwelling / house have?
WALL	*Categorical*		*What type of wall does your dwelling / house have?* *Note: The reference category is "cement, brick, stone or wood" wall.*
WALLMUD	Binary	1 if household has mud brick walls, 0 otherwise.	
WALLTHATCH	Binary	1 if household has thatch walls, 0 otherwise.	
WALLPLAST	Binary	1 if household has plasctic sheet walls, 0 otherwise	
WALLMETAL	Binary	1 if household has metal sheet, 0 otherwise.	
WALLOTHER	Binary	1 if household has other type of wall, 0 otherwise.	
WATER	*Categorical*		*What is the main source of drinking water for members of this household?* *Note: The reference category is "piped water through house connection or yard."*
WATERPUBLIC	Binary	1 if access to water is via public standpipe, 0 otherwise.	

Table 5.1 continued …

Household Environmental Risks cont…	***Variable type***	***Definition***	***Survey Question and Notes***
WATERTUBE	Binary	1 if access to water is via protected tube well or bore hole, 0 otherwise.	
WATERPRO	Binary	1 if access to water is via protected dug well or protected spring, 0 otherwise.	
WATERUNPRO	Binary	1 if access to water is via unprotected dug well or spring, 0 otherwise.	
WATERRAIN	Binary	1 if household collects rainwater (into tank or cistern), 0 otherwise.	
WATERPOND	Binary	1 if water taken directly from pond-water or stream, 0 otherwise.	
WATERTANKER	Binary	1 if access to water is via tanker truck or vendor, 0 otherwise.	
WATERTIME	Interval-censored (imputed continuous)	Minutes spent collecting water and bringing it back home.	How long does it take to get there, get water and come back? Household respondents chose interval; <5 minutes, between 5 and 30 minutes, between 30 and 60 minutes, between 60 and 90 minutes and >90 minutes. Note: The imputed continuous variable is computed following Royston (2007). Lower limit 0 for <5 minute category. Upper limit 360 minutes for >90 minute category. Imputed regression of WATERTIME on GENDER, AGE, FLOOR, EXPENDITURE, TOBACCO, EDUCATION. Only answered if household does not use piped water through house connection or yard.
WATER20	Binary	0 if 20 litres of water per person available per day in the household, 1 otherwise.	Are there at least 20 litres of water per person (about one bucket) available per day (for drinking, cooking, personal hygiene etc.) in the household? Note: Only answered if household does not use piped water through house connection or yard.
TOILET	*Categorical*		*What type of toilet facilities does your household use?* *Note: The reference category is flush to piped sewage system.*
TOILETSEPTIC	Binary	1 if flush to septic tank, 0 otherwise.	
TOILETPOUR	Binary	1 if pour flush latrine, 0 otherwise.	
TOILETCOV	Binary	1 if covered dry latrine, 0 otherwise.	
TOILETUNCOV	Binary	1 if uncovered dry latrine (without privacy), 0 otherwise	
TOILETBUCKET	Binary	1 if bucket latrine (where fresh excrement are manually removed), 0 otherwise.	
TOILETNONE	Binary	1 if no facilities, 0 otherwise.	

Table 5.1 continued …

Household Environmental Risks cont …	***Variable type***	***Definition***	***Survey Question and Notes***
TOILETOTHER	Binary	1 if other, 0 otherwise.	
TOILETLOCATE	*Categorical*		*How far is the facility from your dwelling/house?* *Note: The reference category is within property / yard, used by single household.*
TOILETSHARE	Binary	1 if toilet within property / yard, used by multiple households, 0 otherwise.	
TOILETOUT	Binary	1 if toilet outside the property / yard and private, 0 otherwise.	
TOILETOUTSHARE	Binary	1 if toilet outside property / yard and shared, 0 otherwise.	
COOKING	*Categorical*		*What type of fuel does your household mainly use for cooking?* *Note:The reference category includes gas and electricity.*
COOKINGCOAL	Binary	1 if household uses kerosene, coal, charcoal or wood to cook, 0 otherwise.	COOKINGCOAL consolidates households that use kerosene, coal, charcoal or wood into one variable. These are separate categories in original WHS returns.
COOKINGCROP	Binary	1 if household uses agriculture / crop, animal dung or shrubs / grass or other to cook, 0 otherwise.	Households that use agriculture / crop, animal dung or shrubs / grass. These are separate categories in original WHS returns.
STOVECOOK	*Categorical*		*What type of heating stove is used in your house?* *Note: WHS omits these risk factors if households use either electric or gas fuel. In which case all three variables below are recorded as 0.*
STOVECHIMNEY	Binary	1 if household uses closed stove with chimney to cook, 0 otherwise.	
STOVECOOKOPEN	Binary	1 if household uses open fire or stove to cook, 0 otherwise.	
STOVECOOKOTHER	Binary	1 if other cooking method used, 0 otherwise.	
KITCHEN			*Where is cooking usually done?* *Note: KITCHEN is conditional on either COOKINGCOAL=1 or COOKINGCROP=1. WHS omits these risk factors if households use either electric or gas fuel. If electric or gas is used KITCHEN is zero for all variants.*
KITCHENLIVING	Binary	1 if cooking occurs in a room used for living and sleeping, 0 otherwise.	

Table 5.1 continued …

Household Environmental Risks cont …	***Variable type***	***Definition***	***Survey Question and Notes***
KITCHENSEPARATE	Binary	1 if cooking occurs in a separate place, 0 otherwise.	*Where cooking is done "in a separate room used as a kitchen" or "in a separate building used as a kitchen."*
KITCHENOUTDOOR	Binary	1 if cooking occurs outside, 0 otherwise.	
HEATHOUSE	Binary	1 if household is heated, 0 otherwise.	Do you heat your house when it is cold?
HEATING	*Categorical*		*What type of fuel does your household mainly use for heating?* *Note: Gas or electricity is the reference category. HEATING is conditional on HEATHOUSE. WHS excludes these risk factors if households answer "no" to HEATHOUSE.*
HEATCOAL	Binary	1 if kerosene, coal, charcoal or wood used for heating, 0 otherwise.	
HEATCROP	Binary	1 if agriculture / crop, animal dung or shrubs / grass or other used for heating, 0 otherwise.	
STOVEHEAT	*Categorical*		*What type of heating stove is used in your house?* *Notes: STOVEHEATOPEN is conditional on either HEATCOAL=1 or HEATCROP=1 which are themselves conditional on HEATHOUSE=1.*
STOVEHEATCLOSED	Binary	1 if a household uses closed stove with chimney, 0 otherwise.	
STOVEHEATOPEN	Binary	1 if a household uses open fire or stove, 0 otherwise	
STOVEHEATOTHER	Binary	1 if a household uses another method, 0 otherwise.	
ACCESS	Binary	1 if the respondent (or their child) could get healthcare when needed, 0 otherwise.	The last time you [your child] needed health care, did you get health care? Note: Some respondents reported never having required healthcare. These cases are not considered to have been denied access.
Country-specific variables			
CONFLICTDEATH	Continuous by country	Average annual deaths from conflict occurring in any year 1990 – 2001 in the country in which the household resides.	Note: Calculated annual average UCDP "best estimate" for battle-related deaths for the period 1990 – 2001 inclusive.

Table 5.1 continued …

Country-specific variables cont …	***Variable type***	***Definition***	***Survey Question and Notes***
PUBLICHEALTH	Continuous by country	Natural logarithm of per capita government health expenditure in year 2001 in PPP Int. $.	Note: PPP series derived by WHO from 2005 International comparison project and estimated by World Bank.
ADULTMORT	Continuous by country	Probability of dying between 15 and 60 years per 1000 population in a given country for the year 2000.	Note: See WHOSIS http://apps.who.int/whosis/data/.
Household controls			
GENDER	Binary	1 if male, 0 otherwise.	Record sex as observed.
AGE	Continuous	Age of household respondent.	How old are you? (years)
AGE^2	Continuous	Square of the age of household respondent.	
EDUCATION	Quasi-continuous	Number of years of formal education completed.	How many years of school, including higher education, have you completed? (Excludes short courses or religious education such as Bible school or Koranic School. Note: In many cases individuals reported the highest level of education attained but not the number of years completed. In these cases the number of years completed is inferred by the typical number of years required in order to attain a given level of education in that particular country.
EXPENDITURE	Continuous	Total household expenditure in the four weeks preceding the survey.	In the last 4 weeks how much did your households spend in total? Notes: (1) Local currencies reported in WHS converted into 2005 $ using LCU per international $ for the year in which the survey was conducted for a given country. (2) Conversion data for Bosnia-Herzegovina become available from 2005. (3) Ghana transitioned from 2nd to 3rd cedi beginning Jan 2007 because of inflation. WHS local currency measured in 2nd cedi. Hence, World Bank conversion replaced with conversion for 2nd cedi. (4) Hungarian and Turkish households' total expenditures are not reported in WHS. Total expenditure in LCU calculated, ex post, as the sum of expenditure on housing, education, health, insurance and other goods and services. (5) Conversion data for UAE available only in 2005. (6) Slovenia joined the euro in 2007. The conversion rate was 239.640 tolars per euro. WHS households reported in tolars but World Bank conversion rate is in euros. The conversion rate is adjusted to reflect this. (7) Slovakia joined the euro on 1 Jan 2009. The conversion rate of Slovak koruna to euros (in 2002) was 43:1. WHS households report in korunas, but the World Bank conversion rate is in euros. Rates are adjusted as for Slovenia. (8) Estonia joined euro on 1 Jan 2011. The exchange rate of Estonian kroon (reported in WHS) to euros was set at 15.6466 krooni per euro in 2004. This figure is used in the conversion of krooni to PPP 2005 $. (9) Zimbabwe experienced significant depreciation against the dollar over the period of the survey. The LCU conversion factor is that of January 2002.

Table 5.1 continued …

Household controls cont …	***Variable type***	***Definition***	***Survey Question and Notes***
CHILD	Quasi-continuous	Number of children under 5.	This variable was constructed using the reported contemporaneous ages of children either in days, months or years. Children under 5 years (or equivalent) were counted to give a household total.
JOB	*Categorical*		*What is your current job?* *Note: (1) The reference category is defined as "not working for pay." (2) Mexico and Turkey contain responses not accounted for in the original questionnaire. These observations are assigned missing.*
JOBGOVT	Binary	1 if government employee, 0 otherwise.	
JOBPRIV	Binary	1 if non-government employee, 0 otherwise.	
JOBSELF	Binary	1 if self-employed, 0 otherwise.	
JOBEMPLOY	Binary	1 if employer, 0 otherwise.	
MARRIAGE	Binary	1 if currently married or cohabiting, 0 otherwise	What is your current marital status? Note: Categories in WHS include never married, currently married, separated, divorced, widowed and cohabiting. MARRIAGE=1 if currently married or cohabiting, 0 if otherwise.

Notes: See World Health Survey – Household Questionnaire, Long version, for high income countries, World Health Survey – Household Questionnaire, Long version, for Low Income Countries, World Health Survey – Individual Questionnaire, Long version, Rotation A/B/C/D (http://www.who.int/healthinfo/survey/en/).

Table 5.2 Sample characteristics for total sample, selected tobacco sample and tobacco regression sample

	Total Sample (1)	*Tobacco sample (2)*	*Tobacco regression sample (3)*
Independent Variables			
Household Environmental Risks			
FLOOR	0.26	0.26	0.30
WALL			
WALLMUD	0.15	0.15	0.17
WALLTHATCH	0.05	0.05	0.06
WALLPLAST	0.01	0.01	0.00
WALLMETAL	0.02	0.02	0.02
WALLOTHER	0.04	0.04	0.04
WATER			
WATERPUBLIC	0.13	0.13	0.15
WATERTUBE	0.13	0.13	0.17
WATERPRO	0.07	0.07	0.08
WATERUNPRO	0.08	0.08	0.09
WATERRAIN	0.01	0.01	0.01
WATERPOND	0.04	0.04	0.05
WATERTANKER	0.02	0.02	0.02
WATERTIME	-	-	7.31
WATER20	0.06	0.06	0.07
TOILET			
TOILETSEPTIC	0.14	0.14	0.14
TOILETPOUR	0.11	0.11	0.15
TOILETCOV	0.17	0.17	0.19
TOILETUNCOV	0.06	0.06	0.07
TOILETBUCKET	0.01	0.01	0.01
TOILETNONE	0.17	0.17	0.19
TOILETOTHER	0.01	0.01	0.01
TOILETLOCATE			
TOILETSHARE	0.08	0.08	0.08
TOILETOUT	0.07	0.07	0.08
TOILETOUTSHARE	0.25	0.25	0.23
COOKING			
COOKINGCOAL	0.09	0.09	0.08
COOKINGCROP	0.41	0.41	0.47
STOVECOOK			
STOVECHIMNEY	0.03	0.03	0.00
STOVECOOKOPEN	0.40	0.40	0.48
STOVECOOKOTHER	0.04	0.04	0.00
KITCHEN			
KITCHENLIVING	0.09	0.09	0.08
KITCHENSEPARATE	0.48	0.47	0.36

Table 5.2 continued …

	Total Sample (1)	*Tobacco sample (2)*	*Tobacco regression sample (3)*
Household Environmental Risks cont …			
KITCHENOUTDOOR	0.11	0.11	0.11
HEATHOUSE	0.30	0.31	0.31
HEATING			
HEATCOAL	0.19	0.19	0.19
HEATCROP	0.02	0.02	0.02
STOVEHEAT			
STOVEHEATCLOSED	0.04	0.04	0.03
STOVEHEATOPEN	0.16	0.16	0.16
STOVEHEATOTHER	0.02	0.02	0.02
ACCESS	0.03	0.03	0.04
Country-specific risks			
CONFLICTDEATH	-	-	606.93
PUBLICHEALTH	-	-	107.76
ADULTMORT	-	-	274.80
Household controls			
GENDER	0.56	0.58	0.60
AGE	40.77	40.83	39.85
AGE2			
EDUCATION	7.19	7.15	6.70
EXPENDITURE	1263.13	529.05	476.96
CHILD	0.18	0.21	0.26
JOB			
JOBGOVT	0.09	0.09	0.08
JOBPRIV	0.15	0.15	0.13
JOBSELF	0.34	0.33	0.33
JOBEMPLOY	0.01	0.01	0.01
MARRIAGE	0.67	0.67	0.68
Observations	263501	247126	124154

Notes: Means and proportions are reported for continuous and binary variables respectively. The total sample (1) and selected tobacco sample (2) includes data from all households which responded to at least one question in the questionnaire. In the selected tobacco sample the 6.7% of respondents which reported that they smoked, but not daily, are dropped and new means and proportions are reported. In (1) and (2) the number of observations reported are the number of households surveyed and the number of households surveyed minus the number of households that smoked, but not daily. Many household questionnaires were incomplete . Summary statistics are calculated using nonmissing data for each variable. Many households did not respond to the survey. In (3) and (4) summary statistics are reported for complete observations in the regression sample of households for TOBACCO. WATERTIME and country-specific risks are only reported for complete observations. WATERTIME is an imputed interval-censored variable which requires complete observations for auxiliary variables. Country-specific risks are reported only for complete observations. In the table the actual $ values rather than the log of

household expenditure and public health expenditure are reported. The regression analysis uses the natural logarithm of these values instead.

Table 5.3 Probit, Truncated Regression and Tobit models

	Dependent Variable: TOBACCO			
	Probit regression (1)	***Truncated regression (2)***	***Tobit regression (3)***	***Tobit / σ (4)***
Independent Variables				
Household Environmental Risks				
FLOOR	$-1.64x10^{-2}$	-0.163	-0.352	$-8.60x10^{-3}$
	$(2.01x10^{-2})$	(0.603)	(0.775)	
WALL				
WALLMUD	$1.16x10^{-2}$	0.649	0.735	$1.79x10^{-2}$
	$(2.16x10^{-2})$	(0.658)	(0.832)	
WALLTHATCH	$9.15x10^{-2}$***	1.27*	3.28***	$8.01x10^{-2}$
	$(2.28x10^{-2})$	0.618	(0.868)	
WALLPLAST	0.249	3.73	$1.00x10^{2}$*	$2.44x10^{-2}$
	0.132	(3.71)	(5.03)	
WALLMETAL	$1.58x10^{-2}$	-0.500	0.519	$1.27x10^{-2}$
	$(3.78x10^{-2})$	(0.962)	(1.41)	
WALLOTHER	0.114***	2.64***	5.00***	0.122
	$(2.67x10^{-2})$	(0.709)	(1.01)	
WATER				
WATERPUBLIC	$1.89x10^{-2}$	1.172	1.34	$3.27x10^{-2}$
	$(2.16x10^{-2})$	0.637	(0.839)	
WATERTUBE	$-1.17x10^{-3}$	-0.410	$-2.88x10^{-2}$	$-7.03x10^{-4}$
	$(2.12x10^{-2})$	(0.622)	(0.813)	
WATERPRO	$4.30x10^{-2}$	-0.428	1.42	$3.47x10^{-2}$
	$(2.60x10^{-2})$	(0.768)	(1.00)	
WATERUNPRO	$5.56x10^{-2}$*	0.650	2.44*	$5.96x10^{-2}$
	$(2.65x10^{-2})$	(0.768)	(1.02)	
WATERRAIN	$8.58x10^{-2}$	-1.45	3.61	$8.82x10^{-2}$
	$(5.56x10^{-2})$	(1.62)	(2.13)	
WATERPOND	0.138***	1.18	5.15***	0.126
	$(3.05x10^{-2})$	(0.850)	(1.16)	
WATERTANKER	$1.50x10^{-2}$	0.165	0.413	$1.01x10^{-2}$
	$(4.56x10^{-2})$	(1.40)	(1.77)	
WATERTIME	$6.43x10^{-4}$	$2.82x10^{-2}$	$3.50x10^{-2}$	$8.55x10^{-4}$
	$(5.05x10^{-4})$	$(1.50x10^{-2})$	$(1.94x10^{-2})$	
WATER20	$2.75x10^{-2}$	0.598	0.793	$1.94x10^{-2}$
	$(2.39x10^{-2})$	(0.674)	(0.912)	
TOILET				
TOILETSEPTIC	$-9.76x10^{-2}$***	-1.39*	-3.69***	$-9.07x10^{-2}$
	$(2.04.x10^{-2})$	(0.600)	(0.781)	
TOILETPOUR	$-5.92x10^{-2}$**	-0.129	-1.52	$-3.71x10^{-2}$
	$(2.43x10^{-2})$	(0.714)	(0.930)	
TOILETCOV	$-3.25x10^{-2}$*	0.673	-0.773	$-1.89x10^{-2}$
	$(2.39x10^{-2})$	(0.703)	(0.916)	

Table 5.3 continued …

Dependent Variable: TOBACCO				
Household Environmental Risks cont…	***Probit regression (1)***	***Truncated regression (2)***	***Tobit regression (3)***	***Tobit / σ (4)***
TOILETUNCOV	$7.39x10^{-2}$*	0.406	3.02*	$7.37x10^{-2}$
	($3.10x10^{-2}$)	(0.892)	(1.18)	
TOILETBUCKET	$3.84x10^{-2}$	0.885	1.83	$4.47x10^{-2}$
	($5.60x10^{-2}$)	(1.58)	(2.13)	
TOILETNONE	$3.69x10^{-2}$	1.85	2.10	$5.13x10^{-2}$
	($3.82x10^{-2}$)	(1.12)	(1.47)	
TOILETOTHER	$1.19x10^{-2}$	2.73*	1.69	$4.13x10^{-2}$
	($5.23x10^{-2}$)	(1.38)	(1.97)	
TOILETLOCATE				
TOILETSHARE	0.123*	0.716	1.75*	$4.27x10^{-2}$
	($3.01x10^{-2}$)	(0.648)	(0.871)	
TOILETOUT	$-1.86x10^{-2}$	-1.81**	-1.02	$-2.49x10^{-2}$
	($2.25x10^{-2}$)	(0.628)	(0.856)	
TOILETOUTSHARE	0.123***	-2.28**	3.54**	$8.64x10^{-2}$
	($3.01x10^{-2}$)	(0.867)	(1.15)	
COOKING				
COOKINGCOAL	$1.29x10^{-2}$	-2.14	0.814	$1.99x10^{-2}$
	($6.23x10^{-2}$)	(2.29)	(2.50)	
COOKINGCROP	$5.67x10^{-2}$	-0.660	2.77	$6.76x10^{-2}$
	($6.43x10^{-2}$)	(2.25)	(2.43)	
STOVECOOK				
STOVECHIMNEY	0.101	-0.865	2.73	$6.67x10^{-2}$
	0.135	(4.34)	(5.21)	
STOVECOOKOPEN	$8.06x10^{-2}$***	1.52*	3.21***	$7.84x10^{-2}$
	($2.50x10^{-2}$)	(0.731)	(0.958)	
STOVECOOKOTHER	$9.70x10^{-2}$	4.56	4.47	0.109
	0.128	4.28	(4.91)	
KITCHEN				
KITCHENLIVING	$7.86x10^{-2}$	-1.08	1.99	$4.86x10^{-2}$
	($6.46x10^{-2}$)	(2.32)	(2.52)	
KITCHENSEPARATE	$-6.44x10^{-2}$	-2.41	-3.36	$-8.21x10^{-2}$
	($6.19x10^{-2}$)	(2.25)	(2.41)	
KITCHENOUTDOOR	$-2.30x10^{-2}$	-2.32	-1.83	$-4.47x10^{-2}$
	($6.52x10^{-2}$)	(2.33)	(2.54)	
HEATHOUSE	$-5.70x10^{-2}$*	$9.17x10^{-2}$	-2.68**	$-6.54x10^{-2}$
	($2.68x10^{-2}$)	(0.770)	(1.02)	
HEATING				
HEATCOAL	$-7.97x10^{-2}$	-3.04	-3.28	$-8.01x10^{-2}$
	($8.13x10^{-2}$)	(2.51)	(3.12)	
HEATCROP	-0.182*	-5.04	-7.56*	-0.185
	($8.84x10^{-2}$)	(2.67)	(3.38)	

Table 5.3 continued …

Dependent Variable: TOBACCO				
Household Environmental Risks cont…	***Probit regression (1)***	***Truncated regression (2)***	***Tobit regression (3)***	***Tobit / σ (4)***
STOVEHEAT				
STOVEHEATCLOSED	$6.61x10^{-2}$	2.83	2.56	$6.25x10^{-2}$
	$(8.23x10^{-2})$	(2.63)	(3.31)	
STOVEHEATOPEN	0.109	2.43	4.75	0.116
	$(8.32x10^{-2})$	(2.56)	(3.19)	
STOVEHEATOTHER	0.141	3.58	5.64	0.138
	$(8.41x10^{-2})$	(2.58)	(3.22)	
ACCESS	$7.36x10^{-2}$**	-0.262	2.60*	$6.35x10^{-2}$
	$(2.84x10^{-2})$	(0.800)	(1.09)	
Country-specific risks				
CONFLICTDEATH	$-7.75x10^{-5}$	$-3.32x10^{-3}$	$-3.41x10^{-3}$	$-8.33x10^{-2}$
	$(7.75x10^{-5})$	$(3.24x10^{-3})$	$(3.07x10^{-3})$	
PUBLICHEALTH	$3.89x10^{-3}$***	$2.53x10^{-2}$***	0.129***	$3.15x10^{-3}$
	$(4.25x10^{-4})$	$(5.57x10^{-3})$	$(9.09x10^{-3})$	
ADULTMORT	$1.06x10^{-3}$***	$1.15x10^{-2}$	$4.63x10^{-2}$***	$1.13x10^{-3}$
	$(1.64x10^{-4})$	$(8.35x10^{-3})$	$(6.55x10^{-3})$	
Household controls				
GENDER	-1.05***	-8.14***	-42.0***	-1.03
	$(1.28x10^{-2})$	(0.413)	(0.54)	
AGE	$6.31x10^{-2}$***	1.64***	2.69***	$6.57x10^{-2}$
	$(2.04x10^{-3})$	$(6.26x10^{-2})$	$(7.85x10^{-2})$	
AGE2	$-6.42x10^{-4}$***	$-7.90x10^{-3}$***	$-2.50x10^{-2}$***	$-6.11x10^{-4}$
	$(2.17x10^{-5})$	$(6.31x10^{-4})$	$(8.26x10^{-5})$	
EDUCATION	$-3.48x10^{-2}$***	-0.208***	-1.28***	$-3.13x10^{-2}$
	$(1.51x10^{-3})$	$(4.49x10^{-2})$	$(5.81x10^{-2})$	
EXPENDITURE	$-5.05x10^{-3}$	0.783***	$4.46x10^{-2}$	$1.09x10^{-3}$
	$(5.37x10^{-3})$	(0.156)	(0.205)	
CHILD	$-8.32x10^{-2}$***	-0.603	-2.31***	$-5.64x10^{-2}$
	$(1.22x10^{-2})$	(0.505)	(0.485)	
JOB				
JOBGOVT	$8.06x10^{-2}$***	0.838	3.40***	$8.30x10^{-2}$
	$(2.30x10^{-2})$	(0.669)	(0.881)	
JOBPRIV	0.167***	0.288	6.26***	0.153
	$(1.80x10^{-2})$	(0.533)	(0.694)	
JOBSELF	1.52***	1.88***	5.66***	0.138
	$(1.46x10^{-2})$	(0.430)	(0.563)	
JOBEMPLOY	0.138***	2.87***	6.08***	0.148
	$(4.13x10^{-2})$	(1.07)	(1.56)	
MARRIAGE	$-9.92x10^{-2}$***	0.219	-4.16***	-0.102
	$(1.31x10^{-2})$	(0.383)	(0.502)	

Table 5.3 continued …

Dependent Variable: TOBACCO				
	Probit regression (1)	***Truncated regression (2)***	***Tobit regression (3)***	***Tobit / σ (4)***
Log-likelihood	-35066.47	-64672.45	-104634.13	-
σ	-	16.44	40.95	-
Wald p -value	-	0.000	-	-
λ	-	-	-	-
Observations	124154	16246	124154	-

Notes: Regression coefficients are reported to three significant digits. Country dummies are included in all models but are not reported. In the probit model country dummies for Comoros, Dominica and Spain were omitted due to collinearity. Standard errors in parentheses. * P<0.05, **P<0.01, ***P<0.001.

Table 5.4 Double Hurdle and Box Cox Double Hurdle models

	Dependent Variable: TOBACCO			
	Hurdle participation (5)	***Hurdle Intensity (6)***	***Box-Cox Hurdle Participation (7)***	***Box-Cox Hurdle intensity (8)***
Independent Variables				
Household Environmental Risks				
FLOOR	-2.56x10^{-2}	-0.314	-1.88x10^{-2}	-0.461
	(2.01x10^{-2})	(0.557)	(2.32x10^{-2})	(0.572)
WALL				
WALLMUD	7.82x10^{-2}	-1.38*	4.89x10^{-2}	-3.21***
	(2.16)	(0.682)	(2.53x10^{-2})	(0.719)
WALLTHATCH	9.55x10^{-2}***	-1.52*	0.134***	-1.98**
	(2.28x10^{-2})	(0.682)	(2.72x10^{-2})	(0.635)
WALLPLAST	0.241	1.63	0.246	1.92
	(0.132)	(4.03)	(0.148)	(4.14)
WALLMETAL	1.74x10^{-2}	-1.72	3.63x10^{-2}	-1.90
	(3.78x10^{-2})	(0.997)	(4.56x10^{-2})	(1.01)
WALLOTHER	0.116***	1.32	0.143***	6.81x10^{-2}
	(2.67x10^{-2})	(0.728)	(3.20x10^{-2})	(0.758)
WATER				
WATERPUBLIC	2.36x10^{-2}	0.529	2.59x10^{-2}	7.03x10^{-2}
	(2.15x10^{-2})	(0.675)	(2.46x10^{-2})	(0.705)
WATERTUBE	4.44x10^{-3}	-0.875	1.75x10^{-2}	-1.28
	(2.11x10^{-2})	0.629	(2.40x10^{-2})	(0.655)
WATERPRO	4.70x10^{-2}	-2.20**	7.22x10^{-2}*	-2.59**
	(2.60x10^{-2})	(0.816)	(2.97x10^{-2})	(0.844)
WATERUNPRO	6.34x10^{-2}*	0.446	5.40x10^{-2}	0.510
	(2.65x10^{-2})	(0.816)	(3.02x10^{-2})	(0.844)
WATERRAIN	9.44x10^{-2}	-2.44	0.122*	-3.06
	(5.59x10^{-2})	(1.71)	(6.17x10^{-2})	(1.79)
WATERPOND	0.142***	0.312	0.160***	0.186
	(3.05x10^{-2})	(0.875)	(3.56x10^{-2})	(0.896)
WATERTANKER	1.70x10^{-2}	-1.20	9.19x10^{-3}	-1.29
	(4.56x10^{-2})	(1.51)	(5.20x10^{-2})	(1.51)
WATERTIME	7.95x10^{-4}	-1.93x10^{-4})	1.03x10^{-3}	-9.54x10^{-3}
	(5.07x10^{-4})	(1.61x10^{-2})	(5.92x10^{-4})	(1.65x10^{-2})
WATER20	2.18x10^{-2}	2.87***	-2.50x10^{-2}	4.01***
	(2.39x10^{-2})	(0.725)	(2.76x10^{-2})	(0.763)
TOILET				
TOILETSEPTIC	-0.107***	-2.21***	-9.55x10^{-2}***	1.81
	(2.04x10^{-2})	(0.624)	(2.20x10^{-2})	(1.77)
TOILETPOUR	-6.77x10^{-2}**	-3.56***	-4.17x10^{-2}	-3.77***
	(2.34x10^{-2})	(0.685)	(2.69x10^{-2})	(0.742)
TOILETCOV	-3.27x10^{-2}	0.148	-4.87x10^{-2}	0.644
	(2.40x10^{-2})	(0.722)	(2.64x10^{-2})	(0.755)

Table 5.4 continued ...

Dependent Variable: TOBACCO				
Household Environmental Risks cont …	***Hurdle participation (5)***	***Hurdle Intensity (6)***	***Box-Cox Hurdle Participation (7)***	***Box-Cox Hurdle intensity (8)***
TOILETUNCOV	$7.05x10^{-2}$*	-1.91*	$8.59x10^{-2}$*	-1.59
	($3.10x10^{-2}$)	(0.887)	($3.54x10^{-2}$)	(0.923)
TOILETBUCKET	$3.15x10^{-2}$	1.11	$6.53x10^{-3}$	1.81
	($5.61x10^{-2}$)	(1.68)	($6.07x10^{-2}$)	(1.77)
TOILETNONE	$3.30x10^{-2}$	2.36*	$1.79x10^{-2}$	1.72
	($3.83x10^{-2}$)	(1.17)	($4.42x10^{-2}$)	(1.17)
TOILETOTHER	$9.41x10^{-3}$	2.14	$-2.40x10^{-2}$	2.58
	($5.24x10^{-2}$)	(1.47)	($5.95x10^{-2}$)	(1.54)
TOILETLOCATE				
TOILETSHARE	$4.78x10^{-2}$*	0.244	$4.17x10^{-2}$	0.374
	($2.28x10^{-2}$)	(0.695)	($2.71x10^{-2}$)	(0.704)
TOILETOUT	$-1.36x10^{-2}$	1.73**	$-4.58x10^{-2}$	2.70***
	($2.25x10^{-2}$)	(0.637)	($2.53x10^{-2}$)	(0.670)
TOILETOUTSHARE	0.126***	-2.26*	0.133***	-0.465
	($3.01x10^{-2}$)	(0.933)	($3.55x10^{-2}$)	(0.916)
COOKING				
COOKINGCOAL	$2.43x10^{-2}$	0.904	$3.81x10^{-2}$	-0.617
	($6.46x10^{-2}$)	(2.13)	($7.09x10^{-2}$)	(2.15)
COOKINGCROP	$6.40x10^{-2}$	1.66	$8.45x10^{-2}$	-0.270
	($6.25x10^{-2}$)	(2.07)	($6.84x10^{-2}$)	(2.09)
STOVECOOK				
STOVECHIMNEY	0.115	-0.590	$9.97x10^{-2}$	$8.78x10^{-2}$
	(0.135)	(4.57)	(0.143)	(4.49)
STOVECOOKOPEN	$7.83x10^{-2}$**	-0.296	$8.31x10^{-2}$**	0.346
	($2.49x10^{-2}$)	(2.73)	($2.84x10^{-2}$)	(0.787)
STOVECOOKOTHER	0.112	1.05	$7.76x10^{-2}$	4.72
	0.128	(2.77)	(0.140)	(4.44)
KITCHEN				
KITCHENLIVING	$5.85x10^{-2}$	-3.95	$7.66x10^{-2}$	-3.01
	($6.49x10^{-2}$)	(2.12	($7.12x10^{-2}$)	(2.13)
KITCHENSEPARATE	$-4.77x10^{-2}$	-6.42**	$-4.16x10^{-2}$	-6.16**
	($6.54x10^{-2}$)	(2.04)	($6.77x10^{-2}$)	(2.06)
KITCHENOUTDOOR	$-4.77x10^{-2}$	-7.84***	$1.50x10^{-2}$	-7.33***
	($6.54x10^{-2}$)	(2.14)	($7.21x10^{-2}$)	(2.17)
HEATHOUSE	$-6.94x10^{-2}$**	1.27*	$-9.35x10^{-2}$***	3.08***
	($2.68x10^{-2}$)	(0.611)	($2.88x10^{-2}$)	(0.673)
HEATING				
HEATCOAL	$-8.05x10^{-2}$	-0.796	$-7.38x10^{-2}$	-1.72
	(8.14)	(2.70)	($8.89x10^{-2}$)	(2.78)
HEATCROP	-0.182*	-3.24	-0.112	-5.63
	($8.84x10^{-2}$)	(2.86)	($9.74x10^{-2}$)	(2.95)

Table 5.4 continued …

Dependent Variable: TOBACCO				
Household Environmental Risks cont …	***Hurdle participation (5)***	***Hurdle Intensity (6)***	***Box-Cox Hurdle Participation (7)***	***Box-Cox Hurdle intensity (8)***
STOVEHEAT				
STOVEHEATCLOSED	$6.85x10^{-2}$	2.41	$4.87x10^{-2}$	3.50
	$(8.65x10^{-2})$	(2.81)	$(9.39x10^{-2})$	(2.91)
STOVEHEATOPEN	0.113	-0.296	0.123	-1.44
	$(8.32x10^{-2})$	(2.73)	$(9.10x10^{-2})$	(2.81)
STOVEHEATOTHER	0.145	1.05	0.114	2.44
	$(8.41x10^{-2})$	(2.77)	$(9.19x10^{-2})$	(2.85)
ACCESS	$7.85x10^{-2}$**	-0.465	$7.44x10^{-2}$*	0.107
	$(2.85x10^{-2})$	(0.869)	$(3.28x10^{-2})$	(0.88)
Country-specific risks				
CONFLICTDEATH	$-2.89x10^{-4}$***	$-4.94x10^{-4}$	$-5.62x10^{-4}$***	$-7.35x10^{-4}$*
	$(8.01x10^{-5})$	$(2.79x10^{-4})$	$(7.84x10^{-5})$	$(2.95x10^{-4})$
PUBLICHEALTH	$6.05x10^{-4}$***	$2.44x10^{-3}$**	$1.47x10^{-2}$***	$2.80x10^{-3}$**
	$(5.11x10^{-5})$	$(8.69x10^{-4})$	$(2.40x10^{-3})$	$(9.46x10^{-4})$
ADULTMORT	$2.77x10^{-5}$	$-4.08x10^{-2}$***	$-8.47x10^{-4}$***	$-4.79x10^{-2}$***
	$(2.04x10^{-4})$	$(2.39x10^{-3})$	$(1.58x10^{-4})$	$(3.44x10^{-3})$
Household controls				
GENDER	-1.05***	-	-1.13***	-
	$(1.28x10^{-2})$	-	$(1.43x10^{-2})$	-
AGE	$6.33x10^{-2}$***	1.83***	$2.34x10^{-2}$***	2.19***
	$(2.04x10^{-3})$	$(6.52x10^{-2})$	$(2.95x10^{-3})$	(0.126)
AGE2	$-6.43x10^{-4}$***	$-9.72x10^{-3}$***	$-3.21x10^{-4}$***	$-1.14x10^{-2}$***
	$(2.17x10^{-5})$	$(6.46x10^{-4})$	$(2.78x10^{-5})$	$(8.35x10^{-4})$
EDUCATION	$-3.54x10^{-2}$***	-	$-3.69x10^{-2}$***	-
	$(1.50x10^{-3})$	-	$(1.65x10^{-3})$	-
EXPENDITURE	-	0.111	-	$-6.12x10^{-2}$
	-	(0.157)	-	(0.159)
CHILD	$-8.29x10^{-2}$***	-	-0.125***	-
	$(1.22x10^{-2})$	-	$(1.37x10^{-2})$	-
JOB				
JOBGOVT	$7.96x10^{-2}$**	-	$7.09x10^{-2}$**	-
	$(2.30x10^{-2})$	-	$(2.44x10^{-2})$	-
JOBPRIV	0.167***	-	0.148***	-
	$(1.80x10^{-2})$	-	$(1.96x10^{-2})$	-
JOBSELF	0.151***	-	0.154***	-
	$(1.46x10^{-2})$	-	$(1.59x10^{-2})$	-
JOBEMPLOY	0.137***	-	0.139**	-
	$(4.13x10^{-2})$	-	$(4.47x10^{-2})$	-
MARRIAGE	-0.101***	-	-0.135***	-
	$(1.31x10^{-2})$		$(1.44x10^{-2})$	

Table 5.4 continued …

Dependent Variable: TOBACCO				
	Hurdle participation (5)	***Hurdle Intensity (6)***	***Box-Cox Hurdle Participation (7)***	***Box-Cox Hurdle intensity (8)***
Log-likelihood	-100523.43		-99024.87	
σ	17.68		19.78	
Wald p -value	0.000		0.000	
λ	-	-	1.07	
Observations	124154	124154	124154	124154

Notes: Country dummies are included in the participation regressor vector **(x_1)**. 26 observations of 124154 reported consumption of tobacco over 100 (mean: 171.85 tobacco products per day / std. dev: 74.13 / min: 108 / max: 352). The quantity of tobacco products consumed was censored at 100 for reported consumption over 100 so that maximum likelihood convergence could be attained in the Box Cox Double Hurdle Model. Standard errors in parentheses. * P<0.05, **P<0.01, ***P<0.001.

Table 5.5 Box Cox Double Hurdle estimates for restricted models

	Dependent Variable: TOBACCO									
	(9)	***(10)***	***(11)***	***(12)***	***(13)***	***(14)***	***(15)***	***(16)***	***(17)***	***(18)***
Independent Variables										
Household Environmental Risks										
FLOOR	$9.07x10^{-2}$***	-1.38**	-	-	-	-	-	-	-	-
	$(2.14x10^{-2})$	(0.453)	-	-	-	-	-	-	-	-
WALL							-	-	-	-
WALLMUD	$8.01x10^{-2}$***	-3.37***	-	-	-	-	-	-	-	-
	$(2.45x10^{-2})$	(0.631)	-	-	-	-	-	-	-	-
WALLTHATCH	0.225***	-4.43***	-	-	-	-	-	-	-	-
	$(2.62x10^{-2})$	(0.565)	-	-	-	-	-	-	-	-
WALLPLAST	0.263	1.13	-	-	-	-	-	-	-	-
	(0.145)	(3.60)	-	-	-	-	-	-	-	-
WALLMETAL	$7.76x10^{-2}$	-3.71	-	-	-	-	-	-	-	-
	$(4.45x10^{-2})$	(0.859)	-	-	-	-	-	-	-	-
WALLOTHER	0.195***	-0.380	-	-	-	-	-	-	-	-
	$(3.06x10^{-2})$	(0.640)	-	-	-	-	-	-	-	-
WATER										
WATERPUBLIC	-	-	$9.80x10^{-2}$***	-3.14***	-	-	-	-	-	-
	-	-	$(2.25x10^{-2})$	(0.566)	-	-	-	-	-	-
WATERTUBE	-	-	0.101***	-5.78***	-	-	-	-	-	-
	-	-	$(2.17x10^{-2})$	(0.533)	-	-	-	-	-	-

Table 5.5 continued …

Dependent Variable: TOBACCO										
Household Environmental Risks cont…	***(9)***	***(10)***	***(11)***	***(12)***	***(13)***	***(14)***	***(15)***	***(16)***	***(17)***	***(18)***
WATERPRO	-	-	0.147***	-6.49***	-	-	-	-	-	-
	-	-	($2.77x10^{-2}$)	(0.735)	-	-	-	-	-	-
WATERUNPRO	-	-	0.185***	-3.75***	-	-	-	-	-	-
	-	-	($2.74x10^{-2}$)	(0.659)	-	-	-	-	-	-
WATERRAIN	-	-	0.152**	-3.80**	-	-	-	-	-	-
	-	-	($5.84x10^{-2}$)	(1.48)	-	-	-	-	-	-
WATERPOND	-	-	0.283***	-3.94***	-	-	-	-	-	-
	-	-	($3.29x10^{-2}$)	(0.701)	-	-	-	-	-	-
WATERTANKER	-	-	$8.53x10^{-2}$	-4.36***	-	-	-	-	-	-
	-	-	($5.01x10^{-2}$)	(1.27)	-	-	-	-	-	-
WATERTIME	-	-	$1.73x10^{-3}$**	$-8.53x10^{-3}$	-	-	-	-	-	-
	-	-	($5.70x10^{-4}$)	($1.41x10^{-2}$)	-	-	-	-	-	-
WATER20	-	-	$-9.36x10^{-3}$	3.84***	-	-	-	-	-	-
	-	-	($2.68x10^{-2}$)	(0.666)	-	-	-	-	-	-
TOILET										
TOILETSEPTIC	-	-	-	-	$-8.05x10^{-2}$***	-4.35***	-	-	-	-
	-	-	-	-	($2.11x10^{-2}$)	(0.606)	-	-	-	-
TOILETPOUR	-	-	-	-	$7.31x10^{-3}$	-7.82***	-	-	-	-
	-	-	-	-	($2.54x10^{-2}$)	(0.692)	-	-	-	-
TOILETCOV	-	-	-	-	$3.39x10^{-2}$	-4.02***	-	-	-	-
	-	-	-	-	($2.32x10^{-2}$)	(0.596)	-	-	-	-
TOILETUNCOV	-	-	-	-	0.208***	-7.31***	-	-	-	-
	-	-	-	-	($3.21x10^{-2}$)	(0.792)	-	-	-	-

Table 5.5 continued …

	Dependent Variable: TOBACCO									
Household Environmental Risks cont…	***(9)***	***(10)***	***(11)***	***(12)***	***(13)***	***(14)***	***(15)***	***(16)***	***(17)***	***(18)***
TOILETBUCKET	-	-	-	-	$8.75x10^{-2}$	-2.48	-	-	-	-
	-	-	-	-	($5.83x10^{-2}$)	(1.50)	-	-	-	-
TOILETNONE	-	-	-	-	0.123**	-2.45**	-	-	-	-
	-	-	-	-	($4.11x10^{-2}$)	(0.946)	-	-	-	-
TOILETOTHER	-	-	-	-	0.102	-3.39**	-	-	-	-
	-	-	-	-	($5.71x10^{-2}$)	(1.29)	-	-	-	-
TOILETLOCATE										
TOILETSHARE	-	-	-	-	$7.67x10^{-2}$**	-1.31*	-	-	-	-
	-	-	-	-	($2.64x10^{-2}$)	(0.608)	-	-	-	-
TOILETOUT	-	-	-	-	$-1.40x10^{-2}$	1.60**	-	-	-	-
	-	-	-	-	($2.45x10^{-2}$)	(0.569)	-	-	-	-
TOILETOUTSHARE	-	-	-	-	0.178***	-2.02*	-	-	-	-
	-	-	-	-	($3.47x10^{-2}$)	(0.799)	-	-	-	-
COOKING										
COOKINGCOAL	-	-	-	-	-	-	$7.48x10^{-2}$	-1.35	-	-
	-	-	-	-	-	-	($6.96x10^{-2}$)	(1.90)	-	-
COOKINGCROP	-	-	-	-	-	-	0.167*	-0.541	-	-
	-	-	-	-	-	-	($6.67x10^{-2}$)	(1.84)	-	-
STOVECOOK										
STOVECHIMNEY	-	-	-	-	-	-	0.103	2.17	-	-
	-	-	-	-	-	-	(0.142)	(3.90)	-	-

Table 5.5 continued …

Dependent Variable: TOBACCO										
Household Environmental Risks cont…	***(9)***	***(10)***	***(11)***	***(12)***	***(13)***	***(14)***	***(15)***	***(16)***	***(17)***	***(18)***
STOVECOOKOPEN	-	-	-	-	-	-	0.118***	-1.14	-	-
	-	-	-	-	-	-	($2.75x10^{-2}$)	(0.679)	-	-
STOVECOOKOTHER	-	-	-	-	-	-	0.104	5.02	-	-
	-	-	-	-	-	-	(0.137)	(3.85)	-	-
KITCHEN										
KITCHENLIVING	-	-	-	-	-	-	0.106	-1.73	-	-
	-	-	-	-	-	-	($6.96x10^{-2}$)	(1.86)	-	-
KITCHENSEPARATE	-	-	-	-	-	-	$-4.72x10^{-2}$	-5.78**	-	-
	-	-	-	-	-	-	($6.65x10^{-2}$)	(1.83)	-	-
KITCHENOUTDOOR	-	-	-	-	-	-	4.08	-7.13***	-	-
	-	-	-	-	-	-	(7.07)	(1.92)	-	-
HEATHOUSE	-	-	-	-	-	-	-	-	-0.115***	6.73***
	-	-	-	-	-	-	-	-	($2.84x10^{-2}$)	(0.664)
HEATING				-						
HEATCOAL	-	-	-	-	-	-	-	-	$-6.48x10^{-2}$	-2.57
	-	-	-	-	-	-	-	-	($8.68x10^{-2}$)	(2.47)
HEATCROP	-	-	-	-	-	-	-	-	$-6.53x10^{-2}$	-6.22*
	-	-	-	-	-	-	-	-	($9.51x10^{-2}$)	(2.63)
STOVEHEAT										
STOVEHEATCLOSED	-	-	-	-	-	-	-	-	$4.33x10^{-2}$	3.05
	-	-	-	-	-	-	-	-	($9.14x10^{-2}$)	(2.58)

Table 5.5 continued …

Dependent Variable: TOBACCO										
Household Environmental Risks cont…	***(9)***	***(10)***	***(11)***	***(12)***	***(13)***	***(14)***	***(15)***	***(16)***	***(17)***	***(18)***
STOVEHEATOPEN	-	-	-	-	-	-	-	-	0.156	-2.51
	-	-	-	-	-	-	-	-	$(8.88x10^{-2})$	(2.50)
STOVEHEATOTHER	-	-	-	-	-	-	-	-	0.101	1.66
	-	-	-	-	-	-	-	-	$(8.96x10^{-2})$	(2.53)
ACCESS	$9.03x10^{-2}$**	-0.536	$9.43x10^{-2}$**	-0.651	$8.74x10^{-2}$**	-0.286	$8.72x10^{-2}$**	-0.323	0.102**	-0.687
	$(3.22x10^{-2})$	(0.767)	$(3.20x10^{-2})$	(0.755)	$(3.21x10^{-2})$	(0.769)	$(3.22x10^{-2})$	(0.776)	$(3.24x10^{-2})$	(0.815)
Country-specific risks										
CONFLICTDEATH	$-5.45x10^{-4}$***	$-3.38x10^{-4}$	$-5.58x10^{-4}$***	$-2.12x10^{-4}$	$-5.45x10^{-4}$***	$5.58x10^{-5}$	$-5.70x10^{-4}$***	$-5.55x10^{-4}$*	$-5.54x10^{-4}$***	$-8.19x10^{-4}$**
	$(7.66x10^{-5})$	$(2.44x10^{-4})$	$(7.63x10^{-5})$	$(2.36x10^{-4})$	$(7.71x10^{-5})$	$(2.40x10^{-4})$	$(7.76x10^{-5})$	$(2.47x10^{-4})$	$(7.72x10^{-5})$	$(2.69x10^{-4})$
PUBLICHEALTH	$1.50x10^{-2}$***	7.44***	$1.41x10^{-2}$***	$5.68x10^{-3}$***	$1.46x10^{-2}$***	$4.83x10^{-3}$**	$1.57x10^{-2}$***	$4.57x10^{-3}$***	$1.40x10^{-2}$***	$5.60x10^{-3}$***
	$(2.32x10^{-3})$	$(8.30x10^{-4})$	$(2.29x10^{-3})$	$(7.88x10^{-4})$	$(2.32x10^{-3})$	$(7.94x10^{-4})$	$(2.32x10^{-3})$	$(7.83x10^{-4})$	$(2.30x10^{-3})$	$(8.81x10^{-4})$
ADULTMORT	$-7.45x10^{-4}$***	$-3.88x10^{-2}$***	$-7.56x10^{-4}$***	$-3.97x10^{-2}$***	$-7.92x10^{-4}$***	$-4.40x10^{-2}$***	$-8.75x10^{-4}$***	$-3.72x10^{-2}$***	$-5.48x10^{-4}$***	$-4.88x10^{-2}$***
	$(1.47x10^{-4})$	$(2.99x10^{-3})$	$(1.48x10^{-4})$	$(3.01x10^{-3})$	$(1.50x10^{-4})$	$(3.25x10^{-3})$	$(1.47x10^{-4})$	$(2.85x10^{-3})$	$(1.51x10^{-4})$	$(3.46x10^{-3})$
Household controls										
GENDER	-1.12***	-	-1.11***	-	-1.12***	-	-1.12***	-	-1.12***	-
	$(1.43x10^{-2})$	-	$(1.472x10^{-2})$	-	$(1.42x10^{-2})$	-	$(1.47x10^{-2})$	-	$(1.43x10^{-2})$	-
AGE	$2.80x10^{-2}$***	1.88***	$2.85x10^{-2}$***	1.86***	$2.88x10^{-2}$***	1.87***	$2.70x10^{-2}$***	1.93***	$2.52x10^{-2}$***	1.99***
	$(3.08x10^{-3})$	(0.127)	$(3.03x10^{-3})$	(0.123)	$(3.03x10^{-3})$	(0.123)	$(2.98x10^{-3})$	(0.121)	$(3.10x10^{-3})$	(0.130)
AGE2	$-3.60x10^{-4}$***	$-1.01x10^{-2}$***	$-3.63x10^{-4}$***	$-1.00x10^{-2}$***	$-3.64x10^{-4}$***	$-9.94x10^{-3}$***	$-3.51x10^{-4}$***	$-1.03x10^{-2}$***	$-3.39x10^{-4}$***	$-1.07x10^{-2}$***
	$(2.86x10^{-5})$	$(8.27x10^{-4})$	$(2.82x10^{-5})$	$(8.06x10^{-4})$	$(2.83x10^{-5})$	$(8.05x10^{-4})$	$(2.79x10^{-5})$	$(7.94x10^{-4})$	$(2.87x10^{-5})$	$(8.49x10^{-4})$

Table 5.5 continued …

Dependent Variable: TOBACCO										
Household controls cont …	***(9)***	***(10)***	***(11)***	***(12)***	***(13)***	***(14)***	***(15)***	***(16)***	***(17)***	***(18)***
EDUCATION	$-4.31x10^{-2}$***	-	$-4.33x10^{-2}$***	-	$-3.97x10^{-2}$***	-	$-4.11x10^{-2}$***	-	$-4.64x10^{-2}$***	-
	($1.55x10^{-3}$)	-	($1.54x10^{-3}$)	-	($1.58x10^{-3}$)	-	($1.58x10^{-3}$)	-	($1.53x10^{-3}$)	-
EXPENDITURE	-	$3.05x10^{-2}$	-	$-6.84x10^{-2}$	-	$5.02x10^{-2}$	-	-0.239	-	-1.35
	-	(0.135)	-	(0.135)	-	(0.138)	-	(0.140)	-	(0.143)
CHILD	-0.105***	-	-0.102***		-0.112***	-	-0.109***	-	-0.102***	-
	($1.34x10^{-2}$)	-	($1.33x10^{-2}$)		($1.34x10^{-2}$)	-	($1.34x10^{-2}$)	-	($1.34x10^{-2}$)	-
JOB										
JOBGOVT	$7.36x10^{-2}$**	-	$8.08x10^{-2}$***	-	$7.66x10^{-2}$**	-	$7.53x10^{-2}$**	-	$7.41x10^{-2}$**	-
	($2.42x10^{-2}$)	-	($2.41x10^{-2}$)	-	($2.42x10^{-2}$)	-	($2.42x10^{-2}$)	-	($2.43x10^{-2}$)	-
JOBPRIV	0.155***	-	0.160***	-	0.153***	-	0.154***	-	0.153***	-
	($1.93x10^{-2}$)	-	($1.93x10^{-2}$)	-	($1.94x10^{-2}$)	-	($1.93x10^{-2}$)	-	($1.95x10^{-2}$)	-
JOBSELF	0.170***	-	0.173***	-	0.162***	-	0.164***	-	0.178***	-
	($1.56x10^{-2}$)	-	($1.56x10^{-2}$)	-	($1.57x10^{-2}$)	-	($1.57x10^{-2}$)	-	($1.57x10^{-2}$)	-
JOBEMPLOY	0.153***	-	0.156***	-	0.145***	-	0.152***	-	0.157***	-
	($4.41x10^{-2}$)	-	($4.69x10^{-2}$)	-	($4.42x10^{-2}$)	-	($4.41x10^{-2}$)	-	($4.43x10^{-2}$)	-
MARRIAGE	-0.135***	-	-0.138***	-	-0.135***	-	-0.135***	-	-0.139***	-
	($1.42x10^{-2}$)	-	($1.42x10^{-2}$)	-	($1.42x10^{-2}$)	-	(1.42)	-	($1.43x10^{-2}$)	-
Log-likelihood	-99870.19		-99825.21		-99717.36		-99702.01		-99903.36	
σ	17.23		16.99		17.29		17.53		18.36	
Wald p -value	0.0000		0.0000		0.0000		0.0000		0.0000	
λ	1.02		1.02		1.02		1.03		1.04	
Observations	124154	124154	124154	124154	124154	124154	124154	124154	124154	124154

Notes: All regression coefficients, standard errors and lambda estimated by maximum likelihood. Standard errors in parentheses. * P<0.05, ** P<0.01, *** P<0.001.

Figure 5.1 **Frequency Distribution of TOBACCO**

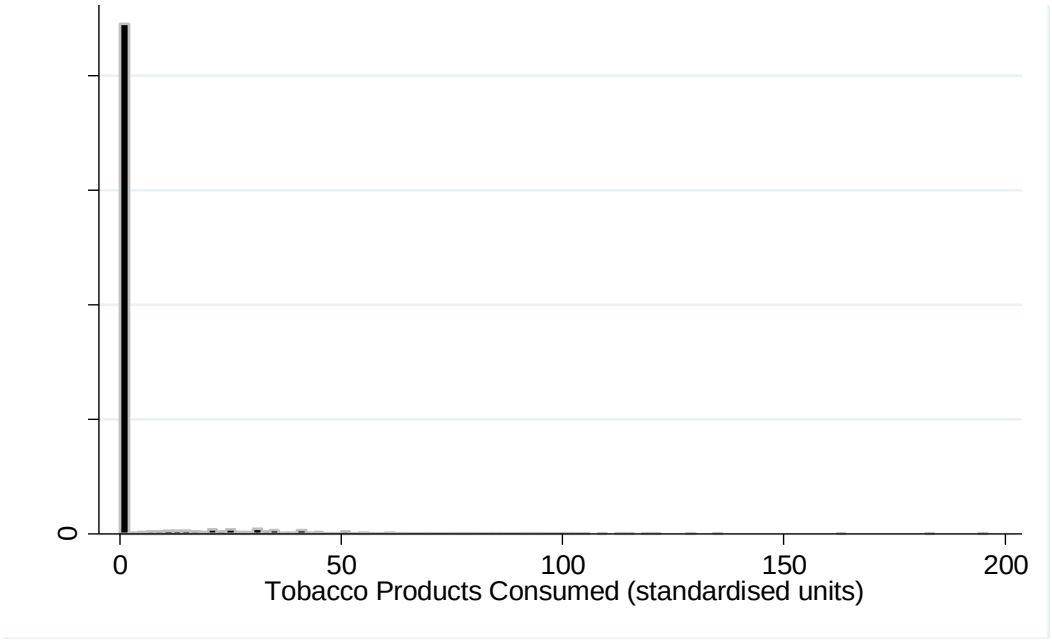
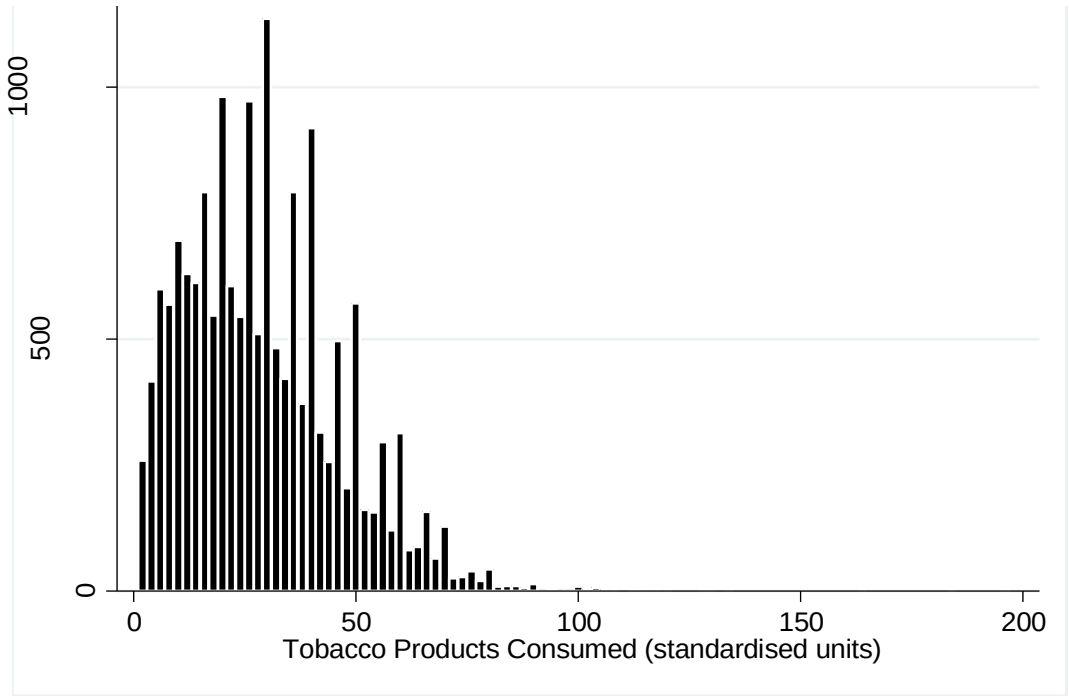


Figure 5.2 **Frequency Distribution of TOBACCO > 0**



5.9 Appendix B

Table 5.6 Countries Surveyed in the WHS in partnership with WHO, 2002 – 2004

WORLD HEALTH SURVEY 2002						
		Households surveyed *				
Country	**Abbreviation**	**Urban**	**Peri-Urban**	**Rural**	**Total ****	**Environmental data collected *****
Australia	AUS	N/A	N/A	N/A	N/A	N
Austria	AUT	781	0	274	1055	N
Bangladesh	BAN	2042	0	3900	5942	N
Belgium	BEL	824	0	184	1012	Y
Bosnia-Herzegovina	BOS	436	0	595	1031	N
Brazil	BRA	4000	0	840	5000	Y
Burkina Faso	BUR	2013	0	2932	4948	Y
Chad	CHA	1201	0	3665	4875	Y
China	CHI	1587	0	2407	3994	Y
Comoros	COM	548	0	1287	1836	Y
Congo, (Rep. of)	CON	2439	0	631	3077	Y
Cote d'Ivoire	COT	1975	0	1270	3251	Y
Croatia	CRO	659	0	334	993	Y
Czech Republic	CZE	678	0	271	949	Y
Denmark	DEN	605	0	398	1003	Y
Dominican Rep.	DOM	2774	0	2253	5027	N
Ecuador	ECU	3784	0	1853	5677	Y
Estonia	EST	670	0	351	1021	Y
Ethiopia	ETH	805	0	4285	5090	Y
Finland	FIN	623	0	390	1013	Y
France	FRA	554	0	454	1008	N
Georgia	GEO	1298	0	1589	2947	N
Germany	GER	1086	0	173	1259	Y
Ghana	GHA	1626	0	2533	4165	N
Greece	GRC	720	0	280	1000	Y
Guatemala	GUA	1719	300	2767	4890	N
Hungary	HUN	869	0	550	1419	Y
India	IND	2980	0	7703	10692	Y
Ireland	IRE	601	0	413	1014	Y
Israel	ISR	1399	0	129	1536	N
Italy	ITA	685	0	315	1000	N
Kazakhstan	KAZ	2685	0	1814	4499	N
Kenya	KEN	1482	0	3157	4640	Y
Lao PDR	LAO	1278	0	3711	4989	Y
Latvia	LAT	634	0	295	929	Y
Luxembourg	LUX	700	0	0	700	Y
Malawi	MLW	873	0	4672	5551	N
Malaysia	MLY	3621	0	2524	6145	Y
Mali	MLI	1306	0	3887	5209	Y
Mauritania	MTA	1655	0	2189	3907	Y

Table 5.6 continued …

WORLD HEALTH SURVEY 2002

Country	Abbreviation	Households surveyed * Urban	Peri-Urban	Rural	Total **	Environmental data collected ***
Mauritius	MTS	1766	0	2201	3968	Y
Mexico	MEX	29531	0	9215	37846	Y
Morocco	MOR	2638	0	2078	5000	Y
Myanmar	MYA	1482	0	4563	6045	Y
Namibia	NAM	2078	0	2299	4379	Y
Nepal	MEP	1279	0	7543	8822	Y
Netherlands	NLD	N/A	N/A	N/A	1091	Y
Norway	NOR	N/A	N/A	N/A	984	N
Pakistan	PAK	2814	0	3688	6502	N
Paraguay	PRY	2481	0	2807	5288	Y
Philippines	PHL	5928	0	4155	10083	Y
Portugal	PRT	573	0	457	1030	Y
Russian Federation	RUS	4067	0	359	4427	N
Senegal	SEN	1657	0	1394	3465	Y
Slovakia	SLK	2332	0	200	2535	Y
Slovenia	SLN	N/A	N/A	N/A	687	Y
South Africa	RSA	1571	0	1030	2629	Y
Spain	SPA	4571	0	1802	6373	Y
Sri Lanka	SRI	1039	0	5692	6805	Y
Swaziland	SWA	776	0	2294	3121	Y
Sweden	SWE	550	0	450	1000	Y
Tunisia	TUN	3247	0	1952	5203	N
Turkey	TUR	5783	0	5696	11481	Y
UAE	UAE	908	0	275	1183	Y
United Kingdom	GBR	1062	0	82	1200	Y
Ukraine	UKR	1998	0	593	2860	N
Uruguay	URU	2491	0	495	2996	Y
Vietnam	VIE	1014	0	3159	4174	Y
Zambia	ZAM	1692	0	2473	4166	Y
Zimbabwe	ZIM	1528	0	2736	4292	Y

Notes: * Households are assigned a unique Identification (or ID). In some questionnaires multiple individuals from a single household are sampled. Data used here is derived only from questionnaires of the primary respondent. ** Totals may differ from the sum of urban, peri-urban and rural because of missing observations recording the setting of the household. *** Y=Yes, N= No.

Conclusion to thesis

Our investigation of the first episode in the control of tuberculosis in chapter 1 revealed how local authorities in Britain, emerging from the brutality of the First World War, sought to organise and deliver better healthcare services to those for whom there was need. During the years following the First World War the role of the State in the administration and direction of healthcare and health related resources increased. The scope of the 1929 health audit was broad and reviewed local healthcare practices as eclectic as the control of infectious disease (including tuberculosis, venereal disease, whooping cough), the provision of mental health services, the quality of urban sanitation services, and the provision orthopaedic, maternal and child services. However, the drive towards standardisation in reporting procedures quickly revealed differences between counties and towns. Counties with parochial approaches were reproached by the Ministry of Health with the threat of takeover. There were, however, many towns and counties with outstanding performance, and although the drive for centralisation and standardisation was a temptation for those in authority, the interwar healthcare system should be recognised for its ability to deliver improvements in local tuberculosis outcomes by experiment and through an acceptance and appreciation of local circumstances. That the system presided over, and influenced the success of, substantial improvements tuberculosis control is a testament to the viability of decentralised economic policy in healthcare. Chapter 1 contributed to the literature by revealing that it is too restrictive to assess performance of county towns and boroughs in Britain in terms of local expenditure alone. Naturally a more nuanced picture of tuberculosis healthcare services emerged once the returns of the Ministry of Health; Local Government Act 1929 were fully explored

Auditors collecting information on local health services in the 1920s have produced a trove of research material for the economic historian. Their contribution was not simply a matter of collecting the quotidian material relating to service timetables, pay scales and reviewing local authority compliance with data collection requirements, necessary for an effective and emerging public health service. They

also had the sagacity to review and explore general environmental conditions, the historical legacy of each location and the quality of relationships between different agencies of healthcare provision and how they related to outcomes and institutional formation. It is surprising the extent to which, in the first general audit of its kind, the Ministry of Health was prepared to take a holistic approach. But the audit was not without limitation. The investigation in chapter 1 relied on the reliability of reporting procedures and the nature of that which was reported. We arrived at the conclusion, for example, that the quality of local health services was a function of the quality of the relationships between various agencies of the State and also of the private and voluntary sectors. Often the impressions of the Minister of Health of these relationships were simply that; impressions. The real quality of relationships, their heterogeneity, and their consequences for tuberculosis control, remain necessarily opaque. Reports concerning the competence of local doctors, for example, to carry out their operations and their attitudes towards healthcare innovation are at best proxies for tuberculosis outcomes. Nevertheless, chapter 1 contributed to the literature by presenting evidence that suggests a more nuanced view than that presented by either proponents of the 'official view' or of the 'negative view' of healthcare in pre-Welfare Britain. In terms of the former we found, in general, that local authorities were successful, given the medical limitations of the period, in driving down tuberculosis mortality rates. Poorer areas were able to succeed as wealthier areas so long as institutions were effective and adapted to local circumstances. In terms of the latter, chapter 1 offered powerful evidence to suggest that where one lived affected health outcomes. In some locations, especially those tied to traditional industry, we found marked differences in performance between the most and least improved. It was not a simple matter of expenditure either; rather tuberculosis outcomes depended on the role of individuals and their families, socio-economic conditions that determine health, the coordination of nascent public health initiatives and the differences in the experience of rural and urban local authorities. It was also discovered that no one particular method was necessarily successful. Thus a picture emerges from the discussion in chapter 1, one in which local authorities experimented with different (or indifferent) policies that affected outcomes within the context of a generally declining burden from tuberculosis disease.

Econometric work in chapter 1 discovered that there were clear standard of living

predictors of tuberculosis outcomes in a cross section of English counties and towns. Larger families, higher urban population density and higher living density were associated with lower incidence of tuberculosis and fewer deaths. We also discovered that living in a rural environment was independently protective. Medical and administrative measures relied on accurate and reliable reporting of local authorities' health statistics. Much effort was expended by the Ministry of Health to ensure that the data submitted for collation was standardised and met the requirements of the collection methodology. Regardless of local adherence, authorities were subject to the bureaucratic and scientific limitations of the era. Our findings rely on accurate reporting at the local level as well as accurate diagnosis of tuberculosis in the population as a whole via available technologies, such as sputum tests and new, but poor quality, X-ray procedures. Identification of the disease is an issue to which we returned in chapters 2 and 3. Further research will offer criticism no doubt of methods presented in chapter 1. In particular the extent to which the local sanatoria were able to control transmission requires further investigation. One possibility not explored here is that, where isolation of the infected was more successful, transmission within local authorities was curtailed, generating positive externalities for bordering towns and counties. Sanatoria were available in most counties and towns, but there existed important differences between them. Some were poorly maintained, others did not offer effective isolation despite the name sanatoria and still others provided adequate isolation from contacts, but staff were so poorly equipped that they were unable to perform many important functions within the facility. Thus, the role of sanatoria reducing tuberculosis case rates on a cross-county and cross-town basis requires far more than a cursory investigation. Another limitation of the chapter 1 is that it does not account for the cross-cooperation between counties and towns alluded to in the 1929 audit and elsewhere. Often local authorities would cooperate in identification of infection and isolation of the infected. Future statistical analyses may deliver more robust results by accommodating for this reality, provided suitable data can be found.

Fortunately for the people of Britain and elsewhere in the West, tuberculosis ceased to be of such dire consequence and focus on the disease ebbed substantially in these countries as new 'diseases of civilisation' came to the forefront of public health concern in the post Second World War era. During this time, however, the

consequences of miracle drugs capable of delivering humankind from tuberculosis disease was still to be felt on a truly global scale. Chapter 2 contributed to the literature in three important ways. Firstly, evidence was collated over a wider range of countries than previously explored which allowed us to identify a divided world, one in which some individuals were free from the threat of debilitating tuberculosis disease and many were not. Secondly, chapter 2 revealed how the process of development itself can create the conditions in which mycobacteria can thrive. Thirdly, we investigated why, in an era where medical science was able to identify, isolate and cure the infected, why so many in the developing world continued to suffer with and die from the disease.

Chapter 2 discovered that in many parts of the world, the medical model for the arrest of tuberculosis disease in the developed world was not applicable in the developing world. As with English counties and towns, countries experienced differing success when tackling the tuberculosis problem over the period 1950 to 1980. The important finding of chapter 2 was to identify the problems encountered by developing economies in implementing effective vaccination and treatment programs, in accurately identifying the infected, in accurately recording metrics appertaining to tuberculosis control, and in the dissemination of treatment to dispersed and impoverished communities that were afflicted not only by tuberculosis but also numerous other equally pressing health issues. The other important finding that development itself constitutes an important risk factors for the development of tuberculosis provoked the search for statistical corroboration for the arguments made in the historical literature, that urbanisation, overcrowding and poor diet contributed to the burden of tuberculosis. More recent data from the WHO provided an opportunity to complement the findings in chapter 1 that overcrowding was indeed a significant risk factor, but also to expand upon the analysis through the introduction of nutrition related variables otherwise unavailable in the interwar era. Hence, the focus of chapter 3; to use data to corroborate findings from both the interwar era but also to corroborate or not the arguments made in the largely qualitative literature concerned with assessing the circumstances in which tuberculosis disease thrived. In doing so chapter 3 contributed to the literature by using statistical evidence to test hypotheses proposed in the qualitative literature.

WHO and other international institutions recognised the importance of measuring health metrics on a global scale. They embarked on the task of coordinating the collection of health data from not only European countries, with existing reporting methodologies and the resources to do so, but also aspired to collect data from all the countries that they represented. Reporting practices of newly independent developing countries were still in their infancy and coaxing reliable data from them was difficult, especially in those early years. Thus, data from WHO used in chapter 3 is highly dependent on the quality of the data collection practices of individual countries. Although the data are rich, their reliability is subject to the ability of local diagnostic facilities and the capacity of national health administrations to submit to standardised reporting. We believe, however, that the WHO offers the best cross-country data available for the period, the caveat remaining that it is likely subject to significant measurement error for which the econometric analysis only partially accommodates.

With these limitations in mind, it is reassuring that the findings of chapter 3 with regards to overcrowding are corroborated by chapter 1. We found that the hypothesis raised in chapter 2 that development itself constitutes a tuberculosis risk was supported when a tuberculosis Kuznet's curve was observed independently of factors traditionally used to explain variation in country level tuberculosis rates in a dynamic panel model. Urbanisation was also modelled as using a second order polynomial and similar to income, it was found to have an inverted-U shaped relationship with tuberculosis mortality, with both variables exhibiting independent effects when both included in the model. Despite our efforts, collection of a comprehensive variable that captured the level of industrialisation for the sample of countries in the period of interest was not possible. Were better data available it is expected that industrialisation would have a significant and negative relationship with tuberculosis morbidity and mortality.

With regards to nutrition, the findings are corroborated by arguments in the 'war literature' that nutritional deficiency was linked to tuberculosis mortality increases in Belgium, France and the Netherlands and also, but to a lesser extent, in Italy, Spain and Hungary (Biraud, 1943/4, p. 687). Daniel noted that 'in thickly populated areas of the South of France where the food situation was bad there was a continuous rise

in TB … while in Brittany where there had always been a very high TB death rate but where during the war the food situation was relatively good the death rates fell steadily throughout the war' (Daniels, 1946, p. 204). The contribution of chapter 3, that it is total calories that matter for reducing the rate of tuberculosis mortality is corroborated by recent meta-analyses of the role of BMI in conferring increased cell-mediated immunity. Thus in a period in which death from tuberculosis was in theory, avoidable, practical issues remained stubborn obstacles to treatment but as in the first episode, socio-economic conditions could either be protective or a danger. Chapter 3 discovered that greater calories derived from meat, fish , milk or eggs was not associated with fewer cases or deaths across a sample of developed, newly industrialising and developing countries; in fact the results indicated the reverse. Nor were the number of calories derived from any other food source important in explaining patterns in mortality and morbidity, except fruits, vegetables, pulses and nuts which were significantly negatively associated with tuberculosis mortality. The literature has argued that populations that lack of animal protein in their diet suffer from a tuberculosis problem (Johnston, 2003). This view may be applicable, especially in crises such as war, but the evidence suggests that under normal circumstances, the quantity of calories derived from animal protein is not a determinant of tuberculosis outcomes.

Further research will exploit local quantitative data as well as qualitative sources to investigate the true epidemiological conditions and state of treatment that prevailed in developing countries during the period immediately following Second World War. A picture emerged in chapter 3 that resembled the qualitative picture of tuberculosis outcomes developed in sections 5 and 6 of chapter 1.When ‘good’ policy countries were compared to ‘bad’ policy countries it was found that good policy countries had lower turning points in their observed tuberculosis Kuznets curve relation for both morbidity and mortality. They more quickly reversed the rise in tuberculosis disease that seems to accompany the early stages of development. Histories are predominantly Western-centric (Daniel, 2006; Herzog, 1998; McCarthy, 2001) and do not attempt to evaluate the mechanisms behind country-specific successes and failures. Studies that do examine the determinants of cross-country incidence do exist (Dye, 2009) but the focus is recent and does not exploit the historical significance of the period that bounds the second episode.

The findings in chapter 3 and those of the corroborative literature motivated an investigation into the recent nutrition science literature. Chapter 4 revealed, however, that the only robust nutritional predictor of tuberculosis incidence so far discovered in high quality studies was BMI. More strikingly, as BMI increased even into the obese range, the relative risk of developing symptoms continued to decline. Promoting obesity to control tuberculosis in locales where antimicrobial drugs have failed is not a defensible public health strategy. A new avenue of research is looking into the mechanisms behind the observed and robustly established relationship between lower tuberculosis incidence and higher BMI. Abundant adipose tissue improves the ability of the thymus to facilitate the maturation of T-cells; cells necessary for the containment of *mycobacteria tuberculosis*. Given that the results were found in mice the science is still at an early stage of investigation and there is no guarantee that similar phenomena will be observed in humans or that any low cost measure that mimics the mechanism can be used as an intervention. In the third episode of tuberculosis control the innovation must come from prophylactic measures if traditional lines of innovation, such as new antimicrobial remedies, do not yield positive results. Chapter 4 reviewed existing work that has sought non-nutritive socio-economic interventions to attenuate tuberculosis pathogenesis but found that in some instances these measures did not address the fundamental issue, raised in chapter 2, that in many cases people suffering from tuberculosis cannot afford to wait for development to occur. Many of these suggestions, in any case, do not differ in helpful ways from the natural consequences of long run economic growth and development.

To prevent passage from the second to third episodes individuals, healthcare professionals and governments will need to look for new methods to control tuberculosis based on old wisdom. Potentially productive areas for further research into the resistance conferred by nutritive measures are available. The role of nutrient combinations in whole foods is a particularly under-investigated area in large scale cohort studies. Given the purported importance of vitamin D to immune function, alluded to in chapter 3, the influence that the micro-nutrient has on preventing active tuberculosis has received only cursory treatment in the laboratory and in the field. Despite this, there has been significant investigation into the effectiveness of vitamin

D as a supplementary treatment in recovering adult patients (Martineau, 2011; Nursyam, 2006; Wejse, 2009) and children (Morcos, 1998).

A recurring theme in this thesis has been the central role environmental risks play in determining health outcomes. In chapter 5, the focus moved away from special consideration of tuberculosis whilst the focus on environmental determinants of health remained. We wished to concentrate on a case study where the risks taken were within the control of the individual. We asked, do background environmental risks to health not only determine health outcomes, but also affect decision making regarding health? Empirical studies have asked the analogous question, do background risks to wealth affect decision making regarding the allocation of wealth? Both health and wealth are subject to risks, but the former endowment is frequently overlooked in analysis favour of the latter. It was noted that the theoretical contributions of Gollier and Pratt (1996), Kimball (1993) and Pratt and Zeckhauser (1987) had not been applied to the case of background risks to health. Thus, chapter 5 contributed to the literature by applying principles that were developed in the theory of decision making in the presence of multiple risks, to the realm of health.

We were motivated by the observation that environmental risks to health in the developing world are very large when compared with developed countries. Data has typically been scarce[47] but recent times have seen a marked increase in data collection effort by the WHO. The WHO Global Burden of Disease organises risks into six main groups; childhood and maternal under-nutrition, other nutrition-related risk factors and physical activity, addictive substances, sexual and reproductive health, other selected risks and environment risks. The study reports that environmental risks in particular occurring in low- and middle-income countries are sharply divergent from the pattern observed in high-income countries. Deaths from unsafe water, sanitation and hygiene account for some 3.2% of deaths in these

[47] See Measuring the Global Burden of Disease and Risk Factors, 1990-2001, Alan D. Lopez, Colin D. Mathers, Majid Ezzati, Dean T. Jamison, and Christopher J. L. Murray, (2006) p.3 for an elucidation of the problems with describing epidemiological environment in developing countries. See also Cooper, R. S., B. Osotimehin, J. S. Kaufman, and T. Forrester.1998..Disease Burden in Sub-Saharan Africa: What Should We Conclude in the Absence of Data?.Lancet 351 (9087): 208.10.

regions, compared with less than 0.1% in high income countries. Indoor smoke from household use of solid fuels is reported to account for more than 3.7% of deaths in low- and middle- income countries, but in high-income countries mortality accruing from this source is all but absent[48] (Lopez, 2006). Despite these risks, it was found in chapter 5 that in a primarily developing and emerging market sample of countries, the presence of background environmental risks did not always reduce the willingness of individuals to engage in risky behaviour. Chapter 5, therefore arrived at a conclusion that did not support the generalisation of the theory to risks over health and suggested extending the existing theory to accommodate for the properties of health and health-related risky behaviour distinct from wealth.

Hahn (2000) makes the point that

> 'health risk aversion' ... is not intended to suggest the intentional avoidance or seeking of health risk, but rather the de facto observation of higher prevalences of lower and higher numbers of risks than expected by population distributions of possible risks; aversion and affinity may or may not result from deliberate judgements regarding health outcomes (Hahn, 2000, p. 308).

Further research into environmental health risks, and their toll on decision making will complement research in chapter 5 by seeking out data sources that measure respondent's subjective risk evaluation. Bypassing the assumption that individuals simply react to environmental risks if they are present could be overcome by assessing individual subjective health risk evaluations. Although subjective evaluations would present their own measurement and reporting issues, they would accommodate for the subjective nature of individual risk expectation. Further research might also focus on examples of health risk taking that are less ambiguous than smoking. The analysis in chapter 5 is open to criticism insofar as tobacco consumption is addictive and is heavily influenced by individual social environment. Tobacco consumption is a more complex health risk, than, for example, motor racing, but it is also more relevant to daily life. Of the principal risks to health ostensibly under the control of the decision maker, few can escape the criticism that they are addictive. Although tobacco consumption is the quintessential example of an addictive good, alcohol and unhealthy food habits also have addictive properties

[48] See Table 1.2 Deaths and Burden of Disease Attributable to Risk Factors. Low- and Middle-Income Countries, High-income Countries, and World, 2001 p.10 in Lopez, (2006).

(Gearhardt, 2011). Future research will recognise that addiction is often a feature of goods that are risky to health and but also, perhaps, gambles related to wealth.

Bibliography and references

Primary Sources

National Archives

MH 55/130, Ministry of Health: Health Division: Public Health Services, Registered Files (93000 series) and Other Records. Tuberculosis: General. Treatment Schemes of Local Authorities Analysis of progress, 1926-1929.

MH 55/131, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Treatment Schemes of Local Authorities Analysis of progress, 1930-1933. Ministry of Health Memo 131/CT p.1, November 1930.

MH 55/136, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Provision of extra nourishment for tuberculous persons 01 January 1920 - 31 December 1930.

MH 55/136, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Provision of extra nourishment for tuberculous persons 01 January 1920 - 31 December. Note of discussion with Mr. Hurst (Treasury) on 10th September, 1920.

MH 55/139, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Administrative measures. Policy Memorandum (Administrative Measures against Tuberculosis). Memorandum on Administrative measures against Tuberculosis.

MH 55/140, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Promotion of Workshops for tuberculous men and women. After-care of discharged patients. National Association for the Prevention of Tuberculosis, correspondence 13th October, 1924.

MH 55/140, Ministry of Health: Health Divisions: Public Health Services,

Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Promotion of Workshops for tuberculous men and women. Some notes on the virtue and conduct of workshops for tuberculous patients.

MH 55/142, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Public Health (Tuberculosis) Bill, 1921.

MH 55/142, Ministry of Health: Health Divisions: Public Health Services, Registered Files (93,000 Series) and Other Records. Tuberculosis: General. Miscellaneous correspondence.

MH 55/156, Facilities for the treatment and technical education of tuberculous youths at Burrow Hill Sanatorium Colony, Frimley, Surrey. Circular 969, various correspondence.

MH 66/21: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Cambridgeshire. Survey Report.

MH 66/23: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Cambridgeshire. Survey appendices.

MH 66/41 : Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Cumberland. Survey Report.

MH 66/42 Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Cumberland. Survey of Welfare Services for the Blind.

MH 66/55 Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Derbyshire. Re-survey appendices A-N.

MH 66/85: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Essex. Survey appendices A-06.

MH 66/93: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Herefordshire. Survey Report.

MH 66/137: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Leicestershire. Survey appendices.

MH 66/169: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Middlesex. Survey appendices A-O.

MH 66/183: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Northumberland. Survey appendices.

MH 66/188: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Nottinghamshire. Survey appendices I.

MH 66/219: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Southampton. Survey appendices A-N, R-V.

MH 66/223 : Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Staffordshire. Survey Report.

MH 66/258 : Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Warwickshire. Survey Report.

MH 66/275: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. Worcestershire. Survey Appendices A-0.6.

MH 66/278 : Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. East Riding of Yorkshire. Survey Report, 01 January 1933 – 31 December 1934.

MH 66/280: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. East Riding of Yorkshire. Survey Appendices.

MH 66/282: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. North Riding of Yorkshire. Survey Report.

MH 66/286: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. North Riding of Yorkshire. Re-survey appendices A-O.

MH 66/292: Ministry of Health: Local Government Act 1929, Public Health Survey. County Councils. West Riding of Yorkshire. Survey appendices I.

MH 66/307 : Ministry of Health: Local Government Act 1929, Public Health Survey. Metropolitan Boroughs. Bermodnsey. Survey Report.

MH 66/351: Ministry of Health: Local Government Act 1929, Public Health Survey. Metropolitan Boroughs. Holborn. Survey Report.

MH 66/356: Ministry of Health: Local Government Act 1929, Public Health Survey. Metropolitan Boroughs. Islington. Survey appendices.

MH 66/489: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Bristol. Survey Report appendices A-O1G.

MH 66/593: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Eastbourne. Survey Report .

MH 66/742: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Manchester. Survey Report.

MH 66/748: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Mansfield. Survey appendices.

MH 66/754: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Middlesbrough. Survey Report.

MH 66/847: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Rotherham. Survey Report .

MH 66/872: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Sheffield. Survey Report appendices A-N.

MH 66/947: Ministry of Health: Local Government Act 1929, Public Health Survey. Boroughs and Urban District Councils. Wakefield. Survey appendices.

MH 66/1066: Ministry of Health: Local Government Act 1929, Public Health Survey. Survey Reports. County Boroughs. Barnsley to Birkenhead.

MH 66/1079: Ministry of Health: Local Government Act 1929, Public Health Survey. Survey Reports County Boroughs. West Ham to York.

MH 66/1081, Ministry of Health: Local Government Act 1929, Public Health Survey. Survey Reports. Metropolitan Boroughs. Chelsea to Hackney; Tuberculosis Report; Hackney.

West Yorkshire Archives (Wakefield)

WRD7/6/3/10: Annual report of Medical Officer of Health and of School Medical Officer for Barnsley 1938.

WRD7/6/3/216: Annual report of the Medical Officer of Health, Rotherham, 1933.

WRD7/6/3/217: Annual report of the Medical Officer of Health, Rotherham, 1934.

WRD7/6/3/219: Annual report of the Medical Officer of Health, Rotherham, 1936.

WRD7/6/3/282: Annual Report of the Medical Officer of Health for Wakefield, year ending December 1935.

WRD7/6/3/361: Annual report of the Tuberculosis Officer, York, 1924.

Official Publications

Census of Population, 1931, Housing Volume, Tables 12 and 13.

Census of Population, 1931, Occupational Tables, Table 16.

Census of Population, 1931, Family Size: Table 14.

Census of Population, 1931, Population Density, Table 8.

India Statistics, Public Health Statistics.

Statistical Abstract, India, 1955-1956, No. 6 (New Delhi, 1957).

Maddison Historical GDP Data
http://www.worldeconomics.com/Data/MadisonHistoricalGDP/Madison%20Historical%20GDP%20Data.efp

Ministry of Health, *Report of the Committee of Inquiry into the Anti-Tuberculosis Service in Wales and Monmouthshire*, HMSO, 1939.

National Association for the Prevention of Tuberculosis, Tuberculosis in the Commonwealth: 1947 Complete Transactions of the Commonwealth and Empire Health and Tuberculosis Conference, July, (London, 1947).

National Aeronautics and Space Administration Surface Meteorology and Solar Energy database, *eosweb.larc.nasa.gov/sse/* .

Pan American Health Organisation. 1981a Tuberculosis in the Americas, Part 1 Epidemiological Bulletin, 2.5: 1-6.

Pan American Health Organisation. 1981b. Tuberculosis in the Americas, Part II', Epidemiological Bulletin, 2.5: 1-4.

Pan American Health Organisation, 'Review of Mortality and Morbidity in the English Speaking Caribbean, 1970-1980', Epidemiological Bulletin, Vol. 2, No. 3 (1981c): 1-5.

Pan American Health Organisation, 'The Tuberculosis Situation in Argentina', Epidemiological Bulletin, Vol. 4, No. 3 (1983) pp. 8-10.

Pan American Health Organisation, 'Tuberculosis in Chile', Epidemiological Bulletin, Vol. 6, No. 1 (1985): 9-11.

Registrar General's statistical review of England and Wales (Annual), London H.M.S.O.

UCDP, 2013a. UDCP/PRIO Armed Conflict Dataset v.4-2013, 1946 – 2012, see Themnér, Lotta & Peter Wallensteen, 2013. "Armed Conflict, 1946-2012." Journal of Peace Research 50(4).

UCDP, 2013b, UCDP Battle-Related Deaths Dataset v.5-2013, 1989-2012, see Sundberg, Ralph. 2008. "Collective Violence 2002 - 2007: Global and Regional Trends." In Harbom, Lotta and Ralph Sundberg (eds.). States in Armed Conflict 2007. Uppsala: Universitetstryckeriet.

United Nations, Population Division, Demographic Yearbook, annual.

United Nations, Statistical Yearbook for Asia and the Far East, annual.

United Nations, Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat, World Population Prospects: The 2006 Revision and World Urbanization Prospects: The 2007 Revision, The 2007 Revision Population Database (on line), http://esa.un.org/unup.

World Health Organisation, Expert Committee on Tuberculosis, Report on the Fourth Session, (WHO, Geneva: 1950), Technical Report Series No. 7.

World Health Organisation, Expert Committee on Tuberculosis, Report on the Fifth Session, April (WHO, Geneva: 1951), Technical Report Series No. 32.

World Health Organisation, 1959. 'Communicable diseases in Africa: Some Facts and Figure,' *Chronicle*, 13.2: 80- 83.

World Health Organisation, 1959. 'Tuberculosis: A Period of transition,' *Chronicle*, 13.4: 169-171.

World Health Organisation, Expert Committee on Tuberculosis, Seventh Report, (WHO, Geneva: 1960), Technical Report Series No. 195.

World Health Organisation, 1960. 'Tuberculosis in Africa,' *Chronicle*, 15.11: 406-410.

World Health Organisation, 1961. 'Notifiable diseases in the Americas, 1949-1958,' *Chronicle*, 15.4: 133-141.

World Health Organisation, 1962. 'Health in the Americas,' *Chronicle*, 16.5: 151-159.

World Health Organisation, 1965. 'Health in member states,' 19.7: 269-272.

World Health Statistics Report, 1970. Special Report on Government Expenditure.

World Health Organisation World Health Statistics Report Vo1. 27, No. 3-4, (1974a): 150-188.

World Health Organisation World Health Statistics Report Vol. 27, No. 9, (1974b): 563-570.

World Health Organisation, 1982. Tuberculosis Control, Report of a Joint IUAT/WHO Study Group, Technical Report Series 671, (WHO: Geneva), Geneva.

World Health Organisation, Regional Office for the Eastern Mediterranean, Changing Patterns of Disease and their Impact on WHO Collaborative Programmes. EMRO Technical Papers Series 5, (Alexandria, Egypt: 1998a), WHO-EM/CDS/55/E/G/09.98/1000.)

World Health Organisation, WHO Report on the Global Tuberculosis Epidemic (WHO: Geneva, 1998b), 35.

World Health Organisation, Regional Office for the Eastern Mediterranean, 'Changing Patterns of Disease and their Impact on WHO Collaborative Programmes', EMRO Technical Papers Series 5, (Alexandria, Egypt, 1998c, WHO-EM/CDS/55/E/G/09.98/1000.

World Health Organisation, Statistical Information System (WHOSIS), 2001. http://apps.who.int/whosis/data/.

World Health Organisation, 2002-4. World Health Survey. http://www.who.int/healthinfo/survey/en/.

World Health Organisation, 2005. Economics of Immunization: A Guide to the Literature and Other Resources, (WHO: Geneva), WHO/V&B/04.02.).

World Bank, 2013. International Comparison Program Database, World Development Indicators.

World Health Organisation, *Global database on body mass index:* http://apps.who.int/bmi/index.jsp.

World Health Organisation, *Statistics,* Annual.

World Health Organization. *World health statistics annual 1963-1980*. Organisation mondiale de la sante.

Secondary Literature

Abel-Smith, B., 1963. 'Paying for Health Services; A Study of the Costs and Sources of Finance in Six Countries,' *Medical Care,* 1.2: 110-111.

Acemoglu, D., S. Johnson, and J. Robinson, 2001. 'The Colonial Origins of Comparative Development: An Empirical Investigation,' *American Economic Review*, 91, December, 1369-1401.

Acemoglu, D., S. Johnson, and J. Robinson, 2002. 'Reversal of Fortune: Geography and Institutions in the Making of the Modern World Income Distribution,' *Quarterly Journal of Economics*, 117, November, 1231-1294.

Adato, M., and L. Bassett, 2009. 'Social protection to support vulnerable children

and families: the potential of cash transfers to protect education, health and nutrition,' *Aids Care* 21.S1: 60-75.

Adegbola, O., 1987. 'The impact of urbanization and industrialization on health conditions: the case of Nigeria,' *World health statistics quarterly*, 40.1: 74-83.

Alanis, A. J., 2005. 'Resistance to antibiotics: are we in the post-antibiotic era?' *Archives of medical research* 36.6: 697-705.

Armstrong, A. R., 1966. 'The prevalence in Canada of drug-resistant tubercle bacilli in newly discovered untreated patients with tuberculosis,' *Canadian Medical Association Journal*, 94.9: 420.

Arora, S., 2001. 'Health, human productivity and long-term economic growth,' *The Journal of Economic History,* 61.3: 699-749.

Arellano, M., and S. Bond, 1991. 'Some tests of specification for panel data: Monte Carlo evidence and an application to employment equations.' *The review of economic studies* 58.2: 277-297.

Arrow, K., J., 1964. 'The role of securities in the optimal allocation of risk-bearing,' *The Review of Economic Studies*, 31.2: 91-96.

Auster, R., I. Leveson, and D. Sarachek, 1972. 'The production of health: an explanatory study,'' in V. Fuchs (ed). *Essays in the economics of health and medical care*, New York: National Bureau of Economic Research, Columbia University Press, pp. 135-158.

Barnes, D. S., 1995. *The making of a social disease: Tuberculosis in nineteenth-century France*, University of California Pr.

Barnes, D. S., 2000. 'Historical perspectives on the Etiology of tuberculosis,' *Microbes and Infection.* 2.4: 431-440.

Barnett, T. and A. Whiteside, 2006. *AIDS in the Twenty First Century*, second edition, New York: Palgrave Macmillan.

Bates, B., 1992. *Bargaining for Life: A Social History of Tuberculosis, 1876-1938*, University of Pennsylvania Press.

Bernard, T., S. Dercon and A. S. Taffesse, 2011. 'Beyond fatalism-an empirical exploration of self-efficacy and aspirations failure in Ethiopia.'

Besancon, L. J., 1945. 'Public health in France from the invasion to the liberation,' *War Medicine*, (November-December), 8.5: 283-289.

Bhagi, R. P., 2001. 'Changed Face of Tuberculosis in the New Millennium: A 50 Year Overview,' *Indian Journal of Tuberculosis,* 48: 117-118.

Bhargava, A., M. Pai, M. Bhargava, B. J. Marais, D. Menzies, 2012. 'Can social interventions prevent tuberculosis? The Papworth experiment (1918–1943) revisited,' *American journal of respiratory and critical care medicine*, 186.5: 442-449.

Biraud, Y. M., 1943/4. 'Health in Europe: A Survey of the Epidemic and Nutritional Situation', *Bulletin of the Health Organisation,* (League of Nations, Geneva), 10.4: 559-699.

Black, M., 1986. *Children and the Nations; The Story of Unicef* (available via UNICEF website).

Bloom, D. E., D. Canning and M. Weston, 2005. 'The Value of Vaccination,' *World Economics*, 6.3: 15.

Borgdorff, M. W., Floyd K. and J. F. Broekmans, 2002. 'Interventions to reduce tuberculosis mortality and transmission in low- and middle-income countries,' *Bulletin of the World Health Organization*, 80: 217-227.

Bowden, S., J. Jalles, A. Pereira and A. Sadler. 2014. 'Respiratory Tuberculosis and standards of living in post-war Europe,' *European Review of Economic History*, forthcoming.

Boyd Orr, J., 1936. *Food Health and Income,* Macmillan, London.

Brewer, T. F., and S. J. Heymann, 2004. 'To control and beyond: moving towards eliminating the global tuberculosis threat,' *Journal of epidemiology and community health* 58.10: 822-825.

Brimnes, N., 2008. 'BCG Vaccination and WHO's Global Strategy for Tuberculosis Control 1948–1983,' *Social Science & Medicine,* 67.5: 863-873.

Bryder, L., 1988. *Below the Magic Mountain: A Social History of Tuberculosis in 19th Britain,* Oxford.

Bulla, A., 1977. 'Global Review of Tuberculosis Morbidity and Mortality in the world 1961-1971,' *World Health Statistics Report*, 30.1: 2-38.

Burke, W. J., 2009. 'Fitting and interpreting Cragg's tobit alternative using Stata,' *Stata Journal*, 9.4: 584-592.

Burnet, E., 1932. 'General Principles Governing the Prevention of Tuberculosis,' *Quarterly Bulletin of the Health Organisation,* 1.4: 493-657.

Butler, D. and A. Sloman, 1980. *British Political Facts, 1900-1979*, 5th edition, Macmillan, London.

Canetti, G., and M. Le Lirzin, 1972. 'Trends in the prevalence of primary drug

resistance in pulmonary tuberculosis in France from 1962 to 1970: a national survey,' *Tubercle*, 53.2: 57-83.

Carson, S. A., 2010. 'The effect of development of geography, vitamin D, wealth, and agricultural productivity on tuberculosis mortality: The case of the 19th century US,' *Journal of Economic Development*, 35.2: 57-74.

Cegielski, P. and D. N. McMurray, 2004. 'The relationship between malnutrition and tuberculosis: evidence from studies in humans and experimental animals,' *International Journal of Tuberculosis and Lung Disease*, 8:286-98.

Chandra, R. K., 1992. 'Effect of vitamin and trace-element supplementation on immune responses and infection in elderly subjects,' *The Lancet*, 340.8828: 1124-1127.

Cherry, S., 1996. *Medical services and the hospitals in Britain, 1860-1939*, Cambridge University Press, Cambridge.

Clark-Curtiss, J. E. and S. E. Haydel, 2003. 'Molecular genetics of Mycobacterium tuberculosis pathogenesis,' *Annual Reviews in* Microbiology, 57.1: 517-549.

Colgrove, J., 2002. 'The McKeown Thesis: A historical controversy and its enduring influence,' *American Journal of Public Health*, 92.5 (May): 725–729.

Collins, F. M., 1971. 'Relative Susceptibility of Acid-Fast and Non-Acid-Fast Bacteria to Ultraviolet Light,' *Applied Microbiology*, 21.3: 411-413.

Comstock G. W., 1980. 'Advances towards the conquest of tuberculosis,' *Public Health Reports*, (September-October), 9.5: 440-450.

Congdon, P., and H. Southall, 2004. 'Small Areas Variations in Infant Mortality in England and Wales in the Inter-War Period and their Link with Socio-Economic Factors,' *Health Place*, 10.4: 363-382.

Cragg, J. G., 1971. 'Some statistical models for limited dependent variables with application to the demand for durable goods,' *Econometrica*, 39: 829-844.

Crowle, A. J., E. J. Ross and M. H. May, 1987. 'Inhibition by 1,25 (OH)2 vitamin D3 of the multiplication of virulent tubercle bacilli in cultured human macrophages,' *Infect Immun*; 55: 2945-2950.

Cutler, D., A. Deato and A. Lleras-Murray, 2006. 'The Determinants of Mortality,' *Journal of Economic Perspectives,* 20.3: 97-120.

Dai, G., S. Phalen, and D. N. McMurray, 1998. 'Nutritional modulation of host responses to mycobacteria,' *Front Biosci* 3, e110-22.

Dalton, T., P. Cegielski, S. Akksilp, L. Asencios, J. C. Caoili, S. N. Cho and H. Kang,

2012. ‘Prevalence of and risk factors for resistance to second-line drugs in people with multidrug-resistant tuberculosis in eight countries: a prospective cohort study,’ *The Lancet*, 380.9851: 1406-1417.

Daniel, T. M., 1997. *Captain of Death: The Story of Tuberculosis*, Rochester, NY: University of Rochester Press.

Daniel, T. M., 2006. ‘The history of tuberculosis,’ *Respiratory Medicine*, 100.11: 1862-1870.

Daniels, M., 1946. ‘Tuberculosis Mortality and Incidence in Poland’, in United Nations, Relief and Rehabilitation Administration, Health Division, *Epidemiological Information Bulletin*, Washington DC, 2.17: 725-728.

Denis, M., 1991. ‘Killing of Mycobacterium tuberculosis within human monocytes: activation by cytokines and calcitriol,’ *Clinical & Experimental Immunology* 84.2: 200-206.

Dohmen, T., A. Falk, D. Huffman, U. Sunde, J. Schupp and G. Wagner, 2005. ‘Individual risk attitudes: New evidence from a large, representative, experimentally-validated survey.’

Dormandy, T., 1999. *The White Death: A History of Tuberculosis*, The Hambledon Press, London.

Doull, J. A., 1945. ‘Tuberculosis mortality in England and certain other countries during the present war,’ *American Journal of Public Health*, (August) 35.8: 783-787.

Drolet, G., and A. Lowell, 1962. ‘Tuberculosis Mortality among Children: The Last Stage; A Statistical Review of the 1950-1959 Decade in Canada, the United States, England and Wales, and France,’ *CHEST*, 42.4: 364-371.

Drukker, D. M., 2002. ‘Bootstrapping a conditional moments test for normality after tobit estimation,’ *Stata Journal*, 2.2: 125-139.

Dubos, R. and J. Dubos, 1952. *The White Plague: Tuberculosis, Man and Society*, Little, Brown and Company, Boston.

Dye, C., Lönnroth, K., Jaramillo, E., Williams, B. G., & Raviglione, M., 2009. ‘Trends in tuberculosis incidence and their determinants in 134 countries,’ *Bulletin of the World Health Organisation*, 87.9: 683-691.

Eeckhoudt, Louis, Christian Gollier, and Harris Schlesinger, 1996. ‘Changes in background risk and risk taking behavior,’ *Econometrica*, 64.3: 683-689.

Espinal, M. A., 2003. ‘The global situation of MDR-TB,’ *Tuberculosis*, 83.1: 44-51.

Fitzgibbon, B., 1993. 'EPI Implementation in Togo' in Stanley O. Foster (ed), *Immunization in 12 African Countries 1982-1993*, United States Agency for International Development, Africa Regional Project (698-0421), United States Agency for International Development, Chapter 4.

Foster, S. O., 1993. 'Critical Elements in EPI Implementation', in Stanley O. Foster (ed), *Immunization in 12 African Countries 1982-1993*, United States Agency for International Development, Africa Regional Project (698-0421), United States Agency for International Development.

Gagneux, S., C. D. Long, P. M. Small, T. Van, G. K. Schoolnik, and B. J. Bohannan, 2006. 'The competitive cost of antibiotic resistance in *Mycobacterium tuberculosis*,' *Science*, 312: 1944-1946.

Gandhi, N. R., A. Moll, A. W. Sturm, R. Pawinski, T. Govender, U. Lalloo and G. Friedland, 2006. 'Extensively drug-resistant tuberculosis as a cause of death in patients co-infected with tuberculosis and HIV in a rural area of South Africa,' *The Lancet* 368.9547: 1575-1580.

García, J. and J. Labeaga, 1996. 'Alternative approaches to modeling zero expenditure: An application to Spanish demand for tobacco,' *Oxford Bulletin of Economics and Statistics*, 58.3: 489-506.

Gearhardt, A. N., C. M. Grilo, R. J. DiLeone, K. D. Brownell and M. N. Potenza, 2011. 'Can food be addictive? Public health and policy implications,' *Addiction*, 106.7: 1208-1212.

Gollier, C. and J. W. Pratt, 1996. 'Risk vulnerability and the tempering effect of background risk,' *Econometrica*, 64.5: 1109-1123.

Gorsky, M., 2008. 'Public health in interwar England and Wales: did it fail?' *Dynamis*, 28:175-98.

Gorsky, M., 2011. 'Local Government Health Services in Interwar England: Problems of Quantification and Interpretation,' *Bulletin of the History of Medicine,* Fall, 384-412.

Grange, J. M., 1999. 'The Global Burden of Tuberculosis', in John D.H. Porter and John M. Grange (eds.), *Tuberculosis: An Interdisciplinary Perspective*, London: Imperial College Press.

Greene, W. H., 2012. *Econometric Analysis (Seventh Edition)*, New York University, Prentice Hall.

Groth-Peterse, E., J. Knudsen and E. Wilbek, 1959. 'Epidemiological Basis of

Tuberculosis Eradication in an Advanced Country', *Bulletin of the World Health Organisation*, 21.1: 5-49.

Grzybowski, S., 1972. 'BCG in Developing versus developed Countries', Section IV in *Status Of Immunization In Tuberculosis In 1971: Report Of A Conference On Progress To Date; Future Trends And Research Needs*, 26-28 October 1971, US Department of Health, Education and Welfare, Public Health Service, DHEW Publication No. (NIH) 72-68 and Fogarty International Center Proceedings No. 14, Table 8, 131.

Guiso, L. and T. Jappelli, 1996. 'Income risk, borrowing constraints, and portfolio choice,' *The American Economic Review*, 86.1: 158-172.

Guiso, L. and M. Paiella, 2008. 'Risk aversion, wealth and background risk,' *Journal of the European Economic Association*, 6.6: 1109-1152.

Gulaid, L. A., 1993. 'Immunization in Africa: Achievements, Challenges, Resources'. in Stanley O. Foster (ed), *Immunization in 12 African Countries 1982-1993*, United States Agency for International Development, Africa Regional Project (698-0421), United States Agency for International.

Guo, G., 1993. 'Mortality trends and causes of death: a comparison between Eastern and Western Europe, 1960s-1980s', *European Journal of Population*, 9.3: 287-312.

Gupta, U. D., and V. M. Katoch, 2005. 'Animal models of tuberculosis,' *Tuberculosis*, 85.5: 277-293.

Hargreaves, J. R., D. Boccia, C. A. Evans, M. Adota, M. Petticrew and J. D. Porter, 2011. 'The social determinants of tuberculosis: from evidence to action,' *Journal Information*, 101.4: 654-662.

Harris, B., 1995. *The Health of the Schoolchild: A History of the School Medical Service in England and Wales*, Buckingham: Open University Press.

Heaton, J. and D. Lucas, 2000. 'Portfolio choice in the presence of background risk,' *The Economic Journal*, 110.460: 1-26.

Hegsted, M. cited in Register, U.D., et al., 1973. 'The vegetarian diet,' *J Am Diet Assoc*, 62:255.

Hemila, H., 1996. 'Vitamin C and common cold incidence: a review of studies with subject under heavy physical stress,' *International Journal of Sports Medicine*, 78:857-66.

Hemila, H., 1997a. 'Vitamin C intake and susceptibility to the common cold,' *British*

Journal of Nutrition, 77: 59-72.

Hemila, H., 1997b. 'Vitamin C intake and susceptibility to pneumonia,' *The Pediatric Infectious Disease Journal*, 16: 836-7.

Hemila, H., J. Kapiro, P. Pietinen, D. Albanes and O. P. Helnonen, 1999. 'Vitamin C and other compounds in vitamin C rich food in relation to risk of tuberculosis in male smokers,' *American Journal of Epidemiology*, 150.6: 632-641.

Hernandez-Frontera, E. V. A. U. R. E. L. Y., and D. N. McMurray, 1993. 'Dietary vitamin D affects cell-mediated hypersensitivity but not resistance to experimental pulmonary tuberculosis in guinea pigs,' *Infection and Immunity* 61.5: 2116-2121.

Herzog, B. H., 1998. 'History of tuberculosis,' *Respiration*, 65.1: 5-15.

Hillbeboe, H. E. and J. Holm, 1947. 'International Control of Tuberculosis,' *Public Health Reports*, 62.31: 1114-1128.

Hochguertel, S., 1998. 'Households' portfolio choices,' doctoral thesis.

Holt, C. A. and S. K. Laury, 2002. 'Risk Aversion and Incentive Effects,' *American Economic Review*, 92.5: 1644-1655.

Hong, Y. P., S. J. Kim, E. G. Lee, W. J. Lew and J. Y. Bai. 1999. 'Treatment of bacillary pulmonary tuberculosis at the chest clinics in the private sector in Korea, 1993,' *The International Journal of Tuberculosis and Lung Disease*, 3.8: 695-702.

Houben, E., L. Nguyen, and J. Pieters, 2006. 'Interaction of pathogenic mycobacteria with the host immune system,' *Current opinion in* microbiology, 9.1: 76-85.

Howells, G., 1969. 'Primary drug resistance in Australia in 1968,' *Tubercle*, 50.4: 344-349.

Hsieh, C., 1998. 'Health risk and the decision to quit smoking,' *Applied economics*, 30.6: 795-804.

Huong, N. T., B. D. Duong, N. V. Co, H. T. Quy, L. B. Tung, J. F. Broekmans, M. C. Bosman et al. 2006. 'Tuberculosis epidemiology in six provinces of Vietnam after the introduction of the DOTS Strategy,' *Tuberculosis control in Vietnam: Does DOTS do it?* 10.9: 963-969.

Ibs, K. and L. Rink, 2003. 'Zinc-altered immune function,' *The Journal of nutrition*, 133.5: 1452S-1456S.

Institute of Medicine, 2000. *Ending neglect: the elimination of tuberculosis in the United States*, Washington, DC: National Academic Press.

Iskander, M. G., 1987. *UNICEF in Africa South of the Sahara: A Historical Perspective,* United Nations Children's Fund.

Jacobs, D. R. and L. M. Steffen, 2003. 'Nutrients, foods, and dietary patterns as exposures in research: a framework for food synergy,' *Am J Clin Nutr*, 78(suppl): 508S-13S.

Jacobs, D. R and L. C. Tapsell, 2007. 'Food, not nutrients, is the fundamental unit in nutrition,' *Nutr Rev*, 65: 439-50.

Jeon, C. Y., and M. B. Murray, 2008. 'Diabetes mellitus increases the risk of active tuberculosis: a systematic review of 13 observational studies,' *PLoS medicine*, 5.7: e152.

Johnson, R., E. M. Streicher, G. E. Louw, R. M. Warren, P. D. Van Helden and T. C. Victor, 2007. 'Drug resistance in M. tuberculosis,' *Understanding the mechanisms of drug resistance in enhancing rapid molecular detection of drug resistance in Mycobacterium tuberculosis,* 7.

Johnston, W. D., 1995. *The Modern Epidemic: A History of Tuberculosis in Japan*, Cambridge, Mass. Harvard University Press.

Johnston, W. D., 2003. 'Tuberculosis' in The Cambridge Historical Dictionary of Disease (ed. Kenneth Kiple), Cambridge: Cambridge University Press.

Kamat R., J. J. Y. Dawson, S. Devadatta, W. Fox B. Janardhanam, S. Radhakrishna, C. V. Ramakrishnan, P. R. Somasundaram, H. Stott and S. Velu, 1966., 'A Controlled Study of the Influence of Segregation of Tuberculous Patients for One Year on the Attack Rate of Tuberculosis in a 5- year Period in Close Family Contacts in South India', *Bulletin of the World Health Organisation*, 34, 4: 517–532.

Karyadi, E., W. Schultink, R. H. Nelwan, R. Gross, Z. Amin, W. Dolmans, J. W. M. van der Meer, J. G. A. Hautvast and C. E. West, 2000. 'Poor micronutrient status of active pulmonary tuberculosis patients in Indonesia,' *The Journal of nutrition* 130.12: 2953-2958.

Kearns, G., 1995. 'Tuberculosis and the Medicalisation of British Society, 1880-1920,' in John Woodward and Robert Jutte (eds.), *Coping with Sickness: Historical Aspects of Health Care in a European Perspective*, European Association for the History of Medciene and Health Pubications: Sheffield, 147-170.

Kenyon, T. A., Mwasekaga M. J., Huebner R., et al., 1999, 'Low levels of drug

resistance amidst rapidly increasing tuberculosis and human immunodeficiency virus co-epidemics in Botswana,' *Int J Tuberc Lung* Dis, 3:4-11.

Kimball, M. S., 1990. 'Precautionary saving and the marginal propensity to consume,' No. w3403. National Bureau of Economic Research.

Kimball, M. S., 1993. 'Standard risk aversion,' *Econometrica*, 61.3: 589-611.

King, M. (ed.), 1966. *Medical Care in Developing Countries*, Lusaka and London: Oxford University Press.

Kuznets, S., 1955. 'Economic growth and income inequality,' *The American economic review*, 1-28.

Lane, J., 2001. *A Social History of Medicine: Health, Healing and Disease in England*, 1750:1950, Routledge, London.

Lee, J., 2008. 'The effect of the background risk in a simple chance improving decision model,' *Journal of Risk and Uncertainty*, 36.1: 19-41.

Leung, C. C., T. H. Lam, W. M. Chan, W. W. Yew, K. S. Ho, G. Leung, W. S. Law, C. M. Tam, C. K. Chan and K. C. Chang, 2007. 'Lower risk of tuberculosis in obesity,' *Archives of Internal Medicine*, 167.12: 1297.

Link, B. G. and J. C. Phelan. 2002. 'McKeown and the idea that social conditions are fundamental causes of disease,' *American Journal of Public Health*: May, 92.5: 730-732.

Lönnroth, K., B. G. Willians, P. Cegielski and C. Dye, 2010. 'A consistent log-linear relationship between tuberculosis incidence and body mass index,' *International journal of epidemiology* 39.1: 149-155.

Lopez, A. D., C. D. Mathers, M. Ezzati, D. T. Jamison and C. J. L. Murray, 2006. 'Global and regional burden of disease and risk factors 2001:systematic analysis of population health data,' *The Lancet*, 367.9524: 1747-1757.

Lopez, A. D. and J. L. Murray, 1997. 'Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study,' *Lancet*, 349.9063: 1436-1442.

Lucas, R., 1988, 'On The Mechanics Of Economic Development,' *Journal Of Economic Theory*, 22.1: 103-124.

Lusk, J. L., and K. H. Coble, 2008. 'Risk aversion in the presence of background risk: Evidence from the lab' in J. C. Cox and G. W. Harrison (Eds), *Risk aversion in experiments* (Vol. 12). Bingley, UK: Emerald, Research in

Experimental Economics.

Powell, M., 2005. 'Coasts and Coalfields: The Geographical Distribution of Doctors in England and Wales in the 1930s,' *Social History of Medicine*, 18.2: 245-263.

Malik, V. S., M. B. Schulze, and F. B. Hu, 2006. 'Intake of sugar-sweetened beverages and weight gain: a systematic review,' *The American journal of clinical nutrition*, 84.2: 274-288.

Martineau, A. R., P. M. Timms, G. H. Bothamley, Y. Hanifa, K. Islam, A. P. Claxton, G. E. Packe and C. J. Griffiths, 2011. 'High-dose vitamin D3 during intensive-phase antimicrobial treatment of pulmonary tuberculosis: a double-blind randomised controlled trial,' *The Lancet*, 377.9761: 242-250.

Martini, C. J., G. H. Allan, J. Davison and E. M. Backett, 1977. 'Health indexes sensitive to medical care variation,' *International Journal of Health Services*, 7.2: 293-309.

McCarthy, O. R. 2001. 'The key to the sanatoria,' JRSM, 94.8: 413-417.

McDougall, J. B., 1945. *Tuberculosis in Greece*, United Nations Relief and Rehabilitation Administration, Health Division, Epidemiological Information and Vital Statistics Report, I, 20, (November 30th).

McDougall, J. B., 1950. 'Tuberculosis Mortality 1937 to 1949,' Epidemiological and Vital Statistics Report, III, 10 (October, World Health Organisation: Geneva.

McDougall, J. B., 1952. 'Epidemiological factors in Tuberculosis control,' *Bulletin of the World Health Organisation*, 7.2: 111-152.

McKeown, T., R. G. Record and R.D. Turner, 1975. 'An interpretation of the decline in mortality in England during the twentieth century,' *Population Studies*, 29.3: 391-422.

McKeown, T., 1976a, *The Modern Rise of Population*, Academic Press, New York.

McKeown, T., 1976b. *The Role of Medicine: Dream, Mirage, or Nemesis?* Nuffield Provincial Hospitals Trust, London, England.

McKinlay, J. B. and S. M. McKinlay, 1977. 'The questionable contribution of medical measures to the decline in the United States in the twentieth century,' *Milbank Memorial Fund Quarterly*, Health and Society, (Summer) 55.3: 405-428.

McMurray, D. N., and E. A. Yetley, 1983. 'Response to Mycobacterium bovis BCG vaccination in protein- and zinc-deficient guinea pigs,' *Infection and Immunity*, 39:755-761.

McMurray, D. N., 1984. ‘Cell-mediated immunity in nutritional deficiency,’ *Progress in food & nutrition science* 8.3-4: 193.

McMurray, D. N., M. A. Carlomagno, C. L. Mintzer, and C. I. Tetzlaff, 1985. ‘Mycobacterium bovis BCG vaccine fails to protect protein-deficient guinea pigs against respiratory challenge with virulent Mycobacterium tuberculosis,’ *Infection and Immunity*, 50: 555-559.

McMurray, D. N., C. L. Mintzer, C. I. Tetzlaff and M. A. Carlomagno, 1986. ‘Influence of dietary protein on the protective effect of BCG in guinea pigs,’ *Tubercle*, 67: 31-39.

McMurray, D. N., R. A. Bartow, C. L. Mintzer and E. Hernandez-Frontera, 1990. ‘Micronutrient status and immune function in tuberculosis,’ *Annals of the New York Academy of Sciences*, 587: 59.

M’Conigle G. C. M. and J. Kirby, 1936. *Poverty and Public Health*, Gollancz, London.

Medical Research Council, 1942. 'Report of the Committee on Tuberculosis in War Time,' *Special Report Series*, No. 246, London.

Messina, M., J. W. Lampe, D. F. Birt, et al., 2001. ‘Reductionism and the narrowing nutrition perspective: time for reevaluation and emphasis on food synergy,’ *J Am Diet Assoc*, 101: 1416-9.

Migliori, G. B., R. Loddenkemper, F. Blasi and M. C. Raviglione, 2007a. ‘125 years after Robert Koch's discovery of the tubercle bacillus: the new XDR-TB threat. Is “science” enough to tackle the epidemic?’ *European Respiratory Journal*, 29.3: 423-427.

Migliori, G. B., G. De laco, G. Besozzi, R. Centis and D. M. Cirillo, 2007b. ‘First tuberculosis cases in Italy resistant to all tested drugs,’ *Euro surveill*, 12.5: E070517.

Miller A. B., R. Tall, W. Fox, M. J. Lefford, D. A. Mitchison, 1966. ‘Primary drug resistance in pulmonary tuberculosis in Great Britain: second national surve,’ *Tubercle*, 47: 92-107.

Milstien, J., 1993. *Tuberculosis, The Immunological Basis For Immunization Series*, WHO Global Programme For Vaccines And Immunization: Expanded Programme On Immunization (WHO: Geneva, 1993), WHO/EPI/GEN/93.15.

Moffatt, P. G., 2005. ‘Hurdle models of loan default,’ *Journal of the Operational Research Society*, 56.9: 1063-1071.

Morcos, M. M., A. A. Gabr, S. Samuel, M. Kamel, M. El Baz, M. El Beshry and R. R Michail, 1998. 'Vitamin D administration to tuberculous children and its value,' *Bollettino chimico farmaceutico,*137.5: 157-164.

Morris J. N. and R. M. Titmuss, 1944. 'Health and Social Change,' *Medical Officer*, 26 August (69-71), 2 September (77-79) and 9 September (85-7).

Muir, B., 2002. AIDS Community Research Initiative of America, Spring.

Murray, C. 1994. 'Resource Allocation Priorities: Value for Money in Tuberculosis Control' in J. D. H. Porter and K. P. W. J. McAdam (eds.) Tuberculosis: Back to the Future, Chapter 9: 193-208.

Nair, S., 1954 'Morbidity and mortality statistics regarding tuberculosis in India' *Indian Journal of Tuberculosis,* December.

Neville, J., 2012. 'Explaining local authority choices on public hospital provision in the 1930s: a public policy hypothesis,' *Medical History,* Jan; 56.1: 48-71.

Niemann, S., R. Diel, G. Khechinashvili, M. Gegia, N. Mdivani and Y. Tang, 2010. 'Mycobacterium tuberculosis Beijing lineage favors the spread of multidrug-resistant tuberculosis in the Republic of Georgia,' *Journal of clinical microbiology* 48.10: 3544-3550.

Nursyam, E. W., Z. Amin and C. M. Rumende, 2006. 'The effect of vitamin D as supplementary treatment in patients with moderately advanced pulmonary tuberculous lesion,' *Hemoglobin,* 1500:1500.

Olukunie A. 1987. 'The impact of urbanization and industrialization on health conditions: the case of Nigeria,' *World Health Statistics Quarterly*, 40.1: 74-83.

Onwubalili, J. K., 1988. 'Malnutrition among tuberculosis patients in Harrow, England,' *European Journal of Clinical Nutrition* 42.4: 363-366.

Ott, K., 1996. *Fevered Lives: TB in American Culture since 1870,* Cambridge, Mass; London Harvard University Press.

Owen, N. G. (ed.), 1987. *Death and Disease in Southeast Asia*, Oxford University Press.

Oya-Sawyer, D., R. Fernandez-Castilla, Luis de Melo, and R. Monte-Mor, 1987. 'The impact of urbanization and industrialization on mortality in Brazil,' *World Health Statistics Quarterly,* 40.1: 84-95.

Packard, R. M., 1989. 'Industrial production, health and disease in sub-Saharan Africa,' *Social science and medicine,* 28.5: 475-496.

Peyron, P., J. Vaubourgeix, Y. Poquet, F. Levillain, C. Botanch, F. Bardou, M. Daffe

et al., 2008. 'Foamy macrophages from tuberculous patients' granulomas constitute a nutrient-rich reservoir for M. tuberculosis persistence,' *PLoS pathogens*, 4.11:e1000204.

Phillips, D. R., 1988. *The Epidemiological Transition in Hong Kong; Changes in Health and Disease since the Nineteenth Century,* Centre of Asian Studies: University of Hong Kong.

Rail, L. C., and S. N. Meydani, 1993. 'Vitamin B6 and immune competence,' *Nutrition Reviews*, 51.8: 217–225.

Raviglione, M. C., P. P. Nunn, A. Kochi and R. J. O'Brien. 1996. 'The Pandemic of HIV-Associated Tuberculosis', in Jonathan Mann and Daniel Tarantola, (eds.), *AIDS in the World II; Global Dimensions, Social Roots and Responses*, (Oxford University Press), Chapter 5: 87-96.

Raviglione, M. C., 2003. 'The TB epidemic from 1992 to 2002,' *Tuberculosis*, 83.1: 4-14.

Reingold, A. L. and C. R. Phares, 2006. 'Infectious Diseases' in M. H. Merson, R. E. Black and A. J. Mills (eds.), *International Public Health: Diseases, Programs, Systems and Policies*, 2nd edition, Sudbury, Massachusetts: James and Bartlett Publishers, Chapter 4.

Riley, J. C., 2001. *Rising Life Expectancy: A Global History*, Cambridge: New York: Cambridge University Press, Chapter 4.

Rocha, C., R. Montoya, K. Zevallos, A. Curatola, W. Ynga, J. Franco, F. Fernando et al., 2011. 'The Innovative Socio-economic Interventions Against Tuberculosis (ISIAT) project: an operational assessment,' *The international journal of tuberculosis and lung disease: the official journal of the International Union against Tuberculosis and Lung Disease*, 15.Suppl 2: 50.

Roemer, M. I., 1976. *Health Care Systems in World Perspectives*, Health Administrative Press, Ann Arbor.

Rothman, S., 1994. *Living in the Shadow of Death: Tuberculosis and the Social Experience of Illness in America* (New York, Basic Books).

Roworth, M., S. Beck, P. Christie, S. Ahmed, C. Ramsay, 2002. 'Time to abandon routine school BCG immunisation in Scotland?' *Health Protection Scotland, Weekly Report Articles*, 36.38: 246 – 248.

Royston, P., 2007. 'Multiple imputation of missing values: further update of ice, with

emphasis on interval censoring,' *Stata Journal*, 7.3: 445-464.

Smallman-Raynor, M., and A. D. Cliff., 2003. 'War and disease: some perspectives on the spatial and temporal occurrence of tuberculosis in wartime' in Matthew Gandy and Alimuddin Zumla (eds.), *The Return of the White Plague; Global Poverty and the 'New' Tuberculosis*, Verso: London and New York, Chapter 4, 70-92.

Smallman-Raynor, M. and A. D. Cliff. 2004. *War epidemics; An historical geography of infectious diseases in military conflict and civil strife*, Oxford, Oxford University Press.

Smith, F. B., 1988. *The Retreat of Tuberculosis, 1850-1950*, Croom Helm, London.

Smith, P. G., 'Discussion on "Tuberculosis: The World Situation"' in J. D. H. Porter and K. P. W. J. McAdam, (eds.) *Tuberculosis: Back to the Future*, (John Wiley, 1994), 31-33.

STATA, November, 2000. 'TECHNICAL STB-58.'

Pablos-Méndez, A., M. C. Raviglione, A Laszlo, N. Binkin, H. L. Rieder, F. Bustreo, D. L. Cohn et al., 1998. 'Global surveillance for antituberculosis-drug resistance, 1994-1997,' *New England Journal of Medicine* 338.23: 1641-1649.

Porter, D., 1999. *Health, Civilisation and the State: A History of Public Health from Ancient to Modern Times*, Routledge, London.

Pratt, J. W., 1964. 'Risk Aversion in the Small and in the Large,' *Econometrica*, 32.1-2: 112-136.

Pratt, J. W., and R. J. Zeckhauser, 1987. 'Proper risk aversion,' *Econometrica*, 55.1: 143-154.

Pritchett, L. and L. H. Summers, 1996. 'Wealthier is Healthier,' *Journal of Human Resources*, 31.4: 841-868.

Saha, K. and K. N. Rao, 1989. 'Undernutrition in lepromatous leprosy: V. Severe nutritional deficit in lepromatous patients co-infected with pulmonary tuberculosis,' *European Journal of Clinical Nutrition*, 43: 117-128.

Semple, A. B., 1951. 'The Tuberculosis Problem,' *Public Health*, February, 74-75.

Sen, A., 1999. *Development as freedom*, Oxford University Press.

Seung, K. J., G. H. Bai, S. J. Kim, W. J. Lew, S. K. Park and J. Y. Kim. 1999. 'The treatment of tuberculosis in South Korea,' *International Journal of Tuberculosis Lung Disease*, 7.10: 912-919.

Singer, H. W. 1937, 'Unemployment and Health,' *Pilgrim Trust Enquiry Interim Paper*, No. 4, London.

Smallman-Raynor, M. and A. D. Cliff, 2004. *War epidemics; An historical geography of infectious diseases in military conflict and civil strife*, Oxford University Press.

Smith, F. B., 1988. *The Retreat of Tuberculosis, 1850-1950*, Croom Helm, London.

Snider, D. E., 'Tuberculosis: the World Situation: History of the Disease and Efforts to Combat It,' in J. D. H. Porter and K. P. W. J. McAdam, (eds.) *Tuberculosis: Back to the Future*, (Chichester: John Wiley, 1994).Chapter 1, 19.

Stern, David I., 2004.'The rise and fall of the environmental Kuznets curve.' *World development* 32.8: 1419-1439.

Suárez, P. G., C. J. Watt, E. Alarcon, J. Portocarrero, D. Zavala, R. Canales, F. Luelmo, M. A. Espinal and C. Dye, 2001. 'The dynamics of tuberculosis in response to 10 years of intensive control effort in Peru,' *Journal of Infectious Diseases* 184.4: 473-478.

Szreter, S., 1988. 'The importance of social intervention in Britain's mortality decline c. 1850-1914: a reinterpretation of the role of public health,' *Social History of Medicine*, 1.1: 1-38.

Szreter, S., 2002. 'Rethinking McKeown: the relationship between public health and social change,' *American Journal of Public Health*, 92.5: 722-5.

Teller, M. 1985. *The Tuberculosis Moment*, Routledge, New York.

Titmuss, R. M., 1938. *Poverty and Population: A Factual Study of Contemporary Social Waste*, Macmillan, London.

TO, FROM SOVIET CONTEXT, 2000. 'The Russian equation: an evolving paradigm in tuberculosis control,' *Int J Tuberc Lung Dis*, 4.12: 5160-5167.

Tsukaguchi, K., T. Yoneda, M. Yoshikawa, N. Narita, Y. Enoki, R. Miyazaki, F. Shirai, H. Kitamura and M. Tsukaguchi, 1991. 'Interaction between interleukin-1 and tumor necrosis factor productions by peripheral blood monocytes and nutritional disturbance in active pulmonary tuberculosis,' *Kekkaku:[Tuberculosis]* 66.7: 477.

Tuberculosis Chemotherapy Centre, 1959. 'A Concurrent Comparison of Home and Sanatorium Treatment of Pulmonary Tuberculosis in South India,' *Bulletin of the World Health Organisation*, 21.1: 51-144.

UCDP, 2005. 'UCDP Battle-related Deaths Dataset Codebook: Definitions, sources

and methods for the UCDP Battle-related death estimates,' v. 5.0, Uppsala University.

Wakamatsu, E., H. Oka, H. Kumabe and A. Kobayashi, 1969. 'The Trend of Tuberculosis in Japan during the Period 1953-64; Comparison of the Results of Tuberculosis Prevalence Surveys,' *Bulletin of the World Health Organisation*, 41: 115-136.

Webster, C., 1982. 'Healthy or Hungry Thirties?' *History Workshop Journal*, 13: 110-129.

Webster, C., 1985. 'Health, Welfare and Unemployment during the Depression,' *Past and Present*, 109.1: 204-230.

Webster, C., 1994. 'Saving Children during the Depression: Britain's Silent Emergency, 1919-1939,' *Disasters*, 18.3: 213-220.

Weil, P., 1992. 'Equilibrium asset prices with undiversifiable labor income risk,' *Journal of Economic Dynamics and Control*, 16.3: 769-790.

Wejse, Christian, V. F. Gomes, P. Rabna, P. Gustafson, P. Aaby, I. M. Lisse, P. L. Andersen, H. Glerup and M. Sodemann 2009. 'Vitamin D as supplementary treatment for tuberculosis: a double-blind, randomized, placebo-controlled trial,' *American journal of respiratory and critical care* medicine, 179.9: 843-850.

Welshman, J. 1997. 'The Medical Officer of Health in England and Wales, 1900-1974: watchdog or lapdog?' *Journal of Public Health of Medicine*, December, 19, 4, 443-450.

Wieland, C. W., S. Florquin, E. D. Chan, J. C. Leemans, S. Weijer, A. Verbon, G. Fantuzzi and T. van der Poll, 2005. 'Pulmonary Mycobacterium tuberculosis infection in leptin-deficient ob/ob mice,' *International immunology*, 17.11: 1399-1408.

Wiles, F. J., 1947. 'The effect of tuberculosis in the Bantu' in National Association for the Prevention of Tuberculosis, Tuberculosis in the Commonwealth: 1947 complete transactions of the Commonwealth and Empire Health and Tuberculosis Conference, London July.

Wilkinson, R. J., M. Llewelyn, Z. Toossi, P. Patel, G. Pasvol, A. Lalvani, D. Wright, M. Latif and R. N. Davidson, 2000. 'Influence of vitamin D deficiency and vitamin D receptor polymorphisms on tuberculosis among Gujarati Asians in west London: a case-control study,' *The Lancet* 355.9204: 618-621.

Williame, J., et al. 1989. 'HIV Infection in Patients with Tuberculosis in Kinshasa, Zaire,' *American Review of Respiratory Disease*, 139.5: 1082-1085

Wilson, L. G., 1990. 'The historical decline of Tuberculosis in Europe and America: Its causes and significance,' *Journal of the History of Medicine and Allied Sciences*, 45.3: 366-396.

Winslow, C. E. A., 1951. *The Cost of Sickness and the Price of Health*, World Health Organisation Monograph Series, World Health Organisation, Geneva.

Wrigley E. A., and R. Schofield, 1981. *The Population History of England 1541–1871: A Reconstruction*, Cambridge University Press, Cambridge, England.

World Health Organisation, 1950. *Expert Committee on Tuberculosis, Report on the Fourth Session, Technical Report Series No.* 7 (Geneva).

World Health Organisation, 1951. *Expert Committee on Tuberculosis, Report on the Fifth Session, Technical Report Series No.* 32, April.

World Health Organisation, 1951. *Annual Epidemiological and Vital Statistics 1939 - 1946*. Part 1, Table 52, pp 96 and 97.

World Health Organisation, 1952. *Annual Epidemiological and Vital Statistics 1947-1949*, Part 1, Table 70, pp 214-215

World Health Organisation, 1954. *Vaccination against Tuberculosis; Sixth Report of the Expert Committee on Tuberculosis*, Technical Report Series No. 88.

World Health Organisation, 1960. *Expert Committee on Tuberculosis, Seventh Report*, Technical Report Series No. 195.

World Health Organisation, 1998b. WHO Report on the Global Tuberculosis Epidemic (WHO: Geneva), 35.

World Health Organisation, 2001.'WHO Global Strategy for Containment of Antimicrobial Resistance.'

World Health Organization, 2006. 'Extensively drug-resistant tuberculosis (XDR.TB): recommendations for prevention and control,' *Weekly Epidemiol Record*, 81:430–432.

World Health Organisation, 2013. 'Multidrug-resistant tuberculosis (MDR-TB 2013 Update,' Retrieved 14 June 2013.

Xu, P., J. Peccia, P. Fabian, J. W. Martyny, K. P. Fennelly, M. Hernandez and S. L. Miller., 2003. 'Efficacy of ultraviolet germicidal irradiation of upper-room air In inactivating airbourne bacterial spores and mycobacteria in full-scale studies,' *Atmospheric Environment*, 37.3: 405-419.

Yamaguchi, M., H. Oka, H. Kumabe and H. Yosano, H. 1959. ‘Survey of Tuberculosis Prevalence in Japan, 1954,’ *Bulletin of the World Health Organisation* 21: 145-159.

Yamaguchi, M., H. Oka, H. Kumabe, and A. Kobayashi, 1962. ‘The trend of Tuberculosis in Japan during the period 1953-1958,’ *Bulletin of the World Health Organisation,* 26.1: 19-45.

Young, D., 2009. ‘Animal models of tuberculosis,’ *European Journal of Immunology* 39.8: 2011-2014.