The Contribution of Improved Health to Standards of Living in Twentieth Century England and Wales

DOCTORAL THESIS IN ECONOMIC HISTORY

Department of Economic History
London School of Economics and Political Science

Wholeheartedly for the Light of my Life

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Abstract

The thesis will highlight both qualitatively and quantitatively that during the twentieth century the English population experienced unprecedented improvements in mortality and particularly morbidity, which has provided a substantial boost to standards of living and economic development. Despite the extensiveness of these health improvements, there have been a very limited number of attempts to evaluate and quantify these valuable improvements. None of the existing studies that quantitatively assess improved health actually measure health per se, as they all utilise mortality as a proxy. Furthermore, there have been no historical studies that aim to map the evolution of improving health from the perspective of quality of life for illness sufferers. The thesis will fill all of these voids through developing a quantitative health (mortality and morbidity) measuring tool that is capable of providing (monetary) estimates about the contribution of improved health to standards of living and economic developments in twentieth century England. This will be applied to key case study illnesses (blindness, breast cancer, stomach cancer and tuberculosis) and then extrapolated forward to include all illnesses which will be combined with mortality in order to provide an aggregate health index for twentieth century England.

The results of this exercise provide a significant contribution to the twentieth century health and economic history of England. The thesis findings that, at a most conservative estimate, the value of twentieth century health improvements is in excess of 33 billion (1990 international $) substantially adds to a new view of the economics of health and provides very valuable historical detail. This new view is that improvements in health have been a major contributor to economic welfare in twentieth century England. Put another way: the thesis will highlight that during the twentieth century increases in life expectancy and improvements in the quality of life associated with morbidity have provided a considerable contribution to standards of living and the growth of GDP defined on a utility, 'Fisherian' basis, whereby economic growth nearly doubles, from 1.4 percent for GDP only versus 2.6 percent when GDP is adjusted for improved health.

TABLE OF CONTENTS

List of Tables

All tables relate to England and Wales, but will be summarised as England when cited, unless otherwise referenced.

List of Tables, Figures, Equations

List of Figures

All figures relate to England and Wales, but will be summarised as England when cited, unless otherwise referenced.

List of Equations

List of Abbreviations

BC: breast cancer

CV: contingent valuation

GDP: gross domestic product

GDP pc: gross domestic product per capita

NHS: National Health Service

QALE: quality adjusted life expectancy

QALY: quality adjusted life year

SC: stomach cancer

TNA: The National Archives

VSHLY: value of a statistical healthy life year

VSL: value of a statistical life

WTP: willingness to pay

WTP$_{MB}$: willingness to pay (morbidity)

WTP$_{MT}$: willingness to pay (mortality)

TB: tuberculosis

EuroQol: EuroQol standardised spectrum

PART I

1. Introduction

During the twentieth century there were significant improvements in the life expectancy of the English population. Improvements in life expectancy are one important manifestation of improvements in human welfare[1]. Furthermore, improvements in the quality of life associated with illness increased, which provided important contributions to the standards of living of the twentieth century English population.

Despite the importance of improved health and the numerous implications for standards of living and economic prosperity that these changes in health have created, there have been a very limited number of attempts to evaluate and quantify these developments in mortality, and especially morbidity[2]. The only studies that have attempted to value health (in an extended form of GDP measure, i.e. through 'Fisherian' type considerations[3]) are Nordhaus (1999)[4] for the USA between 1900 and 1995, Crafts (2001)[5] for the UK between 1870 and 1998 and Hickson (2002)[6] for twentieth century Japan. However, none of these studies measure health per se, as they all utilise mortality (i.e. increased life expectancy) as a proxy for health.

Efforts to measure the health of the population pose a new, difficult and increasingly relevant challenge. Unprecedented gains in life expectancy accompanied by concerns related to the level of health in these extended life years have exacerbated the need to measure health per se and consider twentieth century improvements in the quality of life associated with health[7].

The main reason for this shortage in health measurements is related to the difficulty in trying to gauge actual levels of health. Reported levels of morbidity have increased

[1] Maddison, "The World Economy: A Millennial Perspective", p. 29
[2] Nordhaus and Crafts are two of the few authors to make these considerations for mortality (as did Usher, but not to the same extent). Cutler & Richardson and Murray & Chen are the only authors to make considerations about morbidity that are directly related to this thesis.
[3] Fisherian growth is a notion that was coined by Nordhaus (1999) and is defined as the maximum amount that a nation can consume while ensuring that members of all future generations can have life time utility that is at least as high as that of current generations. When this yardstick is utilised, life expectancy is included in the production function and the value of improvements in mortality can be accounted, in terms of consumption. See Appendix 12.16 for methodological algebra.
[4] Nordhaus, "The Health of Nations: The Contribution of Improved Health to Living Standards"
[5] Crafts (2005) "The Contribution of Increased Life Expectancy to Growth of Living Standards in the UK, 1870-1998". Retrieved 17 June 2005, from: www.york.ac.uk/res/wpeg/documents/crafts.pdf
[6] Hickson, "The Contribution of Improved Life Expectancy to Standards of Living in Twentieth Century Japan", MSc Thesis (2002), London School of Economics and Political Science
[7] Cutler & Richardson have considered the improved quality of life associated with illness for the USA, during the last thirty years of the twentieth century. These considerations have not been made for England, and consequently this is one of the knowledge gaps that the thesis will fill.

throughout the twentieth century (in developed economies) for many reasons, some of which are not exclusively related to health (which will be explained in detail in Section 2.2). Moreover, health is an extremely subjective notion. These two factors have meant that, to-date the complications associated with measuring health are virtually insurmountable. Consequently, existing health measures are very specialised and limited, as they only achieve a predetermined narrow objective, usually for clinical outcomes research or policy investigations and predictions.

There have been no historical studies that aim to map the evolution of improving health from the perspective of quality of life for illness sufferers. To do this would require considerations about the shifting burden of disability, disease and death. It would also require deliberations about the contribution of improvements in medical technology, which have probably adversely affected the prevalence of disease, but have also reduced the negativities associated with ill health, and consequently provided an overall contribution to health related quality of life.

Furthermore, because of the dogmatic scope of existing health measures, very few have adopted a multifaceted stance and included as many variables as is desirable to fully consider health, as: *"not merely the absence of disease or infirmity, health is a state of complete physical, mental and social well-being"*[8].

This situation has inspired two key questions, which will be answered by the thesis:

1. What was the extent and value of improvements in health (mortality and morbidity) during different periods of the twentieth century in England?
2. What has been the impact of improved health upon standards of living and the overall health related welfare of the population in twentieth century England?

These questions will be answered throughout the remainder of the thesis. Chapter 2 will examine the theoretical, practical and historical context of the thesis. This will include a detailed investigation into the existing literature and theories related to health and health measurement and a detailed evaluation of England's health in the twentieth century.

[8] Preamble to the Constitution of the WHO as adopted as the definition of health since 1948: World Health Organisation (2005): "Constitution of the World Health Organisation". Retrieved 4 October 2005, from: http://w3.whosea.org/EN/Section898/Section1441.htm

Introduction

Chapter 3 will explain and justify the processes involved in the thesis for valuing health in the thesis. It will highlight how the qualitative illnesses chapters contribute to the quantitative results of the thesis. Chapter 3 will also provide a detailed explanation about the thesis (extended willingness to pay or Quality Adjusted Life Expectancy [QALE]) methodology. This will also entail an outline of how the QALE will be applied and the array of sensitivity analyses that will be included in the thesis methodology.

Part II of the thesis will present the qualitative analysis of the twentieth century health and welfare situations faced by sufferers of the thesis illnesses, namely, blindness, tuberculosis and cancer (breast and stomach). These chapters of the thesis will provide the analysis which bolsters the quantitative results, which will be presented in Part III of the thesis.

The final part of the thesis will provide the results which will contain the application of the QALE methodology, to the thesis illnesses, in order to derive estimates about the (monetary) value of improved health. This contains two broad sections: one which provides the summary qualitative results and their subsequent evolution into quantitative indices and one which presents the subsequent quantitative results. This part of the thesis will also contain a variety of sensitivity analysis approaches and simulation exercises, which will be employed to provide a range of QALE gain results for the three illnesses in different eras of the twentieth century[9]. Part III of the thesis is concluded with the Extended Results chapter, which develops earlier results in order to generate a lower bound estimate about the value of twentieth century health, in its entirety.

Finally, these results will be complemented by the thesis conclusions in Chapter 10, which will summarise the key questions of the thesis: What was the extent and value of improvements in health (mortality and morbidity) during different periods of the twentieth century in England? And, what has been the impact of improved health upon standards of living and the overall health related welfare of the population in twentieth century England? The thesis will conclude by defining the key contribution to knowledge of this analysis.

[9] Although there are actually four illnesses analysed in this thesis (namely: breast cancer, stomach cancer, blindness and tuberculosis) breast and stomach cancer will be considered in a combined chapter and therefore, in general, 3 types of illness will be considered.

2. Context

2.1 Theoretical

2.1.1 Historical Theories

The biggest debate that has surrounded considerations about improvements in mortality and morbidity in twentieth century England is related to the catalyst for these changes. Although much of this debate revolves around the time period that precedes that of the thesis and the results of the thesis do not contribute significantly to this debates, the key themes of this debate will be outlined to provide an indication of the state of health in England at the beginning of the twentieth century and the grand theories most associated with the thesis topic. The debate is also relevant for the thesis because it illustrates the influence of factors other than medical technology as important for early twentieth century health improvements.

2.1.1.1 Historical Theories / Debate about Health Improvements

The debate was initiated by McKeown (1976) in the middle of the twentieth century, who contemplated whether the reduction in mortality achieved in England and Wales since 1750 and particularly 1850, was primarily a by-product of improved standards of living, as manifested primarily in levels of nutritional intake, or whether it was attributed to technical improvements in the means of preventing premature death[10].

McKeown established the foundations of his theory through classifying the following major causes of disease:

1. *An autonomous change in the character of diseases. I.e. a decline in the virulence of the micro-organism itself*[11].

[10] In its fully developed form, McKeown originally presented a grandiose all-encompassing thesis accounting for Britain's demographic growth since the early eighteenth century, which included claims about the relationship between fertility and mortality as well as claims about the causes of declining mortality. The demographic aspects of McKeown's thesis will not be considered here as they are not important to the thesis.
[11] McKeown, "The Modern Rise of Population", p. 89

2. *An improvement in the overall environment so as to reduce the chances of initial exposure to potentially harmful organisms*[12]*. This could either be:*
    a. *Through prevention. I.e. as a result of scientific advances in immunisation techniques*
    b. *Through a reduction in the exposure to diseases. I.e. achieved with public health policy (municipal sanitation or hygiene improvements) designed to sanitise the urban environment.*
3. *An improvement in the human victims' defensive resources after initial exposure to hostile organisms*[13]*. This could occur either:*
    a. *Through treatment. I.e. the development of effective scientific and medical methods for treating symptoms*
    b. *Via an increase in the level and quality of the exposed population's average nutritional intake. I.e. better and more abundant food, thereby improving the individual's own natural defences.*

McKeown's strategy was then to consider the potential influence of each of these prospective causes, and systematically explain away factors other than nutrition (*3b*), as key for the mortality decline. Through a simplistic process of deduction (outlined below), McKeown arrived at his conclusions about the paramount importance of nutritional factors.

McKeown began by explaining away the potential for a change in the nature of diseases (*1*), through claiming that there was no evidence or consensus to suggest that there had been a change in the scientific configuration of disease and the virulence of micro-organisms. However others have claimed the contrary. For example, Magill has attributed the decline in deaths from pneumonia to a change in the nature of the disease[14]. And more generally, Griffith attributed the eighteenth century decline in mortality to medical advances, which changed the nature of disease[15].

McKeown continued by dismissing the environment and potential exposure (*2a* and *3a*) through his claims that: *"Certainly…no one…has sufficient therapeutic proof to argue that the drugs available at the turn of the century were sufficiently potent to have initiated the*

[12] McKeown, "The Modern Rise of Population", p. 91
[13] Ibid
[14] Griffith, "Population Problems of the Age of Malthus"
[15] Ibid

precipitous drop in mortality"[16]. McKeown offers further substance for this claim through a consideration about the timing of the introduction of therapy and the decline in major diseases: with the exception of smallpox and diphtheria, the dates at which either effective immunisation procedures or medical treatment became available were often far too late in time to be attributed to anything more than the final, marginal declines of these diseases[17].

This process leaves two possible causal factors: public health measures (*2b*) or nutritional factors (*3b*). McKeown overcomes this hurdle through considering the aetiology of the key diseases and grouping them into either *'water and food borne diseases'* or *'air borne diseases'*. The latter were much more significant for the mortality decline (as they were more infectious and subsequently rife). According to McKeown, *'air borne diseases'* could only have been reduced as a result of improved resistance in the population, which could only have been brought about through improved dietary status (*3b*).

Therefore, using this a priori argument, McKeown claimed that his data were able to show that the air borne category of disease was responsible for the major decline in death rates and that this constituted irrefutable evidence that improved nutrition had been the cause of the British mortality decline[18]. Furthermore, McKeown felt it legitimate to extrapolate these findings backwards (into the eighteenth century) and forward (into the later part of the twentieth century), on the seeming assumption that the mortality decline was a linear process across all three centuries[19].

Preston (1975) has identified one of the major inaccuracies of McKeown's assumptions through his considerations about the relationship between mortality and per capita income. McKeown's theory of nutritional developments implies a relatively unitary relationship between economic developments and improvements in mortality, through his suggestions that these improved health dimensions (embodied in improved nutrition) can be conceived as direct functions of changes in a country's per capita real income[20]. Preston has conducted an international cross-country study, which considers the relationship between mortality and income, and has concluded, *"It is difficult to devise a plausible model in which the rate of change of mortality is a direct function of the level of income"*[21].

[16] McKeown et al, "An Interpretation of the Decline of Mortality in England and Wales during the Twentieth Century", p. 410
[17] Ibid
[18] Szreter, "The Importance of Social Intervention in Britain's Mortality Decline c. 1850-1914: A Re-interpretation of the Role of Public Health", p. 10
[19] Ibid
[20] Ibid
[21] Preston, "The Changing Relationship between Mortality and Level of Economic Development", p. 233

Furthermore, in his study, Preston rules out nutrition (and literacy) as fundamental mortality change agents in favour of the major contribution of inoculation and vaccination[22].

Other authors have placed more importance upon literacy, female education and state programmes to promote mortality improvements. Caldwell (1979) and others have conducted a series of international and historical studies and concluded that personal health behaviour has been important in the prevention of infectious diseases, as has the pivotal role of government programmes in speeding mortality improvements[23].

Many of McKeown's critics have pointed out that while improved nutrition might be part of the story, McKeown's theory has given too little credit to public health measures and medicine[24]. One of the most famous proponents of this argument is Szreter. The period from the late 1830s to 1875 has come to be seen as encompassing a 'heroic age' of pioneering advances in public health activism and legislation, and the period thereafter was also marked by activity from local governments and municipalities[25]. This inspired numerous developments, for example, improved water systems and sanitation and a great reduction in 'water borne disease' mortality. Furthermore, the problems of housing quality and quantity were gradually being alleviated and there was an expansion of local health and maternity services and regulation of milk supply (which was recognised by McKeown)[26]. These developments helped to foster the later decline in infant mortality, which occurred during the first three decades of the twentieth century.

Therefore, the McKeown theory, which implies that the invisible hand of living standards conceived as an inevitable by-product of economic growth, facilitating an improvement in nutritional status, cannot take the leading role as the mechanism for Britain's mortality decline[27]. Although his thesis is valuable as it emphasises the limited role of medical technology before the twentieth century, which is often carelessly overlooked by historians, there are many inaccuracies and fundamental components that McKeown has disregarded in his dogmatic allegiance to the importance of nutritional factors[28]. Hence,

[22] Preston, "The Changing Relationship between Mortality and Level of Economic Development", p. 243
[23] Preston, "Population Studies of Mortality", p. 533
[24] Frank & Mustard, "The Determinants of Health from a Historical Perspective", p. 5
[25] Szreter, "The Importance of Social Intervention in Britain's Mortality Decline c. 1850-1914: A Re-interpretation of the Role of Public Health", p. 21
[26] Szreter, "The Importance of Social Intervention in Britain's Mortality Decline c. 1850-1914: A Re-interpretation of the Role of Public Health", p. 32
[27] Szreter, "The Importance of Social Intervention in Britain's Mortality Decline c. 1850-1914: A Re-interpretation of the Role of Public Health", p. 35
[28] McKeown, "A Sociological Approach to the History of Medicine", p. 342

public works, sanitation, improved housing and material standards of living, improved nutrition, medical and scientific developments all contributed to the decline in mortality.

2.1.1.2 Implications of Changes

The health transition began in Northwest Europe during the eighteenth century and was well under way by the middle of the nineteenth century, taking the form of a continuing, although not always consistent rise in life expectancy[29]. These increases continued into the twentieth century, which experienced some of the most impressive increases in life expectancy: from approximately 50 years at the beginning of the twentieth century to nearly 80 years by the close of the century. However, accompanying these improvements in mortality has been an increase in the population prevalence of chronic diseases and disabilities. This has occurred as a result of the dynamic between mortality and morbidity, which has often been referred to as the 'substitute morbidity and mortality' effects, defined as *"that disease and mortality which results from a decrease in another specific disease"*[30]. Hence, during the twentieth century, health has been gained on the one hand due to the reduction and elimination of one particular disease category (infectious), however this gain has been somewhat lost on the other hand because of other diseases replacing the original disease category (non-infectious, chronic).

The epidemiological character of twentieth century Britain has come to resemble a scenario of disease without death and consequently a seemingly increased burden of illness. Over the course of the century, the balance between death and disease has shifted towards a longer life but with proportionately more suffering and disabilities. These twentieth century changes in the burden of disease and death raise numerous questions about the standards of living associated with health.

2.1.2 Epidemiological Transition

2.1.2.1 What is the Epidemiological Transition?

In 1971, Abdel Omran detected an 'epidemiological transition', consisting of a passage from a regime in which there was a conversion of the pattern of mortality from one dominated by infectious diseases to one dominated by chronic degenerative diseases, this

[29] Riley, "Rising Life Expectancy: A Global History", p. 33
[30] Van de Water, "Health Expectancy and the Problem of Substitute Morbidity", p. 1820

also entails a decline in the death rates accompanied by a seemingly paradoxical increase in morbidity rates[31].

2.1.2.2 What Happened to Twentieth Century Mortality and Morbidity?

A major component of England's twentieth century epidemiological transition has been the decline in the death rate and the increase in life expectancy, which have both meant that there has been a radical increase in the number of people surviving to old age[32]. The increasing average age of the population is also reinforced by the change in the composition of fatal diseases, as degenerative diseases kill at much older ages than infectious disease, and therefore this transition in the causes of death is characterised by a general redistribution of deaths from young to older ages[33].

The epidemiological transition also describes the shift in the burden of illness, which has been experienced in twentieth century England. At the beginning of the twentieth century the major killers were infectious epidemics. By the end of the twentieth century the major killers were chronic degenerative illnesses. Hence cancer, heart disease and cardiovascular disease represented the main killers and the most prevalent illnesses, by a significant majority at the close of the twentieth century, which marks a stark contrast with the pattern of illness one hundred years earlier when tuberculosis and whooping cough dominated the mortality burden.

The link between ill health and death has a further dimension, which the epidemiological transition literature addresses. In the vast majority of the literature concerning public health and historical changes in the level of health, and even grand theories about the state of health, many have inaccurately used mortality as a proxy for morbidity. During certain situations this proxy is acceptable, for example, when many illnesses within the disease profile of a population cause death in a significant number of the people affected (usually at least five percent) and when this level of case fatality rates remains stable. Hence, in strict epidemiological terms, this is considering the relationship between mortality and the risk of falling sick. During eras before the twentieth century, when the clinical course of most sicknesses was brief (eight weeks or less) and most sicknesses were resolved quickly (by either recovery or death), this association was acceptable[34].

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[31] Omran, "The Epidemiological Transition: A Theory of the Epidemiology of Population Change"
[32] Riley, "The Risk of Being Sick: Morbidity Trends in Four Countries", p. 405
[33] Olshansky & Ault, "The Fourth Stage of the Epidemiological Transition: The Age of Delayed Degenerative Diseases", p. 356
[34] Riley, "The Risk of Being Sick: Morbidity Trends in Four Countries", p. 405

Throughout the twentieth century it became increasingly necessary to look further than death rates when considering changes in health and the quality of life associated with illness. This was a result of the inability of death rates to approximate sickness rates, which is most vividly illustrated by the following mechanisms. First, over time the case fatality rate may describe a different trend than the death rate[35]. Second, not all sicknesses pose a significant risk to death, which became increasingly true as the twentieth century unfolded. As a result of developments in health, the average duration of an illness has increased. For example, treatment of most chronic degenerative diseases does not cure these sicknesses they only ameliorate the symptoms and prolong the period of illness preceding death. The theory of the epidemiological transition implies a shift in the average duration of illness in general, rather than merely in diseases causing death, which is an increasingly important distinction for the time period being considered in the thesis.

A further advantage of considering the changing burden of illness through the framework of the epidemiological transition is for the important distinction it provides between the risks of falling sick, which is a measure of incidence and the risk of being sick, which is a measure of prevalence. By the onset of the twentieth century, previous developments in public health, sanitation, nutrition, and early medical advances had reduced the risk of falling sick and in turn increased the risk of being sick.

Hence, by the end of the twentieth century, most of the population survived to the boundary of entering old age, but much of the population survived with illness and injuries that occupied increasing time. From this perspective the epidemiological transition can be defined more broadly as a shift in the leading causes of death from acute to chronic diseases and therefore a shift from brief to protracted diseases. A society that has gone through such a transition gains additional life years, some in good health and some in poor health. This is the central trend that is reflected in the thesis, which will consider the value of these additional (disease burdened) life years, in terms of individual quality of life and health related welfare of the population as a whole.

2.1.2.3 Implication of the Epidemiological Transition in General

The situation and events which encompass the epidemiological transition (outlined above) combined with the shortage of empirical evidence depicting long term trends in chronic

[35] Case Fatality Rate = Refers to the proportion of persons with a particular disease who die. I.e. The number of persons dying due to a particular disease

disease has resulted in a wide range of opinions and theories about: the meaning of current trends in health, the outlook for future scenarios, and the general nature of the link between mortality and morbidity.

One of the most pessimistic theories to evolve from the epidemiological transition is the 'failure of success' hypothesis. Proponents of this theory, most notably, Kramer (1980) and Gruenberg (1977), have connected medical improvements (for example, the introduction of sulfa, penicillin, Aureomycin, Terramycin, etc) with the postponement of death, rather than a universal improvement in health. Consequently, instead of diminishing disease and enriching life, the twentieth century products of medical developments have served more to prolong disease and increase the proportion of the population suffering from disabling and chronic illnesses[36]. This is vividly highlighted through a consideration of the twentieth century changes in the prognosis of Down's syndrome. As a result of twentieth century (1940s) medical developments, the life expectancy of some individuals with Down's syndrome has been extended from early adolescence to seventy years. The net effect has been a doubling or quadrupling of the prevalence of Down's syndrome in the population[37]. These delay of death effects are also significant for numerous other disorders, for example, arteriosclerosis, schizophrenia, diabetes, spina-bifida, etc[38].

Hence, the key message of the 'failure of success' school is the claim that twentieth century scientific developments in medical care have only made achievements in life-saving technologies, rather than health preserving technologies, and that the net effect has been to worsen the population's health and furthermore, without a concentrated effort to search for preventable causes of chronic illnesses there will be no further enhancements in human health[39].

An alternative, optimistic perspective is the theory of 'dynamic equilibrium'. This optimistic view claims that in the long term, chronic illness will be confined to the last few years of life so that the proportion of healthy life will increase. Certain policy makers, for example, Marshal (1975) in the UK and Robine (1986) in the USA have adhered to this utopian opinion[40].

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[36] Gruenberg, "The Failure of Success", p. 5
[37] Murray & Chen, "Understanding Morbidity Change", p. 495
[38] Ibid
[39] Gruenberg, "The Failure of Success", p. 22
[40] Bebbington, "The Expectation of Life without Disability in England and Wales", p. 321

Along a similar vein is the theory of the 'compression of morbidity', coined by Fries (1989), which envisages a reduction in illness by postponing the age of onset of chronic infirmity relative to the average life duration, such that the period of morbidity is compressed between an increasing age of onset and a relatively fixed life expectancy[41]. This model requires the effects of preventative interventions to have a greater effect on morbidity than mortality and is also dependent upon the population adhering to favourable personal health habits[42].

A final theory, which entails the most optimism about future health trends, is the 'fourth stage' of the epidemiological transition hypothesis. Olshansky (1986) claims that the next phase in mortality and morbidity evolution will be marked by a decline in degenerative diseases. These predictions are bolstered by the improvements in chronic diseases that were achieved in America during the final decades of the twentieth century, for example, the decline in heart disease (by more than 20 percent between 1968 and 1978) and reductions in the death rates for cancer and strokes since the early 1970s[43].

Due to the nature of increased survival and the change in the burden of morbidity and causes of mortality, many of these aforementioned trends and theories are not illogical. However, to claim that health and standards of living associated with it have worsened, a la Gruenberg and Kramer, seems overly pessimistic. This will be justified by the thesis, through applying the thesis' original health evaluation methodology to twentieth century health in order to provide estimates about the value (direction and magnitude) of improved mortality and morbidity, which will enable the thesis to commentate on the plausibility of the above theories.

2.1.2.4 Implication of the Epidemiological Transition for the Thesis

The theory of the epidemiological transition is useful for highlighting the relationship between mortality and morbidity, although it does not extend to quality of life considerations. The thesis will consider the nature of the additional life years which have been fostered by the substantial and unprecedented twentieth century improvements in longevity.

[41] Fries, "The Compression of Morbidity: Near or Far", p. 208
[42] Feldman, "Work Ability of the Aged under Conditions of Improving Mortality", p. 441
[43] Olshansky & Ault, "The Fourth Stage of the Epidemiological Transition: The Age of Delayed Degenerative Diseases", p. 358

First, the increasing average age of the population (which is indicative of improvements in life expectancy and the death rate) was a result of the changing nature and declining virility of diseases. Infectious epidemics kill (typically children and young adults), whereas this is not the case for chronic degenerative diseases (which take their victims in old age). This has ramifications for the increase in life expectancy (mentioned above) and also for the increased proportion of healthy life years.

Second, the change in the case fatality rate has facilitated a more favourable relationship between the chances of contracting an illness and subsequently dying from that particular illness. At the onset of the twentieth century the case fatality rate was much higher, which essentially means that the chances of illness closely foregoing death were much higher. Along a similar vein, it will also be necessary to consider the changing dynamic between incidence and prevalence throughout the twentieth century.

Third, the general result of the epidemiological transition, which states that there has been a gain in life years enjoyed by the average individual but that not all of these additional life years are in good health will be evaluated by the thesis. This trend is undeniable, but the prospect of improving comfort in ill health, and consequently more valuable life years, even when overshadowed by morbidity, has been completely overlooked in the literature to date. The thesis will fill this void through considerations about the changing quality of life years associated with different illnesses during the twentieth century.

Lastly, the thesis will highlight the profound distinction between mortality and morbidity and through close attention to both and a detailed analysis will polarise the error of using death rates as a proxy for health, especially as the twentieth century unfolded.

2.1.3 Choice of Thesis Illnesses

The epidemiological transition provides the most descriptive and authentic theory about the trends in twentieth century mortality and morbidity and consequently provides the best framework for the selection of illnesses for consideration in the thesis. It is not possible to include all illnesses because of time constraints and also because of the changing classification of illnesses and other complications, which would make the task impossible. Therefore the thesis will consider illnesses that correlate with the principles of the epidemiological transition. I.e. the illnesses evaluated in the thesis will reflect the move from infectious epidemics (e.g. tuberculosis) to chronic degenerative diseases (e.g. breast

and stomach cancer). These illnesses will be considered qualitatively and quantitatively in order to generate numerical counterfactual estimates about the monetary value of improvements in health. Blindness will also be considered to provide a proxy for disabilities. The reasons for selecting these particular morbidity states will be outlined in Chapter 3: Section 3.4.

2.2 Practical

2.2.1 General Problems of Measuring Mortality and Morbidity

The classic literature (e.g. McKeown and Szreter) and the epidemiological transition theorists have highlighted the significant change in the twentieth century English mortality and morbidity burden and indicated the subsequent need to try and measure this evolution. However, there are numerous problems with the measurement of health, which are outlined below.

2.2.2 Epidemiological Transition Forces

2.2.2.1 Substitution Effect

The substitution effect is interlinked with the core of the epidemiological transition. The changing burden of disease, i.e. the move from infectious to chronic has meant that the average time of illness preceding death has increased significantly as degenerative and protracted illnesses have replaced acute illnesses. This has been further exacerbated by medical developments, which are increasingly able to prolong life in the diseased state but are unable to cure the disease. Pessimists identify this as an indication of worsening health, with their references to the prolonged average duration of certain diseases and disabilities, rather than producing cures[44].

[44] Gruenberg, "The Failure of Success", p. 5

Context: Practical

A more optimistic stance is taken by those who recognise that sicknesses are lasting longer as a result of improvements in medical technology, which means that they are resolved in disease management rather than death. The positive change in the nature of the key illnesses has meant that on the whole, diseases with a long course and low incidence have replaced illnesses with a short course and high incidence[45]. The longer duration of these long course diseases exceeds their lower incidence in its effect on sickness time in the population[46].

2.2.2.2 Composition Effect

In each successive period within an era of declining mortality, the population composition changes such that people who would have died earlier in the preceding regime are now living longer, and these 'new survivors' are, on average, more likely to experience health problems[47].

Therefore, by virtue of improvements in medical technology, more morbid prone individuals are being kept alive. Consequently (despite the improvement in their death rate) they are contributing to a 'failure of success' type increase in the mortality burden, as there was no cure for their morbidity (only for their potential mortality) throughout the twentieth century. This phenomenon became increasingly evident during the antibiotic era.

2.2.2.3 Time Effect

The claim of this theory is that there has been an increase in ill health as a result of improvements in survey and diagnostic methods. Hence, because diseases, especially chronic ones, are being detected earlier and the life expectancy with these diseases is increasing, the prevalence of these diseases has risen, albeit partially artificially.

Increasingly over time medical science has introduced a variety of means of surveillance and detection, and these techniques were applied to a rising proportion of the population. This meant that individuals were found to be sick sooner than they would have been without these screening and diagnostic innovations, and are adding to the prevalence of disease statistics, without a worsening per se in the general health level of the population[48]. This time effect is sometimes referred to as the 'early patients' phenomenon, as it depicts people being included

[45] Kumar, "Low Mortality and High Morbidity in Kerala Reconsidered", p. 117
[46] Ibid
[47] Verbrugge, "Longer Life but Worsening Health? Trends in Health and Mortality of Middle Aged and Older Persons", p. 515
[48] Morrison, "Screening and Chronic Disease"

in morbidity statistics earlier than they had traditionally been as a result of the improved ability to detect disease in earlier stages, e.g. hypertension, hypercholesterolemia, etc.

Therefore, although reported levels of illness have increased, this is for reasons not strictly related to health. The thesis measurement will be capable of illustrating health improvements that will be isolated from these seemingly perverse effects of the epidemiological transition.

2.2.3 Social Forces

2.2.3.1 Health Expectations

As economies develop and the population becomes wealthier and enjoys a general increase in their standards of living they also become more sensitive to health and adopt more stringent expectations about suitable levels of health. For example, at the end of the twentieth century, morbidity levels were highest in the wealthiest countries[49]. This positive correlation between national income and reported levels of morbidity provides a likely contribution to part of the rise in morbidity in twentieth century England.

2.2.3.2 Stock of Diseases

The above mentioned personal relaxing of the distinction between wellness and illness is also evident from physicians, as they also seem to have lowered the threshold of sickness[50]. As a result, conditions that would not have been regarded as health problems earlier are now recognised as such, e.g. depression and glandular fever. Although it is difficult to explicitly test and approximate the magnitude of this effect, there is clear historical evidence that shows a rising trend in the range of conditions regarded as ailments[51].

Additionally, as a result of medical developments, there is an increasingly recognised stock of diseases and diagnosable conditions, which creates the potential for physicians and patients to diagnose and register their ill health with a disorder. In this sense the incidence rate and overall morbidity in a population is a function of the (increasingly) recognised stock of diseases[52].

[49] Johansson, "The Health Transition: The Cultural Inflation of Morbidity During the Decline of Mortality", p. 40
[50] Riley, "The Risk of Being Sick: Morbidity Trends in Four Countries", p. 419
[51] Riley, "The Risk of Being Sick: Morbidity Trends in Four Countries", p. 419
[52] Johansson, "The Health Transition: The Cultural Inflation of Morbidity During the Decline of Mortality", p. 44

2.2.4 Economic Incentives

Some researchers have considered the effects of economic incentives for encouraging people to more willingly present themselves as sick. This is a product of the notion that labour force participation may become less attractive as a result of the provision of alternative sources of more easily obtained income, such as disability payments[53].

Wolfe and Haveman (1990) illustrate the correlation between the proportions of registered disabled people in America and the level of disability payments over a twenty-year period. In 1962: 7 percent, in 1973: 11 percent and in 1980: 9.5 percent of working age men were classified as disabled[54]. In combination with this trend, the generosity of disability welfare payments also display a 'hump shaped' pattern[55]. These inferences are also consistent with the findings of other authors who have suggested that perhaps one third of the rise in disability rates has been due to the generosity of disability transfer policy[56].

A further type of economic incentive that is potentially increasing registered levels of morbidity is the availability and accessibility of health service facilities. The use of healthcare services can increase morbidity, as the provision of subsidised healthcare is likely to provide an incentive to seek medical help earlier and more frequently. Consequently, ailments are diagnosed more frequently, rapidly and in some cases unnecessarily and this creates a pseudo worsening in the prevalence of disease[57].

Therefore these types of social forces all combine to alter the meaning of illness prevalence data. Hence, the same environment of mortality in 1900 would appear much more detrimental in 2000, because of the change in social attitudes towards illness and because of the magnitude of genuine health improvements. The problem for measuring historical levels of health that this poses is the impossibility of separating the influences of these social forces from actual changes in health. Consequently, a different angle for considering and measuring health needs to be employed in the thesis. This will be achieved through making considerations about the quality of life associated with these illnesses. For example, even though the prevalence of

[53] Riley, "The Risk of Being Sick: Morbidity Trends in Four Countries", p. 420
[54] Wolfe & Haveman, "Trends in the Prevalence of Work Disability from 1962 to 1984, and their Correlates", p. 59
[55] Ibid
[56] For example, Bailey, "Ageing and the Ability to Work: Policy Issues and Recent Trends", and Chirikos, "Accounting for the Historical Rise in Work Disability Prevalence"
[57] Certain passing ailments are being recognised and registered, whereas in earlier times these would have been resolved (in death or recovery) without medical attention and subsequent registration.

arthritis or diabetes has increased between 1900 and 2000, the burden of these illnesses was considerably lower by the close of the century, as a result of improvements in medical therapy which have increased the quality of life associated with these ailments. The benefit of this approach is that the above distortions are overcome.

2.2.5 Recent Studies that Measure Mortality and Morbidity

During the last quarter of the twentieth century there was mounting attention directed towards the concept of measuring health and evaluating the genuine changes in morbidity, as well as those for mortality. The majority of studies and measures of health have been conducted by two broad groups: social policy experts and health sector professionals. Both of these groups are dispersed between the public and private sector. Additionally, there is a peripheral and hybrid collection of professionals with an interest in considering the changes in health. Also on the margin are those who provide commentary about the authenticity of measurement techniques in general. The majority of these critics are economists and philosophers, who have an understanding about notions of utility and measurement.

Despite the significant and varied pool of potential health measurers, there is a dearth of efforts to consider the changes in health, gauge these in a quantitative manner and also to consider broader implications than just health. The table below provides a brief explanation of the most prominent approaches to measuring health, which are largely attributable to medical professionals and secondly, social policy experts.

Table 2.2.1: Summary of most prominent health measurement methodologies[58].

Author	Measure	Explanation	Type of Index/ Data
Bebbington (1988)[59] Policy expert England and Wales Applied study Objective measure	Expectation of Life without Disability (ELWD)	ELWD considers the number of individuals who have been reported as disabled. This information is then developed to provide an estimate of life expectancy without disability (in a similar manner to the formula which utilises death rates to establish life expectancy).	Statistics on the number of registered disabled (which are often ambiguous).
Bergner & Bobbitt (1981)[60] Health professional USA Applied study Subjective measure	Sickness Impact Profile (SIP)	The SIP contains 136 statements about the health related dysfunction in 12 areas of activity. The SIP is designed to be applicable to individuals and groups of diseases and disabilities in order to provide information about the sickness related dysfunction.	Questionnaire.
[Brody] (1985)[61] Health professional USA Theoretical study Subjective measure	Active Life Expectancy	Considers disability and the quality of extended life expectancy. Active Life Expectancy provides a measure that considers the prevalence of good and bad health.	Questionnaire.
Bush et al (1986)[62] Health professional USA Applied study Subjective measure	Quality Well Being (QWB)	Components of QWB: mobility, physical activity, social activity and symptom levels are derived through questioning patients.	Questionnaire.

[58] 1) The details below each author refer to the following; i) Field: considers the broad industry in which the author works, either health professional, policy expert or economist, ii) Country: considers where the study was implemented and / or the type of country specific data the study utilises, iii) Type of study: considers whether the measure is theoretical or whether it was actually applied in a survey, iv) Type of measure considers whether the study is 'objectively' observed by a professional (conducting the study or utilising data) or subjectively derived through questioning the ill. There is still scope for subjectivity between clinicians, and therefore these terms are relatively objective (relative to the subjective measures in the health literature).
2) When an author's name is depicted in brackets, for example, [Brody], it indicates that the measure being described was not introduced by this person, but that it has been better described, developed or implemented by the named author.

[59] Bebbington, "The Expectation of Life without Disability in England and Wales"

[60] Bergner & Bobbitt, "The Sickness Impact Profile: Development and Final Revision of a Health Status Measure"

[61] Brody, "Prospects for an Ageing Population"

[62] Balban & Sagi, "Weights for Scoring the Quality of Well Being Instruments among Rheumatoid Arthritics"

Context: Practical

Cutler & Richardson (1999)[63] Economists USA Applied study Subjective measure	Quality of life Weights	Identifies 10 illnesses that were consistently recorded between 1970 and 1990 and applies quality of life weights that are derived from responses to the National Health Interview Survey (NHIS)[64].	Registratios of disease and disability prevalence + NHIS questionnaire.
Erickson (1998)[65] Health professional USA Theoretical study Subjective measure	Health and Activity Limitation Index (HALex)	Considers perceived health and activity limitation to provide a single score about the quality of life related to illness. This is achieved through utilising a sample of NHIS responses combined with life expectancy data.	NHIS questionnaire + life tables.
Ghana Health Project Team (1981)[66] Health professionals Ghana Applied study Objective measure	Healthy days of life lost due to disease	Similar to Sullivan's methodology but related to specific diseases. They review 48 diseases and consider the number of healthy days of life lost due to each disease.	Census information on disease.
[Ho] (1980)[67] Policy expert (WHO) Not country specific Theoretical study Subjective measure	Functional Ability	Considers the superiority of measures of functional ability instead of symptomatic indicators of morbidity, for indicating the burden of different disabilities and diseases.	Questionnaire.
Hyder et al (1998)[68] Health professionals USA Theoretical study Subjective measure	Healthy Life Years (HeaLY)	The HeaLY measures the amount of healthy life lost due to morbidity and mortality, and early mortality due to morbidity. This can be calculated at the individual or population level. This measure is similar to that developed by the Ghana study team, although the HeaLY authors claim that their model is superior.	Census information on disease.

[63] Cutler & Richardson, "Your Money or Your Life: The Value of Health and what Affects It"
[64] The National Health Interview Survey (NHIS) is the National Centre for Health Statistics' (NCHS) annual, nationwide survey of about 36,000 households in the U.S. This is a principal source of information on the health of the civilian non-institutionalised population. It has been conducted every year since 1957. As such, it is one of the most significant sources of health data on the US population. The NHIS includes a set of core questions which change very infrequently, as well as a series of supplemental special topic questions that are modified from year-to-year in response to current interest and need for data. The core questions make up about 50 percent of the questionnaire and provide estimates of acute conditions, injuries, restriction of activity due to chronic conditions, respondent-assessed health status, and the use of medical services, including physician contacts and short-stay hospitalisation. The detail and versatility of this measure have continually improved since its introduction in 1957: National Health Interview Survey (2004). "About the National Health Interview Survey", Retrieved 10 January 2004, from http://www.chas.uchicago.edu/healthdata/national/nhis/
[65] Erickson, "Evaluation of a Population Based Measure of Quality of Life: A Health and Activity Limitation Index"
[66] Ghana Health Assessment Project Team, "A Quantitative Method of Assessing the Health Impact of Different Diseases in Less Developed Countries"
[67] Ho, "Measuring Health as a Component of Living Standards"
[68] Hyder & Rotland & Morrow, "Measuring the Burden of Disease: Healthy Life Years"

Context: Practical

Mehrez & Gafni (1991)[69] Economists Not country specific Theoretical study Subjective measure	Healthy Years Equivalents (HYE)	They market the HYE as a superior version of the QALY[70] because the HYE is (apparently) a better measure of utility. Many others have highlighted that the HYE is simply a TTO[71] QALY and therefore not superior[72].	Consumer preferences elicited through standard gamble questionnaire techniques.
Murray & Lopez (1996)[73] Health professionals and policy experts Global Applied study Objective measure	Disability Adjusted Life Year (DALY)	DALYs are weights for different illnesses. They are elicited through a series of revealed preference studies, which are conducted on a group of medical experts.	Medical expertise in conjunction with weight eliciting questionnaires.
Sullivan (1971)[74] Health professional USA Theoretical study Subjective measure	Disability Free Life Expectancy (DFLE)	Considers the number of life years in good health through using simple data (life tables and surveys). DFLE only provides effective results for long term, gradual changes[75].	Life tables + questionnaire.
[Wilson] (1981)[76] Health professional USA Theoretical study Subjective measure	Functional Ability + Life Expectancy	Wilson suggests that functional ability should be used in conjunction with life expectancy to provide a quality of life indicator. These claims are similar to considerations currently being made by the OECD and other organisations.	Life tables + questionnaire.

[69] Mehrez & Gafni, "The Healthy Years Equivalents: How to Measure them using the Standard Gamble Approach"
[70] QALY = Quality Adjusted Life Year, which is essentially a life year that has been adjusted for the burden of disease and disability.
[71] TTO = Time Trade Off, which is a methodology for eliciting revealed preferences about the perceived burden of a disability, which essentially requires the participant to trade off healthy life years for diseased and disabled ones.
[72] Cuyler & Wagstaff, "QALYs versus HYEs" and Bleichrodt, "QALYs and HYEs: Under what Conditions are they Equivalent?" and numerous others have also made this claim.
[73] Murray & Lopez, "The Global Burden of Disease Study"
[74] Bone, "International Efforts to Measure Health Expectancy"
[75] Mathers & Robine, "How Good is Sullivan's Method for Monitoring Changes in Population Health Expectancies?"
[76] Wilson, "Do Health Indicators Indicate Health?"

Context: Practical

The table above displays the two major approaches towards the measurement of health: subjective evidence, which considers how the participants feel and perceive their health burden, (functional ability, the SIP, results of the NHIS) and behavioural evidence, which considers health through reviewing what has often been referred to as 'the 5Ds' (death, disease, disability, discomfort, dissatisfaction), which are reflected through rates of absenteeism, confinement and seeking medical care (for example, The Ghana study, The Global Burden of Disease study and also measures like the ELWD and HeaLY indices) [77].

The most common and increasingly popular method of assessing health is through subjective evidence about an individual's perceived ability to perform tasks of daily living, expressed through responses to some form of questionnaire. It is generally agreed that these types of consideration about functional ability in conjunction with life expectancy provides the most proficient approach for indicating improvements in health[78].

Despite the popularity of functional ability, there was still a broad range of health measurement methodologies and results. One of the reasons for this is the difficulty involved in defining and measuring morbidity. Additionally, there exists inconsistency among theorists about what the most important proxy variables are concerning health[79]. Numerous authors have indicated that the variables that are selected, and the manner in which the health questions are conveyed, exert a major influence upon the results obtained[80]. Another problem, related to the study design, is the unreliability of data collection as a result of morbidity rates being variable in quality.

Further weaknesses of the questionnaire approach are a consequence of the problems with the respondents, e.g. symptoms do not always directly reflect the actual disease[81], the subjective reporting of morbidity is influenced by cultural and individual differences in attitudes towards

[77] Balinsky & Berger, "A Review of Research on General Health Status Indexes", p. 286
[78] Numerous authors have stressed the desirability of creating a health measure that combines some proxy for morbidity with life expectancy, for example; Balinsky & Berger, Wilson, Bone, Bowling, etc.
[79] As Table 2 indicates, there are numerous health measures that consider different aspects of disease. This is partly because there is no agreement or proof about the most indicative proxies to question and also about the most efficient methodology for framing the questions and therefore this debate still remains unresolved. This debate has no crucial relevance for the thesis and therefore it will not be visited in any greater detail than what is provided here.
[80] Llewellyn-Thomas, "Describing Health States: Methodological Issues in Obtaining Values for Health States", p. 550
[81] Ho, "Measuring Health as a Component of Living Standards", p. 10

health, and accounting for handicap at the personal level or the significance of functional disability are not catered for in the majority of these questionnaires[82].

The diversity between the results that are yielded from observed (e.g. clinical studies) and self reported (e.g. functional ability, questionnaire studies) studies implies that self-perceived morbidity and observed morbidity may be measuring different aspects of illness[83]. When considering the authenticity of clinical studies of morbidity, it is worrying to note the wide variation in the diagnostic skills of clinicians. Elinson and Trussel (1957) compared clinical diagnoses by two physicians in the same sample of patients and identified marked differences in their diagnoses[84].

In addition to these general weaknesses in the above studies there are also numerous specific weaknesses for the demands of the thesis.

2.2.6 Recent Studies: Mortality and Morbidity Measurement Weaknesses for the Thesis

There are two fundamental shortcomings of the aforementioned measures for the requirements of the thesis: the lack of historical content and the failure of these measures to explicitly connect the burden of morbidity with the quality of life of illness sufferers.

2.2.6.1 Lack of Historical Content

Virtually all existing approaches to monitoring health are redundant when trying to evaluate health from a historical perspective. Self-perceived measurement techniques consist of asking or observing present-day sufferers about how they perceive the burden of their diseases and/or disabilities. Observed measures of mortality require a physician to monitor and evaluate the burden of illnesses upon contemporary sufferers. As a result of the historical nature of the thesis it will be impossible to estimate morbidity through interviewing or observing contemporary sufferers. And, because efforts to measure health only became commonplace towards the end of the twentieth century, there are no existing historical estimates of the morbidity burden for any earlier eras.

[82] Ibid
[83] Murray & Chen, "Understanding Morbidity Change", p. 490
[84] Elinson & Trussel, "Some Factors Relating to Degree of Correspondence for Diagnostic Information as Obtained by Household Interviews and Clinical Examinations"

Context: Practical

2.2.6.2 Void of Quality of Life Concerns

The major focus of existing health measures is typically on the effects of different illnesses (and the efficacy of therapies for these illnesses) in medical terms or for their implications upon the economy in policy terms. These measures are significantly lacking in scope for reflecting the implications of the changing burden of morbidity upon quality of life for the individual and standards of living of the population in general.

Moreover, there is currently a significant void in the literature that considers the changing twentieth century burden of morbidity, and none of these studies have attempted to make quantitative estimates about the value of this changing morbidity burden, for the quality of life of the English population during the twentieth century. As a result existing measures are unsuitable for the demands of the thesis.

2.2.7 Conceptual Requisites of an Ideal Historical, Mortality and Morbidity, Quality of Life Measure

2.2.7.1 Conceptual Requisites of an Ideal Measurement

The form of measurement that the thesis requires is one that accords with the requisites for a representational (as defined by representational theory of measurement) and reliable measure.

2.2.7.2 Representational Measurement

The core and virtue of this theory is that measurement is a process of assigning numbers to objects in such a way that the relevant qualitative empirical relations among the objects are reflected in the numbers themselves as well as in important properties of the number system. This will be achieved through the measurement instrument of the thesis aspiring to accord with the basic criteria of representational measurement[85]:

1. *The measurement will be 'accurately indicative' through precisely representing the relative quantities of the variables being measured (e.g. EuroQol component) as well as indicating the relations between the variables (e.g. between different EuroQol components)*[86].

[85] Points 1 to 4 have been developed from Boumans, "Representation and Stability in Testing and Measuring Rational Expectations", p. 383

[86] EuroQol depicts the foundation matrix of the thesis measure, i.e. it is from the EuroQol matrix that the Quality Adjusted Life Year (QALY) will be derived and ultimately the Quality Adjusted Life Expectancy (QALE) will be determined. The EuroQol contains the template and framework of elements that will be considered collectively to form the basis of the health related quality of life thesis measure. Please see Chapter 3: Methodology for further explanations about the EuroQol matrix.

Context: Practical

2. *The desirability of consistency in the measurement necessitates that the approach to measurement is maintained throughout the thesis, for different times and health states. For example, whether measuring the health burden of breast cancer in 1900, tuberculosis in 1950 or blindness in 2000, the EuroQol matrix will be designed and implemented to yield consistent measurements of the QALY, which will ultimately provide a consistent estimate about the quality of life related to health across time (twentieth century) and space (different diseases and disabilities).*
3. *Sensitivity in measurement in order to accurately and entirely gauge changes in the objects of measurement. For the demands of the thesis the sensitivity demands are:*
    i. *To identify marginal and tacit changes in the burden of illness.*
    ii. *To better achieve the ceteris neglectibus condition (see below for details) through minimising the effects of 'other circumstances' (OC) not associated with health related quality of life[87]. For example, worsening in the reported health of the population as the twentieth century unfolded as a result of changes in health expectations, the epidemiological transition and improved medical technology will be minimised through adhering to the requisites of sensitivity.*
4. *In order to achieve the above criteria of representational measurement there is a need for a steadfast scale of measurement.*

As a result of the need to measure *quantity* (in this respect the quantity of health and quality of life related to health), there is an order relation system which enables the measurer to order quantity in a way which has formal similarity to the relations equal, greater and lesser[88], which provides the basis for developing a scale of measurement[89].

Measurement scales can be formed in two ways[90]:

1. Direct mapping (from a quality relational system to a numerical relational system).
2. Indirect mapping (through a relation to the quality to be measured and other qualities, for which measurement scales have been defined, because it is impossible to set up a satisfactory measurement scale directly).

[87] See Equation 1 below for definition of OC.
[88] Finkelstein, "Theory and Philosophy of Measurement", p. 12
[89] Ibid
[90] Finkelstein, "Theory and Philosophy of Measurement", p. 15

Context: Practical

The thesis utilises the second type of measurement. Quality of life will be measured through identifying a QALY as a result of the impossibility of directly gauging health related quality of life. The worked examples below provide a further explanation about the mechanics of indirect and representational measurement.

In this measurement framework, the representative acts as an instrument for measuring attribute X. The table below highlights two examples of the relationship between components in an ideal representative measure.

Table 2.2.2: Comparison of thermometer and thesis QALE components of representational measurement[91]

Components of Representational Measurement	**Example 1: Temperature**	**Example 2: Quality of Life (QoL)**
Instrument:	Thermometer	EuroQol Index
Attribute X:	Temperature	Quality of life (QoL)
Correlate of attribute X:	Height of mercury	QALY index (0 → 1)
Function:	T = *f* (h) T = temperature, h = height of mercury	QoL = *f* (QALY)

In example 1: the thermometer is the representative of temperature in which the height of the mercury column correlates with the temperature. The formal analogy of that correlation is the function between height and temperature, T= *f* (h).

In example 2: the EuroQol index is the representative of quality of life in which the value of the QALY index correlates with the overall QoL. The formal analogy of that correlation is the function between the QALY and the quality of life, QoL = *f* (QALY).

[91] Referenced and developed from Chang, "Spirit, Air and Quicksilver: The Search for the Real Scale of Temperature"

In both of these examples, the only restriction which must be placed upon *f* is that T and QoL must be defined for every value which h and QALY may assume, and the numerical order of the temperature and QoL numbers must correspond to the temperature and QoL order.

2.2.7.3 Reliability of an ideal measure

When trying to develop an indirect measurement tool in the form of representational theory there are numerous conditions that need to be achieved. These conditions are further complicated when the measurement and the tool developed to gauge it are economical (e.g. QALY and QoL) rather than physiological (e.g. thermometer). When conceptualising an ideal measurement scale for assessing health related quality of life the initial objective is to achieve standardisation and satisfy the condition of minimalist over determination.

Standardisation is achieved through accuracy and stable correlations in order to minimise the influence of 'other circumstances' (OC) not associated with health related quality of life[92]. This has been adhered to in the development of the thesis methodology through the selection of the most precise EuroQol variables.

Chang (2001) has developed a procedure of minimalist over determination, which is designed to identify the best measure under conditions of uncertainty, i.e. non ceteris paribus environments (which were derived from the history of the development of a standard thermometer in the nineteenth century)[93]. In order to achieve the conditions of minimalist over determination Chang outlined the following process:

> *Build a series of instruments that are each based on a different but very minimal and consistent set of theoretical assumptions, and test these instruments under different circumstances. The instrument for which the results are most coherent, under these different circumstances is the instrument which can be chosen to be the standard measuring device*[94].

According to Chang, the point of this history of the thermometer is that for endorsing the air thermometer as the best standard, Regnault did not need to prove that the expansion of air is

[92] See Equation 1 below for definition of OC
[93] Chang, "Spirit, Air and Quicksilver: The Search for the Real Scale of Temperature"
[94] Ibid

uniform. Through minimalist over determination, the air thermometer was the only thing left as all other types of thermometer failed to meet the necessary, minimalist conditions of consistency. Hence, the art of practicing this strategy (of minimalist over determination) lies in the ability to contrive over determined situations on the basis of as little as possible.

Minimalist over determination has been achieved in the thesis through selecting the most indicative variables and versatile methodologies for measuring historical health related quality of life.

When applying over determination to history or economics, the process becomes more complicated as a result of the inability to create a stable environment under which to compare different measuring instruments. Accuracy is achieved through adhering to the 'ceteris neglectibus' condition[95].

The ceteris neglectibus condition for example 2 (QoL) is shown below[96]:

$$\nabla QALY = \nabla QALY(I; QoL, OC) = \frac{\partial QALY(I)}{\partial QoL}\nabla QALE + \frac{\partial QALY(I)}{\partial OC}\nabla OC \qquad (1)$$

where (I) indicates the components of the QALY. The influence of other circumstances (OC), besides quality of life (QoL), on the QALY index QALY (I) is denoted by the partial derivative:

$$\frac{\partial QALY(I)}{\partial OC} \approx 0$$

Equation (1) clarifies the key components that need to be considered when trying to construct the ideal measure. The foundations of the health measure that has been developed as part of this thesis are more secure as they have been constructed to satisfy the criteria of a good measuring tool. Subsequently, the thesis health measure is capable of measuring the phenomenon associated with mortality and morbidity most accurately (and minimise the effect

[95] Boumans, "Representation and Stability in Testing and Measuring Rational Expectations", p. 386
[96] All formulae is referenced and developed from Boumans, "Representation and Stability in Testing and Measuring Rational Expectations", p. 386

of OC), in order to adhere (as closely as possible) to the criteria of an ideal representational measurement (namely: accurately indicative, consistent, sensitive and scaled), through achieving standardisation and minimalist over determination and subsequently satisfying the ceteris neglectibus condition when measuring health over space (different illnesses) and time (different eras of the twentieth century).

2.2.7.4 Ideal Measure in Conjunction with the Demands of the Thesis

The difficulty in achieving this objective (of an ideal general economic measure) is further complicated when combined with the difficulties of defining and measuring health in general and specifically in conjunction with the historical measurement of quality of life associated with health considerations contained in the thesis.

A major part of the thesis will entail the development of a morbidity (and to a lesser extent, mortality) measuring tool that is coherently constructed, statistically reliable, sensitive, applicable and valid. Finally, it will be capable of providing estimates about the value and contribution of improved health to standards of living in twentieth century England, which is to date lacking. The construction of this new measure, namely, the QALE (quality adjusted life expectancy) is explained in Chapter 3.

2.2.8 Hickson QALE

As a result of the aforementioned difficulties and shortcomings associated with existing health measurement methodologies, in conjunction with the original, historical and quality of life considerations entailed in the thesis, a new health measure will have to be constructed.

This new health measure will be capable of simultaneously considering the value of:

1. Improvements in the quality of life associated with morbidity (from a health and welfare perspective) at different points during the twentieth century, and
2. Increases in life expectancy at different points during the twentieth century.

This will enable the thesis to identify the improvements in the quality of life associated with morbidity and mortality experienced by the English population during the twentieth century. Once these developments have been highlighted the thesis will endeavour to quantify the extent and impact of these improvements (in qualitative and quantitative terms) upon

standards of living and for their contribution towards an extended ‘Fisherian’ notion of economic growth.

2.3 Historical

In order to fully establish the historiography underlying this epidemiological transition and the general motivation for the thesis this chapter will answer the central underlying question: what happened to the health of the English population during the twentieth century? As well as considering the change in life expectancy during the twentieth century, it will also be necessary to examine the shift in the burden of diseases, particularly as some of the most important improvements in health were a product of the changing pattern of morbidity[97].

During the twentieth century England experienced unparalleled improvements in health. Life expectancy improved from 47 years in 1900 to nearly 80 years by 2000[98]. The burden of diseases experienced a remarkable transformation as it shifted from infectious illnesses (accounting for 25 percent of deaths in 1911 and 0.5 percent in 1991[99]), such as tuberculosis to chronic degenerative disorders (accounting for over 80 percent of deaths by 1991[100]), such as cardiovascular disease and cancer.

Surrounding this change in the health profile are numerous debates and issues. The most important for the thesis will be considered. These include the debates about the effect of the Great Depression and World Wars on health, which are some of the most frequently visited

[97] The World Health Organisation's wider definition of health (1979) will be adopted as the definition of health throughout the thesis: health is a state of complete physical, social and mental well being and not merely the absence of disease: World Health Organisation (2005). "Constitution of the World Health Organisation". Retrieved 4 October 2005, from: http://w3.whosea.org/EN/Section898/Section1441.htm

[98] 1900: Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 44 and 2000: WHO Core Health Indicators, www.who.org (LE 2000 = 77 years): World Health Organisation (2004), "Core Health Indicators". Retrieved 24 February 2004, from: http://www3.who.int/whosis/core/core_select.htm

[99] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 44

[100] Ibid

arguments when considering English health in the twentieth century. Also, health inequalities and the associated shortcomings with the NHS have received consistent attention throughout the twentieth century for a variety of reasons, for example, in the 1930s as an adversity of the Great Depression and in the 1980s as a concern about the performance of the National Health Service, and therefore need to be analysed by the thesis.

2.3.1 Infant Health

Many of the improvements in standards of nutrition, hygiene and living in general were manifest in the death rate of 'certain diseases of infancy', which began to decline steadily after 1901[101]. By the early twentieth century gastrointestinal infections, such as cholera, enteric fever and dysentery had been controlled. This was particularly attributed to the purification of water supplies[102]. The incidence of smallpox dropped to negligible proportion during the first decade of the twentieth century[103]. A similar trend was evident for scarlet fever and whooping cough (which declined most impressively between 1870 and 1900)[104]. These observations are confirmed by reviewing the infant mortality rate (IMR) time series.

[101] Gage, "The Decline of Mortality in England and Wales 1861-1964: Decomposition by Cause of Death and Component Mortality", p. 57
[102] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 1
[103] Fraser, "A History of English Public Health", p. 285
[104] Mercer, "Relative Trends in Mortality from Related Respiratory and Airborne Infectious Diseases", p. 141

Context: Historical

Table 2.3.1: Infant mortality rates (per 1,000) and indexed infant mortality rates (150=100), 1890s-2000[105].

Period	**IMR (per 1,000)**	**Indexed IMR** (150=100)
1890s	150	100
1901-1905	138	92
1911-1915	110	73
1916-1920	90	60
1931-1935	62	41
1936-1940	55	37
1941-1945	50	33
1951-1955	27	18
1965	19	13
1980	12	8
2000	6	4

Table 2.3.1 considers the improvement in the infant mortality rate between 1890 and 2000. This is presented in terms of the actual infant mortality rate and also as an index in order to more vividly highlight the substantial improvements in infant mortality.

By the last period in the above table the infant mortality rate had declined to about 4 percent of the level it was at in the initial period. In addition to improved sanitation and living conditions numerous developments in immunisation against many of the most fatal diseases of infancy facilitated the decline. The table below provides the chronology of such developments, with the most pertinent vaccine introductions highlighted.

[105] (Rows 1-4 and 6): Fraser, "A History of English Public Health", p. 355. (Rows 5 and 7-9): Parish, "Victory with Vaccines: The Story of Immunization", p. 218. (Row 10): National Statistics (2006), "Infant Mortality Rate, 1975-2000". Retrieved 7 March 2006, from: http://www.statistics.gov.uk/STATBASE/xsdataset.asp?More=Y&vlnk=4288&All=Y&B2.x=108&B2.y=7 (Row 11): WHO Core Health Indicators: World Health Organisation on (2004), "Core Health Indicators". Retrieved 24 February 2004, from: http://www3.who.int/whosis/core/core_select.htm

Table 2.3.2: Chronology of the introduction of major vaccines[106]

Disease / Viruses	**Year of vaccine introduction**	**Efficacy of vaccine**
Smallpox	1796	Effective for 10-20 years
Rabies	1885	Effective
Yellow Fever	1936	Effective
Influenza	1943	Temporary and partial
Polio	1954, 1957	Effective
Measles	1963	Effective
Mumps	1968	Effective
Rubella	1969	Effective
Hepatitis B	1970	-
Disease / Bacteria	**Year of vaccine introduction**	**Efficacy of vaccine**
Cholera	1884	Possible short run immunity
Tuberculosis	1890	Only effective for diagnosis
(BCG)[107]	1906-1921	Often effective for children
Diphtheria	1890	Unresolved
Tetanus	1890	Effective
Plague	1895	Temporary and partial
Scarlet Fever	1907	Ineffective
Whooping Cough	1933	Partial effectiveness
Pneumonia	1945	Effective
Bacterial Meningitis	1998	Effective (expected)

As a result of developments in sanitation, hygiene, nutrition and to a lesser extent (in many cases), scientific developments in the arena of immunisation; diphtheria, tetanus and whooping cough had largely been eliminated by the middle of the twentieth century, which caused a significant improvement to infant survival probabilities. Polio, measles, mumps and rubella were virtually eliminated during the second half of the twentieth century, when the necessary vaccines were discovered and generally available. These breakthroughs are

[106] Riley, "Rising Life Expectancy: A Global History", p. 99

[107] The BCG vaccine protects against TB, usually in infants. The letters B, C and G stand for; B = Bacillus which describes the shape of the bacterium, C = Calmette and G = Guerin, the names of the people who developed the vaccine: World Health Organisation (2005), "BCG-The Current Vaccine for Tuberculosis". Retrieved 12 March 2005, from: http://www.who.int/vaccine_research/diseases/tb/vaccine_development/bcg/en/

highlighted in Table 2.3.2. These improvements resulted in an infant mortality rate that had reached a remarkable 6 per 1,000 by the year 2000[108].

Despite these staggering declines in infectious diseases, it might be possible to claim that developments were postponed because of the discrepancy between the introduction of vaccines for some of the major killers and the introduction of 'publicly notifiable' legislation for the corresponding diseases.

Table 2.3.3: Chronology of notifiable infectious diseases in England, 1889-1988[109]

Year disease became notifiable	**Notifiable infectious disease**
1889	Cholera, Smallpox, Typhus, Diphtheria, Scarlet Fever, Typhoid Fever
1912	Polio, Tuberculosis
1914	Ophthalmia Neonatorum
1918	Acute Encephalitis
1919	Dysentery, Malaria
1940	Measles, Whooping Cough
1951	Leprosy
1960	Anthrax
1968	Meningitis, Viral Hepatitis, Yellow Fever, Tetanus
1976	Rabies, Viral Haemorrhagic Fever
1988	Meningococcal Septicaemia, Mumps, Rubella

If Table 2.3.2 and Table 2.3.3 are jointly considered it becomes clear that there is little relationship between notification and immunisation breakthroughs for these illnesses. In the majority of cases in the two tables, public notification legislation was introduced a long while after the introduction of the vaccine. For example, smallpox inoculation was introduced in 1796 and not notifiable until 1889, a ninety three year lag.

[108] WHO Core Health Indicators: World Health Organisation (2004), "Core Health Indicators". Retrieved 24 February 2004, from: http://www3.who.int/whosis/core/core_select.htm
[109] Ibid

In contrast, polio, measles and scarlet fever became notifiable diseases before the discovery of the necessary vaccinations, and therefore adopt a more logical and expected pattern. I.e. one would have thought that once a disease is recognised as a public health hazard increased efforts are dedicated to the initiatives necessary to combat the disease. When all of the twentieth century notifiable diseases are considered in conjunction with their corresponding vaccine history, there is very little, if any correlation. It seems reasonable to conclude that there is minimal interaction between these two features of public health. Moreover, it is also noteworthy that the reduction of many of these diseases was accomplished before the discovery of their corresponding immunisation. This highlights the importance of improvements in public health for the decline in many of the major diseases, for example, smallpox, scarlet fever and whooping cough.

2.3.2 Adult Health

Between 1900 and 1950 the trend of improved survival prospects through a decline in the prevalence of infectious diseases was distributed fairly evenly across all age groups. Since 1950 ages 0-1 and 15-44 have experienced the sharpest declines while the opposite applies to the oldest age groups, although it is noteworthy that, in 1911 the oldest age groups had the lowest incidence of infectious disease mortality[110]. The graphs below provide an illustration of the twentieth century health transformation, in support of the epidemiological transition. Figure 2.3.1 contains all seventeen international classification of diseases (ICD) categories and Figure 2.3.2 details the five most notable disease categories in order to highlight the key trends of twentieth century health.

[110] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 45

Context: Historical

Figure 2.3.1: Percentage of deaths in each International Classification of Disease (ICD) category 1911-1991[111*].

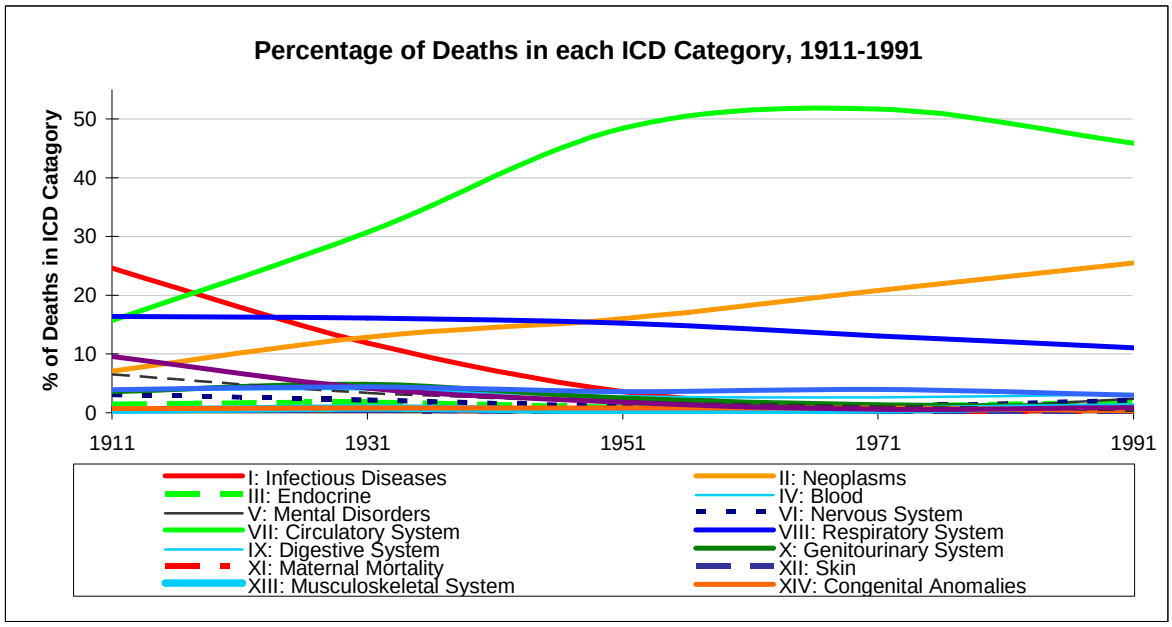
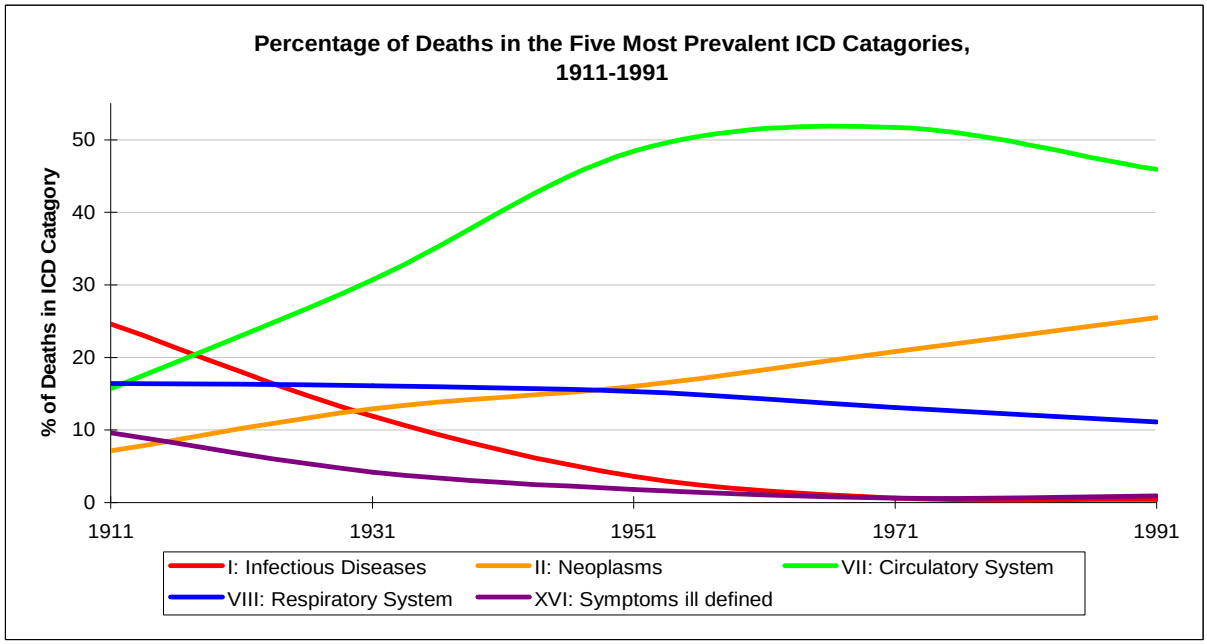


Figure 2.3.2: Percentage of deaths in the five most prevalent International Classification of Disease (ICD) categories, 1911-1991[112].



[111] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 44

[*] Note: In order to make comparisons over time it is necessary to bridge the various coding systems that have been used (from ICD1 in 1901 to ICD9 since 1979). The statistics used here, from Charlton & Murphy have converted historical data to their ICD9 equivalents.

[112] Ibid

2.3.2.1 Infectious Disease Decline

The infectious disease category contains numerous diseases. The six diseases that were especially significant for the decline in deaths from infectious diseases are listed below:

1. Tuberculosis
2. Diphtheria
3. Whooping Cough / Pertussis
4. Measles, Mumps, Rubella
5. Influenza
6. Communicable diseases (including STDs)

Although tuberculosis peaked in incidence before the twentieth century, it persisted to be a health burden until about 1950. Improved ventilation and host resistance helped to reduce the virulence of tuberculosis. In contrast to this 'McKeown type argument', others have claimed that the reduction in other infectious diseases was more important in abating tuberculosis because this reduced the impact of a trigger disease, in this case smallpox[113]. Although there is some accuracy in this claim, as tuberculosis is recognised as a sequel to smallpox, it probably only accounts for about 20 to 30 percent of tuberculosis deaths[114]. The most important factor in the reduction of tuberculosis was the introduction of specific chemotherapy in the 1950s[115]. This facilitated a rapid decline and virtual elimination of tuberculosis because the new drugs quickly rendered patients non-infectious and thus swiftly controlled a potential source of infection for further cases[116].

Diphtheria was a major contributor to childhood mortality from the 1890s until the 1940s, when successful immunisation was introduced. By the late twentieth century, the disease had almost disappeared[117]. E.g. during the 1980s only 30 cases were reported and many of them had been imported[118].

Whooping cough has a very long role in the health history of England. Deaths remained at about 10,000 per year from the 1840s until 1910. By 1950 improved host resistance facilitated

[113] Mercer, "Relative Trends in Mortality from Related Respiratory and Airborne Infectious Diseases", p. 145
[114] Ibid
[115] Davies, "The Pharmaceutical Industry – A Personal Study", p. 197
[116] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 8
[117] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 13
[118] Ibid

a fall in the mortality rate, to less than 400 per annum[119]. Widespread immunisation, which began in the 1950s, facilitated a continuation and enhancement in this downward trend, although public health measures ought to be attributed to the decline in whooping cough as this had largely been achieved before the introduction of widespread immunisation. However, the decline in whooping cough was interrupted between 1974 and 1994, when there were fears that immunisation was linked to encephalopathy and vaccination levels fell to 30 percent[120]. This link was disproved in 1985, and by 1994 vaccination levels had returned to nearly complete coverage (93 percent) of the infant population[121].

The decline in measles, mumps and rubella is more attributable to medical technology, as the prevalence of these diseases declined rapidly when immunisation became widespread in the late 1960s[122]. Notification fell by about two thirds as a result of the introduction of the vaccines, to a level of about 90,000 reported cases per year in the 1980s[123]. When the MMR vaccine was introduced in 1988 further declines in mortality from these diseases were facilitated such that the 1990s annual notification fell to about 9,000 cases per annum[124].

There have been three influenza pandemics during the twentieth century: 1968, 1947, and most notably 1918-19, which was of unprecedented scale and particularly detrimental as it mainly affected young adults[125].

Prior to the establishment of special VD clinics and the introduction of penicillin, STDs were responsible for considerable mortality (syphilis) and morbidity (gonorrhoea)[126]. Despite inflation in the number of reported STDs during the 1960s and 1970s, which is likely to be caused by changes in population trends of sexual behaviour, by the close of the twentieth century most STDs were under control. For example, the number of deaths from syphilis declined from 4,375 in 1910 to 18 in 1990[127]. Much of the concern regarding STDs in the late

[119] Ibid
[120] Ibid
[121] Ibid
[122] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 21
[123] Ibid
[124] Ibid
[125] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 10
[126] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 21
[127] Ibid

twentieth century had centred on Chlamydia, Herpes simplex and HIV/AIDS, for which the number of reported cases increased every year since the disease was recognised in 1982[128].

2.3.2.2 Respiratory Diseases

The decline in infectious diseases was the most important classification of mortality reduction in twentieth century England. Second in importance were respiratory diseases. When considering diseases of the respiratory system it is necessary to differentiate between those which are infectious and non-infectious, as the reduction in infectious diseases becomes more pronounced.

The decline of mortality from infectious respiratory illnesses (e.g. influenza, bronchitis, pneumonia, etc) began in the early twentieth century and was not especially aided with the advent of antibiotics or the National Health Service although both are thought to be conceivable explanations for the continuing downward trend in respiratory related deaths[129]. E.g. the introduction of antimicrobial chemotherapy accelerated the decline of pneumonia in the 1930s[130].

Decreases in respiratory disease mortality pre 1950 were largely a result of the reduction in infectious respiratory diseases. After 1950 two forms of respiratory disease related to smoking became the most prevalent: lung cancer and chronic obstructive pulmonary disease (COPD)[131]. By the year 2000 lung cancer was the most important respiratory cause of death.

Because this ICD category contains infectious diseases, e.g. pneumonia, as well as diseases that reflect long term damage to the lungs, e.g. asthma, there was a mixture of age related trends during the twentieth century[132]. For ages below 65, mortality has been falling, especially for ages 0-14 between the 1940s and 1950s and again from the late 1960s onwards, this trend was also evident for ages 15-24, but to a lesser extent[133]. In ages 25-44 rates fell, especially in the post war period, but have been rising since 1980 for men and static for

[128] Ibid
[129] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p.94
[130] Ibid
[131] Ibid
[132] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 49
[133] Ibid

women[134]. There has been a fall in the oldest ages over the twentieth century as a whole, although the pattern is more volatile as these age groups are particularly prone to flu epidemics.

2.3.2.3 Degenerative Disease Increase

The increase in degenerative diseases is related to longer, richer lifestyle characteristics, which tend to be correlated with unhealthy lifestyle practices, for example, smoking, high levels of alcohol consumption and fat intake, and low levels of exercise.

2.3.2.4 Cardiovascular Diseases

The most striking trend in Figure 2.3.2 is the major increase in diseases of the circulatory or cardiovascular system: from about 16 percent in 1911, to a peak of nearly 52 percent in 1971, and in the year 2000 about 45 percent of the population died from circulatory system diseases[135].

Of cardiovascular disease deaths there are two sub-categories that are particularly noteworthy:

1. Ischaemic heart disease (IHD) / Coronary heart disease
2. Stroke / Cerebrovascular disease

By the year 2000, 25 percent of all deaths were caused by IHD and 11 percent related to stroke[136]. Although strokes account for less mortality they have a significantly greater adverse impact on morbidity. By the 1990s strokes had become the second most prevalent cause of hospital admissions[137].

Economic transition, urbanisation and industrialisation have initiated lifestyle changes that promote heart diseases. These risk factors include tobacco use, physical inactivity and unhealthy diet[138]. Smokers of all ages have IHD death rates 2-3 times higher than non-smokers and physical inactivity doubles the risk of dying from IHD or stroke[139]. The table below

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[134] Ibid
[135] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 44
[136] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 61
[137] Ibid
[138] American Heart Association: American Heart Association (2004). "Healthy Lifestyle". Retrieved 26 September 2004, from: http://www.americanheart.org/presenter.jhtml?identifier=1200009
[139] Ibid

highlights the increase in average intake of fat during the twentieth century, which is another significant aggravating factor for cardiovascular diseases.

Table 2.3.4: Summary of all studies that evaluate mean intake of fat as a percentage of energy, 1900-1985[140]

Time Period	No. of Studies	No. of Subjects	Fat (% energy)
1900-1909	1	57	24.6
1910-1919	3	348	24.1
1920-1929	13	5,220	25.9
1930-1939	8	916	33.1
1940-1949	15	2,385	33.2
1950-1959	13	1,568	38.4
1950-1969	10	3,873	40.1
1970-1979	4	2,294	40.3
1980-1985	20	7,384	38.2

The above table highlights the increase in fat intake, which is one of the many unhealthy habits that have become common place as the English economy developed.

Since 1911 there have been very different patterns in the burden of IHD across different age groups and genders. In males aged 35+, IHD increased – apart from an apparent temporary fall during World War Two – until the 1970s and then declined[141]. Female IHD rates experienced a more gradual rise until the 1970s, thereafter rates began to decline for ages 35-44 but ages 65-74 did not experience a decline until the 1980s[142].

2.3.2.5 Neoplasm

The proportion of deaths from neoplasms increased from about 7 percent in 1911 to about 26 percent in 1992, and by 1950 neoplasms represented the second most prevalent mortality category[143]. There are over 200 different forms and locations in which a neoplasm can

[140] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 70
[141] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 48
[142] Ibid
[143] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 30

develop. Therefore, only the major cancer trends will be considered here, namely: lung and breast cancer.

Lung cancer was the most common form of cancer for males. Mortality rates decreased during the last thirty years of the twentieth century, especially at younger ages[144]. For women, who had significantly lower rates, there was an increase since 1970 especially at older ages[145]. This was so extensive that by the 1990s the female mortality rate from lung cancer was very similar to the male rate. These trends in lung cancer generally reflect trends in smoking habits[146].

Despite the increases in female lung cancer, the most common fatal cancer in women during the twentieth century has persisted to be located in the breast. In 1999 one in four female cancers was located in the breast, with around 34,000 new cases diagnosed per annum in England[147]. Since 1970 there has been a decline in breast cancer mortality for ages under 50 and since the late 1980s for ages over 50[148]. These improvements are attributable to the widespread adoption of new diagnostic, surgical and therapeutic regimes.

Cancer mortality in all aged below 25 years experienced increasing rates between 1900 and 1950, and declines thereafter[149]. In ages 25-44 mortality in men shows a similar pattern, although the decline began earlier in the 1940s, while rates for women had been falling throughout the period[150]. In men aged 45-64 the rise was reversed during the 1960s. At ages 65-74 and 75+ male and female mortality rates increased throughout the twentieth century and only began to level off at the end of the 1990s[151].

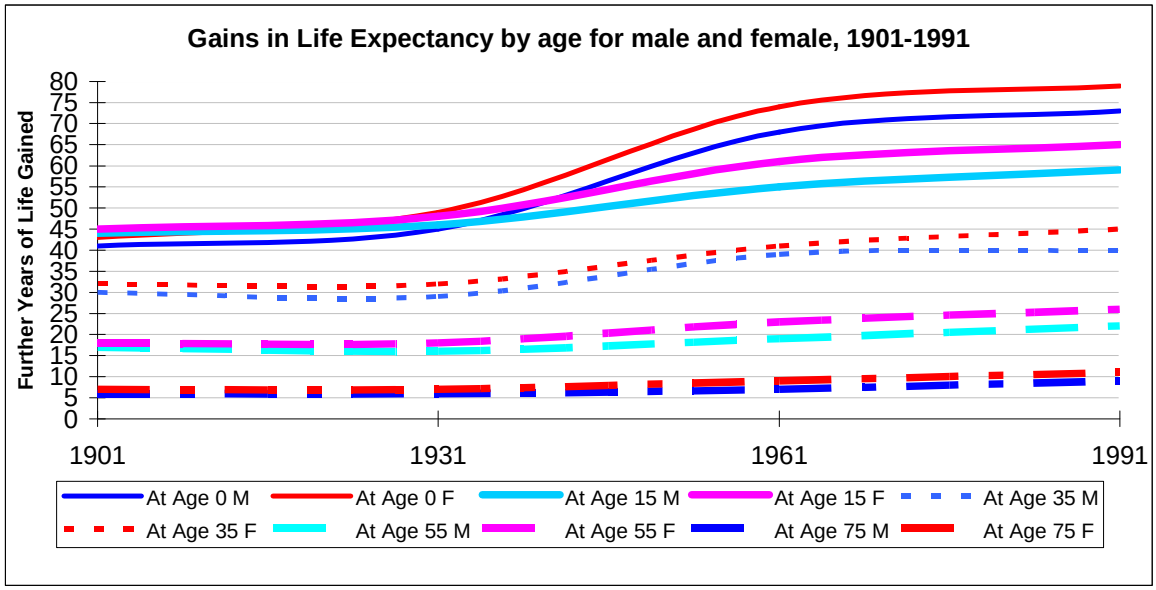
2.3.3 Life Expectancy

By the 1960s deaths from most infectious diseases had declined sharply as a result of continuing improvements in hygiene, sanitation, rising standards of living, immunisation and after the 1930s, specific therapeutic measures and the 1950s with further developments in

[144] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 35
[145] Ibid
[146] Ibid
[147] Cancer Research UK: Cancer Research UK, (2003). "Breast Cancer – Summary". Retrieved 21 November 2003, from: http://www.cancerresearchuk.org/aboutcancer/specificcancers/breastcancer
[148] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume II, p. 36
[149] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 46
[150] Ibid
[151] Ibid

pharmaceuticals[152]. This decline had been substituted with an increase in degenerative diseases, such as heart disease and cancer. This shift in the burden of disease created positive changes in life expectancy.

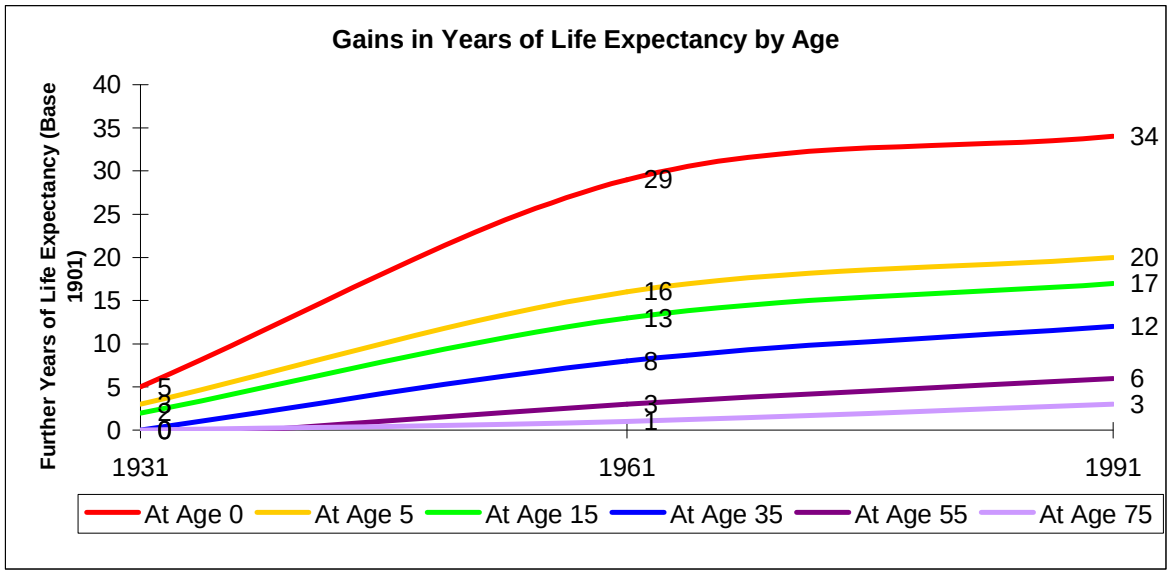
Figure 2.3.3: Gains in life expectancy at age 0, 15, 35, 55, 75, male and female, 1901-1991[153]



The above figure shows the age and gender distributed increases in life expectancy. The most notable trend is the relationship of this increase between different cohorts. The older age groups experienced improvements in their life expectancy increasingly later during the twentieth century.

[152] Davies, "The Pharmaceutical Industry – A Personal Study", p. 2
[153] Charlton & Murphy, "The Health of Adult Britain 1841-1994" Volume I, p. 20

Figure 2.3.4: Average gains in years of life expectancy by age[154]



This graph provides an average of the gains in life expectancy for both sexes, by age and as an addition to 1901 levels of life expectancy at the corresponding ages. The most significant and early increase came for life expectancy at birth (LEo), where between 1901 and 1931 LEo increased by five years, between 1901 and 1961 by 29 years and from 1901 to 1991 by 34 years. This graph clearly illustrates that between 1931 and 1961 LEo experienced the most significant gains compared to the rest of the century and any other time in history. Figures 2.3.4 and 2.3.5 highlight similar trends for life expectancy at other ages (namely: 5, 15, 35, 55, 75) but none of these ages experienced the magnitude of increase enjoyed by infants.

2.3.4 Mortality Change Implications for Morbidity

One of the most significant features of twentieth century health was the shift in mortality from infectious to degenerative diseases. This has numerous implications for the burden and age distribution of morbidity and for mortality/morbidity trade-offs[155].

One of the most important contributions to quality adjusted life expectancy (QALE) and the trade-off was the improved age-death related profile, such that the burden of mortality shifted significantly towards older ages.

[154] Ibid
[155] Trade-off encompasses the notion that although life expectancy has increased, this has been at the cost of an increase in morbid life years, and there is therefore a trade-off between increased life years on the one hand and increased unhealthy years on the other.

Figure 2.3.5: Major cause (aggregate) and all cause of death as a percentage of age specific population 1901-1997[156]

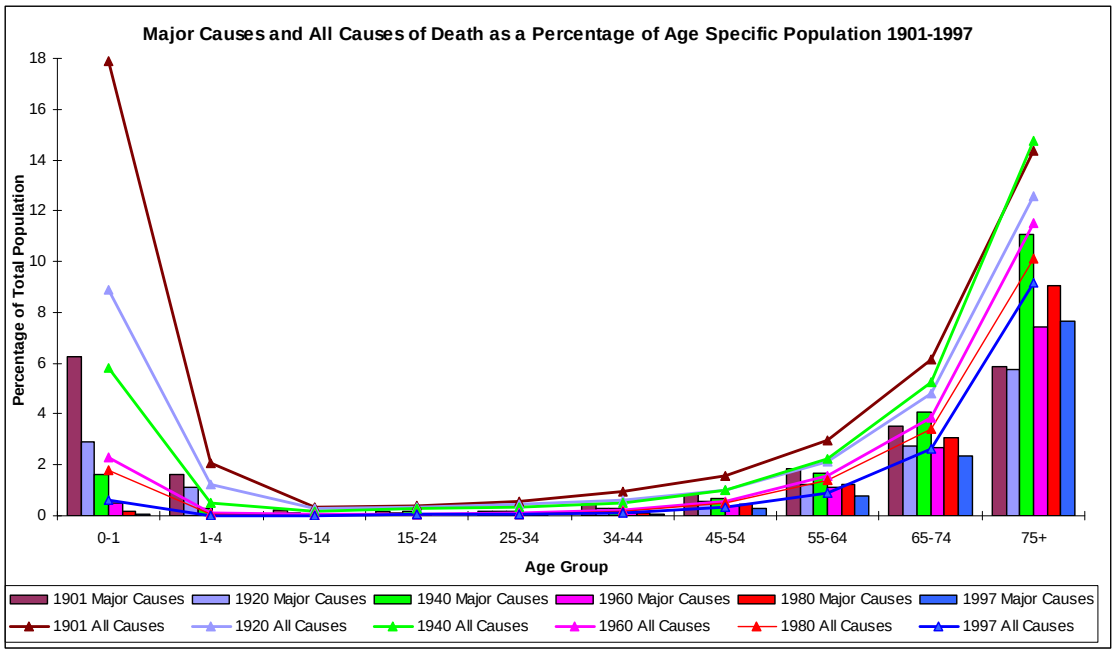


Figure 2.3.5 illustrates the increase in the age of death from 'major causes' (which comprises infectious, respiratory, neoplasm and circulatory mortality, shown by the bars). This trend is also evident for 'all causes' (shown by the lines). This trend is considered in more, mortality cause specific, detail in Figure 2.3.6.

[156] Office of Population, Censuses and Surveys, "Twentieth Century Mortality"

Figure 2.3.6: Death rate (per 1000) for major and all causes 1901-1997[157]

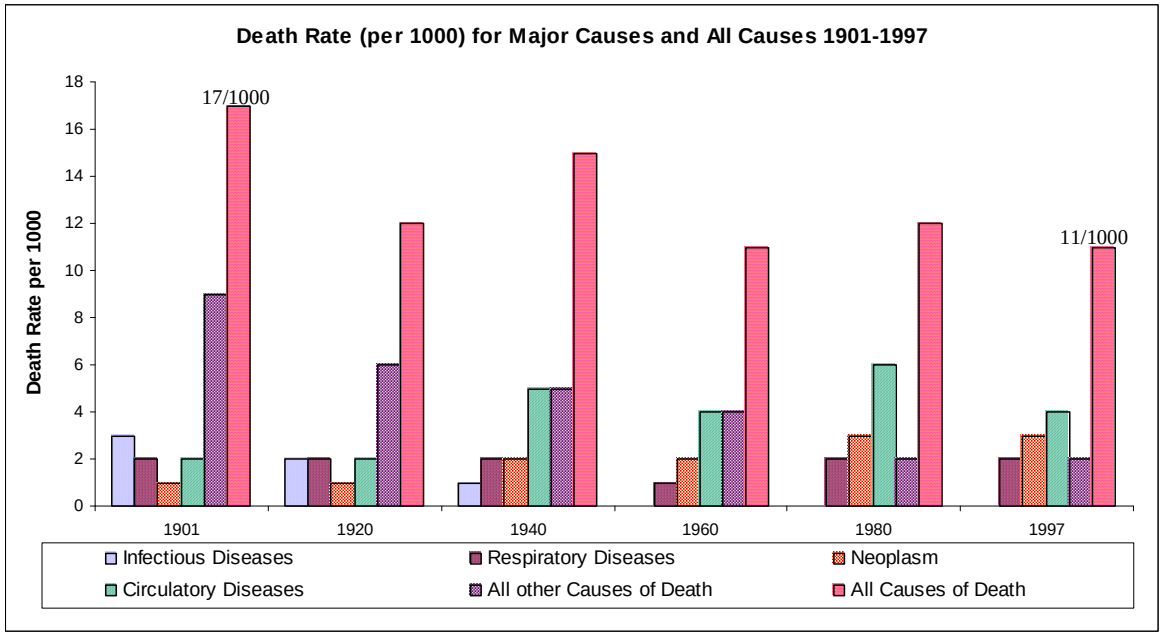


Figure 2.3.6 illustrates three crucial points. First, the decline in the population death rate, from 17/1000 in 1901 to 11/1000 in 1997. Second, the increase in circulatory diseases, which has substituted for the decline in infectious diseases. Third, respiratory diseases mortality remaining relatively constant between 1901 and 1997, which is because this category contains causes of death that are infectious and non- infectious and some of the most persistent infectious mortality. This is illustrated below.

[157] Office of Population, Censuses and Surveys, "Twentieth Century Mortality"

Figure 2.3.7: Distribution of respiratory disease deaths between infectious and non-infectious 1901 and 1997[158]

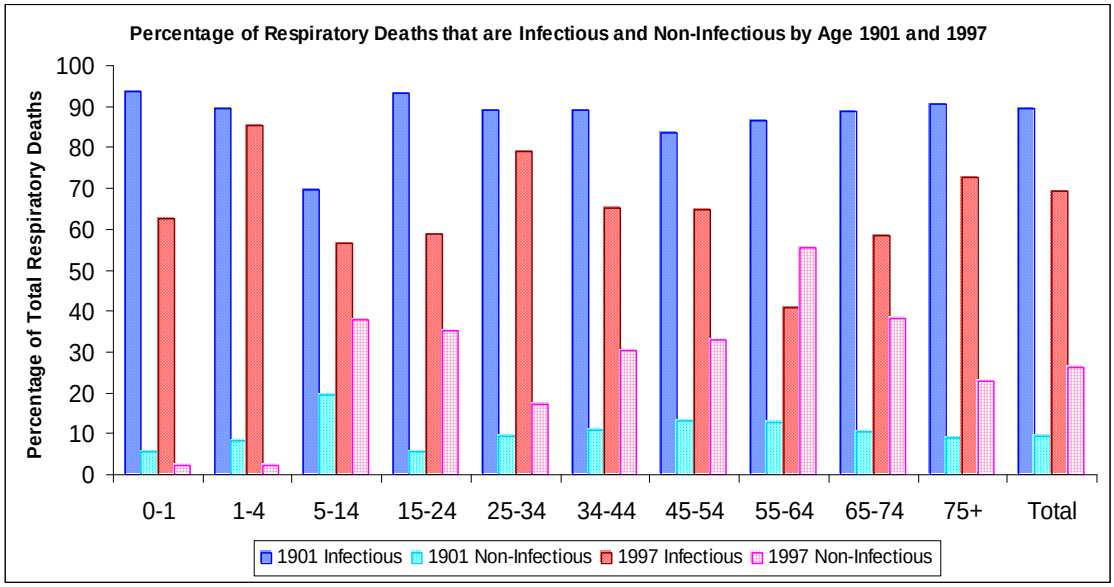


Figure 2.3.7 shows that there have been some expected declines in infectious and corresponding increases in non-infectious respiratory diseases. However, the magnitude is not large enough to explain the whole story. The second explanation is that infectious respiratory diseases represent some of the few remaining infectious diseases, e.g. pneumonia, bronchitis and flu. The increasing age profile of the population perpetuates the remaining prevalence of these infectious diseases.

2.3.5 Mortality/Morbidity Trade-Offs

The previous analysis has highlighted that there is a pattern about the implications for morbidity from the changes in mortality. First, the notion of a trade-off, between reduced mortality at the cost of increased morbidity becomes increasingly pronounced: throughout the twentieth century there was a significant decline in mortality which was accompanied by an increased proportion of the population living in poor health. This is partially a symptom of other factors (exogenous to health per se) and a result of the population living to older ages when degenerative diseases become more prevalent (which exacerbates a decline in killers of young people), but nevertheless there is still some authenticity to the observation of morbidity replacing mortality. This essentially means that 'quality adjusted life expectancy' has not

[158] Office of Population, Censuses and Surveys, "Twentieth Century Mortality"

improved as much as it would seem when only concentrating on death rates. For example, cancer has experienced improvements in mortality but with significant trade-offs for morbidity.

However, to claim that health has worsened because of an increase in the reported prevalence of disease and/or to claim that every additional year of life expectancy in the population is riddled with morbidity is a very simplistic and overly pessimistic conclusion. Even though the changed aetiology of diseases has meant that more years are spent in less than perfect health, it is clear that the trade-off is in favour of overall health. I.e. the thesis' analysis will highlight that the contributions of the improvements in mortality outweigh the associated deteriorations in morbidity and that in many instances the burden of morbidity is less significant than the scenario outlined in contemporary literature. Moreover, as a result of improvements in medical technology, this trade-off became increasingly less pronounced as the twentieth century unfolded. I.e. although medical technology could not cure many ailments it became increasingly able to relieve many of the associated adverse symptoms.

2.3.6 Twentieth Century Health Debates

Despite the seemingly continual improvements in health since the onset of the twentieth century, there has been much debate during certain times about the direction and distribution of health improvements. Most noteworthy was the debate about what happened to health during the inter-war period and issues concerning the persistent health inequalities. These will be analysed below.

2.3.6.1 Effects of the World Wars and the Great Depression on English Health

The pessimistic commentary on the effects of World War One claimed that standards of living declined as nutrition and health levels worsened. The foundations of these claims are reports of poor nutrition and housing, caused by a lack of income due to unemployment[159]. Winter (1994) indicated the contrary through identifying improvements in nutrition and life expectancy[160]. The evidence provides weight for Winter type claims, as there was no interruption in the decline of infectious diseases and mortality (with the exception of life expectancy at age 35, - Figure 2.3.3).

[159] Jones, "Health and Society in Twentieth Century Britain", p. 58
[160] Jones, "Health and Society in Twentieth Century Britain", p. 34

Along a similar vein, Webster (2001) concedes that the British people did experience some overall improvement in living standards during the 1920s and 1930s, but that these improvements were slow[161]. The table below provides weight for this type of argument.

Table 2.3.5: Consumption per head per week (in lbs) for the United Kingdom, 1909-1913 and 1924-1928 relative to 1934-1935[162]

Product	**Years (lbs)**			**% + or – of 1934-35 compared to**	
	1909-13	**1924-28**	**1934-35**	**1909-13**	**1924-28**
Butter	0.30	0.31	0.49	+63	+58
Cereals	4.45	4.11	4.04	-9	-2
Cheese	0.14	0.18	0.19	+36	+6
Eggs, in shell	1.93	2.34	2.90	+50	+24
Fish	0.79	0.80	0.87	+10	+9
Fruit	1.19	1.75	2.23	+87	+27
Margarine	0.11	0.23	0.15	+36	-35
Meat	2.58	2.56	2.81	+9	+10
Milk / Cream	3.46	3.35	3.26	-6	-3
Other Vegetables	1.38	1.81	2.22	+61	+23
Potatoes	4.68	4.43	4.25	-9	-4
Sugar	1.52	1.60	1.79	+18	+11
Tea	0.12	0.17	0.18	+50	+6

The table above highlights an improvement in nutritional standards between 1909 and 1935, which provides contrary evidence to many of the pessimistic claims about nutrition during this period. The most striking trends in the above table are the improvements in quality and quantity of the average British diet[163]. The increases in pounds worth of produce significantly outweigh the declines. Furthermore, the decline in cereals and potatoes is likely to be a direct result of the increased availability of meat, other vegetables and fish. The increase in fruit and

[161] Webster, "Caring for Health: History and Diversity", p. 151
[162] The National Archives: MH 56(213): Advisory Committee on Nutrition, Ministry of Health Advisory Committee on Nutrition
[163] In this context diet quality considers the increase in more nutritious and wholesome sources of calorie intake

vegetables was substantial and would have provided a valuable contribution to quality of life and health standards in general.

It is important to recognise that this table presents aggregate data and therefore may be masking poor nutrition for the poorer segments of the population, although there is little evidential support for the claims about significant inequality in nutrition. For example, in September 1934, a study of 69 working class families (38 of which had an unemployed breadwinner) in Newcastle-upon-Tyne (conducted by the Advisory Committee on Nutrition) found that the average diet of these families provided 2,960 calories a day per man, which was considered to be *"just about sufficient"*[164]. 90 percent of all families consumed fresh vegetables, 88 percent fresh fruit, 69 percent fresh meat and 71 percent fresh fish[165].

In contrast, there were evident inequalities in the death rate and levels of infant mortality. This is shown in the table below.

[164] The National Archives: MH (56)213: Advisory Committee on Nutrition, Dr. Bradford Hill (London School of Hygiene and Tropical Medicine)
[165] Ibid

Table 2.3.6: Ratio of geographical death rates and infant mortality for all regions of England and Wales, 1938[166]

Region	**Deaths at all ages per 1,000 pop**		**Infant mort per 1,000 births**	
	Crude	**Ratio of adjusted rate**	**I. M. Rate**	**Ratio**
England and Wales	11.6	100	53	100
South East	10.7	89	47	88
Greater London	10.2	91	50	94
Rest of South East	11.4	86	42	80
North	12.3	115	60	114
Midlands	11.3	99	52	99
East	11.8	87	44	84
South West	12.9	92	47	88
Wales	12.9	117	57	108
County Boroughs	12.3	112	60	114
Other Urban Districts	11.8	101	50	94
Rural Districts	11.7	90	47	90

Table 2.3.6 highlights geographic inequalities that correspond to areas that experienced a more severe Great Depression. Comparing North to the South East indicates significant inequalities in the death rate (117:86) and the infant mortality rate (114:88)[167]. These inequalities are exacerbated by the fact that the majority of regions in England and Wales experienced death rates that were lower than the average index (100), whereas fewer experienced higher than average death rates which by definition of this distribution being highly skewed, indicates that these deprived regions were especially depressed.

Moreover, Titmuss used data from the 1930s Registrar General's reports to calculate the 'human wastage' in the deprived and high unemployment regions of the North and Wales, compared to the standards of health achieved in the South of the country, he calculated that

[166] The National Archives: MH 58(401): "General Registrar 1950", Ministry of Health: UK: After adjustment of the 'all ages' rates for differences in the sex-age structure of the regions, compares the regional rates with those of England & Wales taken as 100.
[167] Ibid

50,000 excess deaths were occurring each year because of the presence of intense poverty in these areas[168].

Despite the adversities associated with England's World Wars and depression, there were some more optimistic developments during these eras. The post World War One era and 1920s witnessed the seeds of many developments which facilitated continued improvements in the health of the population throughout the twentieth century. Many campaigns of long term significance got underway, e.g. birth control, family allowance and local authority housing[169], when problems of over-crowding and sub-standard homes began to be faced[170]. Interwar developments are even more commendable when scientific achievements are considered. The 1930s marks the dawn of the modern antibiotic era, when therapies were introduced to treat infectious diseases, namely sulphonamides[171]. These developments were enhanced during the following decade (1940s) when the penicillin / tetracycline group of drugs were discovered and developed for mainstream use. The General Registrar (1950) claimed *"there is no doubt that the application of new drugs and remedies to the treatment of bacterial infections since 1937 has been the main reason for the accelerated decline in all-causes death rates"*[172].

The culmination of these developments meant that there were considerable health improvements in the early 1940s, despite the setbacks outlined above.

2.3.6.2 Health Inequalities

Analysis of English mortality by social class began in 1921 and was conducted with increasing detail throughout the twentieth century[173]. This analysis has continually generated concerning results about the inequality in health, such that poor social classes fair much worse in terms of survival and illness probabilities. This has persisted despite the introduction of the NHS (whose main objective was to eliminate inequalities through providing equal accessibility). This aspect of twentieth century health history is more a prominent theme than an actual debate, as all authors agree on the existence and significance of health inequalities, although some disagree on the timing and magnitude of this trend.

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[168] Ibid
[169] Jones, "Health and Society in Twentieth Century Britain", p. 58
[170] Ibid
[171] Ibid
[172] The National Archives: MH 58(401): "General Registrar 1950", Ministry of Health: UK
[173] Pamuk, "Social Class Inequality in Mortality from 1921 to 1972 in England and Wales", p. 17

Context: Historical

The statistical evidence bolsters the theorists who maintain that social class inequality had not been reduced by the end of the twentieth century. The most noteworthy proponent being the Black Report (1980), which was initiated by the government in order to identify the source of inequalities in health and also problems in the functioning of the NHS[174].

Table 2.3.7: Age standardised mortality ratios to indicate social class mortality differentials, males, 1921-1983[175]

Year	**Age (Years)**	**Age Standardised Mortality Ratios by Social Class: Males**				
		I	II	III	IV	V
1921-23	20-64	82	94	95	101	125
1930-32	20-64	90	94	97	102	111
1949-53	20-64	98	86	101	94	118
1959-63	15-64	76	81	100	103	143
1970-72	15-64	77	81	106	114	137
1979-83	20-64	66	74	103	116	165

Table 2.3.8: Age standardised death rates per 100,000 person years, males, 1986-1999[176]

Year	**Age (Years)**	**Age Standardised Mortality Ratios by Social Class: Males**			
		I and II	III Non-Manual	III Manual	IV and V
1986-92	35-64	460	480	617	776
1993-96	35-64	379	437	538	648
1997-99	35-64	347	417	512	606

[174] Drever & Whitehead, "Health Inequalities", Preface
[175] Wilkinson, "Class Mortality Differentials, Income Distribution, and Trends in Poverty", p. 308
[176] National Statistics: Health Statistics Quarterly (2003), "Trends in Social Class Differences in Mortality by Cause, 1986-2000". Retrieved 7 March 2006, from: http://www.statistics.gov.uk/downloads/theme_health/HSQ20.pdf

Context: Historical

Table 2.3.9: Standard mortality ratios for tuberculosis, heart diseases and stomach cancer mortality in adult males aged 20-64 by social class, 1930-1972[177]

Year	Cause of death	Social Class (Standard Mortality Ratios)				
		I	II	III Non Manual / Manual	IV	V
1930-32	Tuberculosis	61	70	100	104	125
1950	Tuberculosis	64	62	103	95	149
1970-72	Tuberculosis	26	41	84 / 89	124	254
1930-32	Heart Disease	65	92	97	111	112
1950	Heart Disease	61	87	103	102	114
1970-72	Heart Disease	77	80	117 / 103	116	124
1930-32	Stomach Cancer	59	84	98	108	124
1950	Stomach Cancer	57	67	100	114	132
1970-72	Stomach Cancer	50	66	79 / 118	125	147

Table 2.3.10: Standard death rate for respiratory disease, heart diseases and stomach cancer mortality in adult males aged 35-64 by social class, 1986-1999[178]

Year	Cause of death	Social Class (Standardised Death Rate)			
		I and II	III Non-Manual	III Manual	IV and V
1986-92	Respiratory Disease	13	21	37	49
1993-96	Respiratory Disease	16	28	32	44
1997-99	Respiratory Disease	11	40	47	61
1986-92	Heart Disease	160	162	228	270
1993-96	Heart Disease	97	117	159	215
1997-99	Heart Disease	90	117	141	167
1986-92	Stomach Cancer	5	7	15	19
1993-96	Stomach Cancer	3	7	9	8
1997-99	Stomach Cancer	6	6	11	7

[177] Watters, "Class Inequality and Healthcare", p. 123. For 1970-72 Social Class III non-manual / manual estimates are provided, respectively.
[178] National Statistics: Health Statistics Quarterly (2003), "Trends in Social Class Differences in Mortality by Cause, 1986-2000". Retrieved 7 March 2006, from: http://www.statistics.gov.uk/downloads/theme_health/HSQ20.pdf

It should be noted, that because these official figures appear to show that differences in mortality rates have widened even during periods when the consensus believed that socio-economic inequalities were diminishing, they have been regarded with considerable scepticism[179]. For example, the table below provides estimates about social class inequality by life expectancy and contradicts the specific trends implied by the previous table.

Table 2.3.11: Life expectancy at age 0 and 65 by social class, male and female average, 1972-1999[180]

Social Class	**1972-1976**	**1982-1986**	**1992-1996**	**1997-1999**
At Age 0				
I	75.6	77.8	80.6	80.7
II	74.4	76.2	78.5	79.5
III	73.1	74.8	76.9	77.8
IV	71.7	74.0	75.2	75.6
V	70.2	71.5	72.6	74.1
At Age 65				
I	16.8	17.0	18.8	19.2
II	15.2	16.2	17.5	18.4
III	14.7	15.4	16.5	17.2
IV	14.6	15.0	15.5	15.6
V	14.0	13.9	14.5	14.9

The above table highlights that, during the last three decades of the twentieth century life expectancies varied consistently with social class. The table also illustrates that, although each social class has increased its life expectancy over this period, some social classes have gained more than others. However, the most noteworthy feature is the persistence of social class in equality despite twentieth century health developments, especially the National Health Service.

[179] Ibid
[180] National Statistics: Health Statistics Quarterly (2002), "Inequality in Life Expectancy by Social Class, 1972-1999". Retrieved 7 March 2006, from: http://www.statistics.gov.uk/downloads/theme_health/HSQ15.pdf

Despite the slight variation in magnitude and trends between the series of tables above, the overall existence of social class inequalities is undeniable. It is most important to recognise that there were persistent health inequalities.

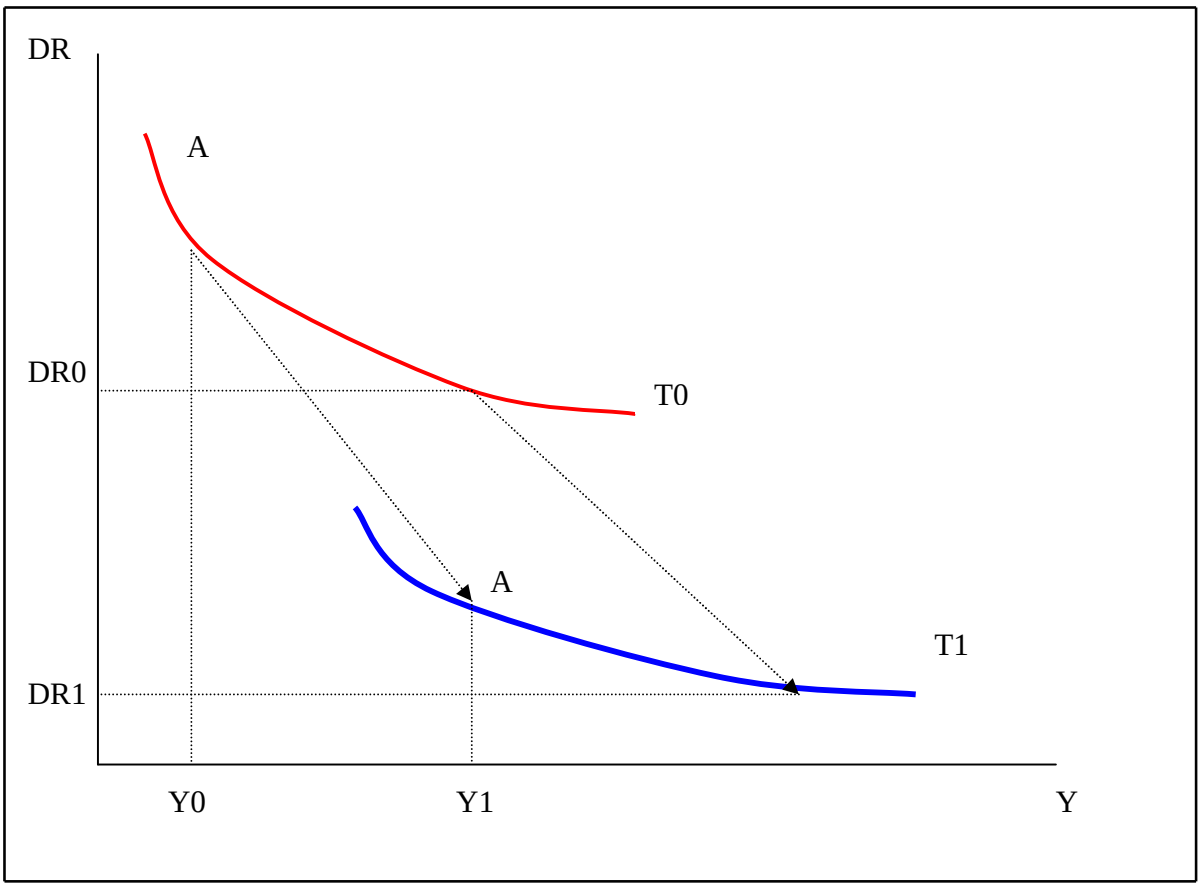
Because of the inconsistencies in the numbers some authors have approached the problem from a different angle. E.g. Wilkinson (1989) considers relative poverty in twentieth century Britain[181]. His cogent reason for thinking that health is more responsive to changes in income at the bottom end of the scale led him to focus more narrowly on trends in relative poverty[182]. As well as providing an insightful analysis, this also provides plausibility to the previous mortality inconsistencies. Wilkinson also provides an explanation for why the problem is so exaggerated in twentieth century Britain[183]:

> *It might be suggested that the whole population would move down and out along the curve so that the death rates of those at the lower end of the income distribution would fall faster than others. That this has not happened adds weight to the claims that we are dealing with relative rather than absolute poverty. Essentially, Britain experienced a situation where as the country got richer, rather than moving along the curve relating income to health, the curve itself moves down and to the right.*

This is depicted in Figure 2.3.8, where this movement from curve T0 to T1 (rather than along curve T0) enables richer people to reduce their death rates further than previously (shown by the move from DR0 to DR1), while the poor have to pay more to keep themselves off the steeply rising part of the curve (shown by the move from Y0 to Y1)[184].

[181] Wilkinson, "Class Mortality Differentials, Income Distribution, and Trends in Poverty"
[182] Wilkinson, "Class Mortality Differentials, Income Distribution, and Trends in Poverty", p. 333
[183] Wilkinson, "Class Mortality Differentials, Income Distribution, and Trends in Poverty", p. 332
[184] Ibid

Figure 2.3.8: Long run GDP/capita increase and income mortality[185]



These mortality inequalities (in income and relative poverty) are also evident in morbidity, and some have claimed to be more pronounced. E.g. by the year 2000 there were no major disease conditions that were more prevalent in wealthier social classes[186]. Even diseases that used to be associated with affluence and were more prevalent in wealthier social classes at the beginning of the twentieth century became more prevalent in poorer social classes by the end of the twentieth century[187]. E.g. cancer and coronary heart disease were all more prevalent in poorer social classes by the close of the twentieth century. This is highlighted in the table below, which depicts social class inequality for a selection of mortality causes in 1960, when this crossover was largely complete.

[185] Graphed from the qualitatively presented description in: Wilkinson, "Class Mortality Differentials, Income Distribution, and Trends in Poverty", p. 332
[186] Gjonca, SA4A2 Lecture 11, January 2003, London School of Economics
[187] Ibid

Table 2.3.12: Cause of death by social class, males,1961[188]

Cause of death	**Standardised mortality ratios: social class**				
	I	**II**	**III**	**IV**	**V**
Tuberculosis	40	54	96	108	185
Malignant Neoplasm	73	80	104	102	139
Psychoses	80	77	96	80	179
Epilepsy	30	39	46	97	251
Rheumatic Fever	40	67	85	113	207
Pneumonia	48	54	88	102	196
Bronchitis	28	50	97	116	194
Stomach ulcer	46	58	94	106	199
Accidents in the home	95	78	81	104	226
Suicide	91	94	87	103	184

A comparative study of mortality rates between 98 area health authorities (AHAs) identified considerable variation within their sample of diseases[189] (which were chosen for their strong association with medical intervention), and this remained even after adjustment for social factors[190]. The table below highlights the extent of the variation in these conditions.

[188] Tunbridge, "The Effect of Social Class on Hospital Use", p. 1
[189] The diseases that were considered in this study are as follows: Hypertensive Disease, Cancer of Cervix Utteri, Pneumonia and Bronchitis, Tuberculosis, Asthma, Heart Disease, Respiratory Disease, Bacterial Infections, Hodgkin's Disease, Abdominal Hernias, Cholecystitis, Appendicitis, Maternal deaths, Deficiency Anemia, Perinatal
[190] Charlton et al, "Geographical Variation in Mortality from Conditions Amenable to Medical Intervention in England and Wales", p. 691

Context: Historical

Table 2.3.13: Distribution of five-year standardised mortality rates (SMRs) among area health authorities of England and Wales, 1974-78[191]

Cause of death	**Age Group**	**SMRs**			**P value**
		Median	**Minimum**	**Maximum**	
Hypertensive Disease	5-64	96	29	213	<0.001
Cancer of the cervix utteri	5-64	99	43	162	<0.001
Pneumonia and Bronchitis	5-49	96	39	294	<0.001
Tuberculosis	5-64	92	19	250	<0.001
Asthma	5-49	97	31	249	<0.001
Heart Disease	5-44	105	0	263	<0.001
Respiratory Disease	5-49	90	0	374	<0.001
Bacterial Infection	5-64	101	16	257	<0.1
Hodgkin's Disease	5-34	98	0	288	<0.1
Abdominal Hernias	5-64	94	18	279	<0.05
Cholecystitis	5-64	99	0	323	NS
Appendicitis	5-64	106	0	228	NS
Maternal deaths (per 1000 births)	10-44	0.6	0.0	1.8	<0.1
Deficiency Anemia	5-64	87	0	508	<0.01
Perinatal (per 1000 births)	-	18.0	10.0	24.0	<0.001
All Causes		101	64	118	<0.001
All 'non-preventable'		101	63	119	<0.001

The above table highlights that there was variation in mortality for nearly all of the disease groups. This will be elaborated below.

[191] Charlton et al, "Geographical Variation in Mortality from Conditions Amenable to Medical Intervention in England and Wales", p. 693

Table 2.3.14: Distribution of social indicators by area health authority (AHA), 1977-78[192]

Social indicator	**Distribution**		
	Median	**Minimum**	**Maximum**
Households without cars (%)	41.9	24.5	64.4
Unskilled workers (%)	6.2	3.2	10.4
Households renting (%)	41.1	28.0	81.3
Birth-weights under 2.5kgs (1980) (%)	7.0	4.4	10.3

This table provides an indication about why a substantial variation in survival was still evident after controlling for social factors, because a significant part of the above variations in mortality is not born from social factors.

Along a similar vein, Tunbridge (1977) conducted a study that considered social class and hospital use[193]. The key finding states that there is *"No social class variation in length of stay, by specialty or diagnosis"* [194]. This supports other studies that have failed to show any relationship between social class and length of hospital stay or use[195].

If the health of lower social classes is generally poorer but their hospital use does not reflect this, then there is a clear indication that higher mortality would ensue as a consequence. If this tentative argument is accepted as a contender in social class mortality inequality, then the most important question would be: what is the cause of the seemingly positive correlation between social class/income and hospital use? I.e. is this a story about education and culture or access to medical care?

The underpinnings of this question about access to medical care in the first half of the twentieth century are related to the markedly varied evaluations of pre-NHS hospitals. For example, while some contemporary observers stated that, *"it is universally acknowledged that*

[192] Ibid
[193] Tunbridge, "The Effect of Social Class on Hospital Use"
[194] Tunbridge, "The Effect of Social Class on Hospital Use", p. 12
[195] For example, Logan et al, "Dynamics of Medical Care: The Liverpool Study into Use of Hospital Resources" and Weiss & Greenlick, "Determinants of Medical Care Utilisation"

our health services are the best in the world", others were generally critical[196]. It is often implied that before the advent of the NHS, the poor were denied the healthcare available to their wealthy counterpart. A closer analysis of the pre-NHS healthcare system implies that might not be strictly true in the pre 1948 era.

Free public and charitable care was available for the working class even in the mid nineteenth century and treatment was provided at a small cost through various types of contributory scheme[197]. From 1911, many workers received free general practitioner care under the National Health Insurance, and only those above the level of destitution began to be charged for their care. Middle class patients were often exempt from such schemes, and by having to pay high private fees they subsidised the care of working class patients[198]. It is possible that in terms of GP care, middle class patients received better treatment, but this does not seem to have been the case in hospitals, as middle class patients were generally confined to the smaller and less well equipped (in both machinery and specialists) private hospitals, as the most proficient hospitals were in the voluntary sector and unavailable to the middle classes[199]. Furthermore, there does not seem to be any affluence inequality (by geographic location in England and Wales) in hospital distribution. The table below presents geographic variations, but without a clear North: South dichotomy, although there does seem to be some evidence of variation between hospitals.

[196] Powell, "Hospital Provision before the National Health Service: A Geographical Study of the 1945 Hospital Survey", p. 483
[197] Watters, "Class Inequality and Healthcare", p. 43
[198] Ibid
[199] Powell, "Hospital Provision before the National Health Service: A Geographical Study of the 1945 Hospital Survey", p. 485

Table 2.3.15: Hospital beds (per 1,000 population) by region in England and Wales, 1938[200]

Region	**Hospital beds (per 1,000)**					
	Acute	**Chronic**	**Maternity**	**Tuberculosis**	**Infectious Diseases**	**Total**
North-Western	3.35	1.04	0.33	0.75	0.88	6.34
North-Eastern	2.43	0.75	0.18	0.84	1.22	5.42
Yorkshire	2.16	1.16	0.23	0.59	1.18	5.32
West Midlands	2.57	1.40	0.20	0.59	0.69	5.45
Sheffield and East Midlands	2.74	1.10	0.27	0.64	0.79	5.55
Eastern	2.18	2.19	0.13	1.04	0.71	6.26
South-Western	2.63	1.62	0.22	0.61	0.75	5.83
Berkshire, Bucks and Oxfordshire	2.20	1.91	0.19	0.35	0.62	5.26
London and South-East	3.77	1.32	0.31	0.76	1.21	7.38
England	3.08	1.28	0.27	0.71	1.00	6.35

Possibly more influential was the variation in financial constraints that was faced by interwar hospitals. Hospitals in the North-West and South-East were equally constrained[201]. Modernisation and increasing medical staff costs seem to be an important determinant of strain and subsequent service of interwar hospitals, which were often unequally distributed between hospitals independently of geographical affluence. Without a central system to redistribute resources from surplus to deficit hospitals these inequalities were not reduced, despite an increase in bed numbers per population in interwar Britain[202]. It was not until 1948 that these differences began to be addressed (through an integrated hospital system that was more able to allocate resources).

[200] Powell, "Hospital Provision before the National Health Service: A Geographical Study of the 1945 Hospital Survey", p. 498
[201] Gorsky et al, "The Financial Health of Voluntary Hospitals in Interwar Britain", p. 539
[202] Gorsky et al, "The Financial Health of Voluntary Hospitals in Interwar Britain", p. 543

Although the NHS reduced some of these problems the NHS has proved to be *"remarkably ineffective"* in reducing class inequalities in health[203]. What is most persistent and vivid is the conclusion that improvements in health (outlined above) were distributed in favour of richer social classes.

2.3.6.3 Inefficiency of the NHS

A consistent theme throughout the findings of the thesis is that the poor performance of the NHS detracted from further improvements in quality of life, in general and also as a result of the health inequalities it failed to eliminate.

Evidence of the shortcomings of the NHS is highlighted by recent considerations about the efficiency of healthcare systems. At the end of the twentieth century evaluations began to be made (by scholars and the World Health Organisation) about the overall performance of healthcare services in a variety of countries. These measures have considered numerous facets that are capable of reflecting efficacy of health care services, for example: the level and distribution of health attainment, responsiveness of the health system and the degree of fairness in financing relative to the country's level of economic and educational development[204]. Hence, to establish the contribution of a healthcare system (or 'mortality amenable to healthcare') it is necessary to determine what the health service achieves in excess of what would have been achieved in its absence, in absolute terms and also relative to healthcare service investment[205].

The results of these studies have highlighted the poor performance of the British NHS. Nolte & McKee (2003) have illustrated the fall in Britain's ranking when 'mortality amenable to healthcare' is considered[206]. For example, when considering disability adjusted life expectancy the UK achieves a world rank of tenth, but when the healthcare service is considered in conjunction with these efficiency proxy outcomes, the UK rank declines to eighteen. Evans et al (2001) have painted an even bleaker picture with their considerations about healthcare

[203] Watters, "Class Inequality and Healthcare", p. 127
[204] Nolte & McKee, "Measuring the Health of Nations: Analysis of Mortality Amenable to Healthcare", p. 1129
[205] Evans & Tandon & Murray & Lauer, "Comparative Efficiency of National Health Systems: Cross National Econometric Analysis", p. 307
[206] Nolte & McKee, "Measuring the Health of Nations: Analysis of Mortality Amenable to Healthcare", p. 1131

efficiency, where the UK only achieves a word rank of twenty-fourth, behind much lesser developed economies such as Jamaica, Malta and Oman[207].

Although it is important to appreciate that these new measures, which consider 'mortality amenable to healthcare' are in their infancy and that these types of studies have had to contend with numerous anomalies and controversies there is still a clear and valid message about the paucity of the performance of the NHS, particularly during the last decade of the twentieth century (when these studies where conducted). When considering 'mortality amenable to healthcare efficiency' the UK experiences significant losses in its performance appraisal.

Finally, in conjunction with being inefficient the NHS was also under funded, both of which have contributed to comparatively poor healthcare for the English population. For example, it was not until the new millennium that the government finally conceded the need to bring its health service spending (on cancer) up to the European average. However, there was still no guarantee that funds would be available on a sustained basis, necessary to bring the NHS standard up to the European average.

The problems of escalating costs, that have plagued the NHS since its introduction, are faced by all health services, but during the second half of the twentieth century they have become increasingly and particularly acute in the UK because of under spending and neglect, which were evident from the birth of the NHS[208].

[207] Evans & Tandon & Murray & Lauer, "Comparative Efficiency of National Health Systems: Cross National Econometric Analysis", p. 308
[208] Webster, "The National Health Service: A Political History", p. 257

3. Methodology

The methodology in this thesis will build upon the most commendable existing health studies, which only consider the value of improved mortality. These studies will be enhanced through the addition of the thesis morbidity considerations, in order to provide an accurate and comprehensive health measure, which is currently void in the literature.

This chapter of the thesis will begin with a detailed consideration about the requirements of the health measure. It will answer the key question: what is the thesis methodology trying to measure? And, why are these features the most important aspects to gauge for considering improved health in twentieth century England?

This will be followed by the solutions to the demands above, through providing an outline about the existing willingness to pay (WTP) model and how this will be developed into an extended willingness to pay or quality adjusted life expectancy (QALE) model which forms the thesis' central methodology.

This chapter will also delineate the sensitivity analysis that will be applied to the results of the QALE methodology, in order to provide a broader range of estimates and a more comprehensive and justified indication of the possible contribution of improved health.

3.1 Methodology Outline

A major component of the thesis is the identification and valuation of improvements in health related quality of life. Once these developments have been documented, the thesis

will endeavour to quantify them so that they can be included in an extended measure of national income. This will be achieved through developing the existing willingness to pay methodology (WTP) to include morbidity improvements as WTP currently only considers mortality improvements. Hence, a significant attribute of the thesis methodology will be the creation of an original (extended) WTP model, which considers morbidity in tandem with mortality. A simplified version of this process is outlined at the end of this chapter in Figure 3.4, which is useful for referencing in order to understand the general features of the QALE methodology.

3.2 Willingness to Pay Methodology (WTP)

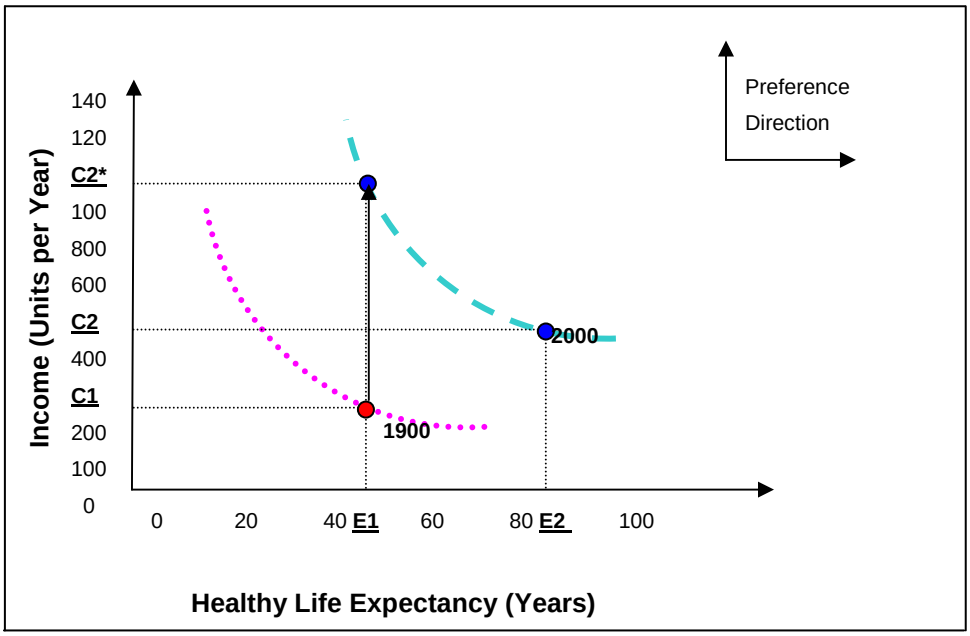
The WTP methodology considers what individuals would be willing to forego in income for an increased probability of survival with healthy life years. I.e. the amount an individual would pay for an increase in their healthy life expectancy in the current period (not for a given [future] age of life expectancy or to prevent specific illnesses).

Numerous authors have highlighted the importance of improved mortality to society, to such an extent that there is a plausible likelihood of an individual sacrificing any other modern standards of living to maintain current mortality rates[209]. Bradford DeLong (2000) has highlighted the contribution of improved health to economic development, which has been so extensive during the twentieth century (in developed countries) that there is not likely to be any magnitude of income that could compensate a year 2000 family for living under 1900 health conditions. Therefore, given the absence of modern vaccines, antibiotics and other technologies in 1900, it is hard to argue that anything less than an astronomical income in 1900 could compensate for the health of 2000[210].

The indifference curve diagram below illustrates these types of claims and subsequently illuminates the basic notion of the thesis methodology.

[209] Usher, "The Measure of Economic Growth", p. 223
[210] Bradford DeLong, "Cornucopia: The Pace of Economic Growth in the Twentieth Century", p. 22

Figure 3.1: Indifference curve diagram to illustrate the rationale of the WTP methodology



Consider a person observed initially at point 1900 and subsequently at point 2000: between 1900 and 2000 life expectancy has increased from E1 to E2 and income has increased from C1 to C2*, not C2, as traditional measures would indicate. Point C2* is the height of the intersection of the indifference curve attained in 2000 with a vertical line at the value of life expectancy in 1900, whereby the individual maintains 2000 income with 1900 life expectancy. The difference between C2 and C2* indicates the income or consumption value of increased life expectancy between 1900 and 2000 and the amount of income that an individual would be willing to pay for the improved health conditions of 2000, compared to 1900.

The notion of imputing national income measures to account for increases in life expectancy was initially proposed by Usher (1980), who recognised the need to consider the value of maximising age specific mortality rates and societies' willingness to pay for this improvement[211]. Usher's main objective was to find a natural way of combining the two social indicators (GNP and mortality rates) into a single comprehensive index, his basic contention being that the growth of GNP alone significantly understates the extent to which current generations are better off than earlier generations.

[211] Usher, "The Measure of Economic Growth", p. 228

These considerations have been developed in a limited number of studies, namely: Nordhaus (1999)[212] for the USA between 1900 and 1995, Crafts (2001)[213] for the UK between 1870 and 1998 and Hickson (2002)[214] for twentieth century Japan. Despite their agreement with Usher's objectives of providing a more indicative national income estimate, none of these studies measure health per se, as they all utilise mortality (i.e. increased life expectancy) as a proxy for health.

In this willingness to pay approach, gains from improved mortality are treated as an imputation for a change in the environment, because increased life expectancy has been largely a result of the accumulation of knowledge on how to cure and prevent diseases that affect all individuals (rich and poor, educated and uneducated), and subsequently this is the reason that these improvements are not included in income measures, and therefore not double counted by WTP imputations[215].

3.2.1 Extended Willingness to Pay Methodology (WTP)

The thesis will enhance existing willingness to pay methods in order to consider the value of increased life expectancy (as has been accomplished in the Nordhaus, Crafts and Hickson studies) and also improved morbidity from a health and welfare perspective. As well as considering the reduction in the death rate and the corresponding value of an improved mortality profile, the thesis will consider the decline in the burden of morbidity and its associated value. Although this more comprehensive health measure can only yield estimates it is still superior as these estimates provide a much more accurate indication about health than the more precise but less detailed mortality only estimates. Hence, through adopting a more comprehensive (utility based) measure of economic growth it is possible to indicate how existing (fiscal only) measures are inadequate for providing a thorough indication of improvements in welfare. Moreover, through adopting a more comprehensive definition of health it is possible to estimate the full gain in utility towards an extended national income. This provides results that are greater than those that are yielded when extending GDP for mortality only.

Finally, the methodological approach used in the thesis to obtain utility national income (which is defined as the maximum amount that a nation can consume while ensuring that

[212] Nordhaus, "The Health of Nations: The Contribution of Improved Health to Living Standards"
[213] Crafts (2005) "The Contribution of Increased Life Expectancy to Growth of Living Standards in the UK, 1870-1998". Retrieved 17 June 2005, from: www.york.ac.uk/res/wpeg/documents/crafts.pdf
[214] Hickson, "The Contribution of Increased Life Expectancy to Standards of Living in Twentieth Century Japan"
[215] Usher, "The Measure of Economic Growth", p. 247

members of generations can have expected lifetime utility the same as that of the current generation), values improvements in quality adjusted life expectancy by considering the change in the population weighted average of age specific mortality rates multiplied by the estimated value of death averted in conjunction with the population weighted average of the morbidity burden multiplied by the estimated value of unhealthy life years averted. This is approximately equal to the increase in quality adjusted life expectancy times the value of an additional healthy life year.

The data required to make these willingness to pay methodological adjustments to the conventional estimates of national income are population by age, death rates by age and the value of death averted (value of a statistical life). The data required to make extended willingness to pay estimates are population by age, prevalence of a given disease, the burden of the disease (quality adjusted life year, QALY) and the value of ill health averted (value of a statistical healthy life year). These two calculations will then be combined to estimate the aggregate health improvement, namely, QALE.

3.2.2 Value of a Statistical Life (VSL)

In order to estimate a society's willingness to pay for reduced mortality it is necessary to establish the amount that a group of people (a society) would be willing to pay for a reduction in the current period probability of death. VSL studies estimate the value of fatal risk reduction (through evaluating the amount that a society is willing to pay) in the expectation of saving one life (of an unidentified person) in the current period[216]. For example, if people are on average willing to pay £10 for a safety improvement that will reduce their individual risk of death during the coming year by 1 in 100,000, this risk reduction would mean that, on average, in a group of 100,000 people there would be 1 less premature death, and these 100,000 people would, between them, be willing to pay £10 x 100,000 = £1 million for the prevention of 1 statistical fatality[217]. Hence in this example the VSL is £1 million.

There is a growing body of empirical evidence concerning premiums individuals are willing to pay to reduce the risk of death by small amounts[218]. One method for deriving such estimates is based on individuals' observed behaviour in production, e.g. labour market risks compensating wage studies. Another approach considers the implications of

[216] Miller, "Variations between Countries in the Values of Statistical Life", p. 170
[217] This is example is taken from: The Department of Health, "Economic Appraisal of the Health Effects of Air Pollutants", p. 2
[218] Blomquist, "The Value of Human Life: An Empirical Perspective", p. 158

individuals' observed behaviour in consumption, e.g. data concerning the time-inconsistency-safety trade offs involved in car seat belt use, motorway speed decisions, the purchase price and the maintenance of smoke detectors and the frequency of car tyre replacement[219]. Both of these approaches are revealed preference studies. The third method, contingent valuation (CV), analyses replies to questionnaires that ask individuals about their willingness to pay for various hypothetical changes in risk.

The majority of revealed preference studies have focussed on risk compensating wage differentials of hedonic price studies, where there is an equilibrium wage risk function such that, at any particular point workers are substituting income for risk of death. By estimating this equilibrium wage risk function – while controlling for other job characteristics – it is possible to identify the compensating wage differential and subsequently estimate the value of a statistical life.

Because labour market studies reflect actual behaviour, with decisions that are consistently repeated, it is thought that this form of VSL study is most reliable and therefore many more studies have been conducted from this perspective, which has generated a much richer variety in risk compensating revealed preference studies (from a collection of time periods and occupation).

Estimates of the VSL range widely: from less than $100,000 to several million dollars[220]. The table overleaf highlights the extent of this variation among the most credible VSL studies conducted over the previous three decades.

[219] Jones-Lee, "The Economics of Safety and Physical Risk", p. 54
[220] Dillingham, "The Influence of Risk Variable Definition on Value of Life Estimates", p. 277

Table 3.1: Estimates of the VSL derived from the most credible studies of the last three decades[221]

Author / Year	Type of VSL study	Estimated VSL
Thaler & Rosen (1973)	Compensating wage differential (USA)	1987£420,000
Smith (1973)	Compensating wage differential (USA)	1987£7,950,000
Melinek (1974)	Compensating wage differential (UK)	1987£990,000
Viscusi (1978)	Compensating wage differential (USA)	1987£2,590,000
Veljanovksi (1978)	Compensating wage differential (UK)	1987£4,550,000
Dillingham (1979)	Compensating wage differential (USA)	1987£400,000
Brown (1980)	Compensating wage differential (USA)	1987£1,270,000
Needham (1980)	Compensating wage differential (UK)	1987£130,000
Olson (1981)	Compensating wage differential (USA)	1987£5,260,000
Arnould & Nichols (1983)	Compensating wage differential (USA)	1987£410,000
Miller (2000)	Compensating wage differential (Average of 13 countries)	1995$3,384,000[222]
Costa & Kahn (2003)	Compensating wage differential (USA value for 1980)	1990$4,500,000[223]
Viscusi & Aldy (2003)	Compensating wage differential (USA)	2000$6,600,000[224]
Viscusi & Aldy (2003)	Compensating wage differential (Average of 10 countries)	2000$5,600,000[225]
Viscusi & Kniesner (2005)	Compensating wage differential distributed by workers' relative position and life-cycle pattern of consumption (USA)	2005$4,750,000[226]
Melinek (1974)	Time-inconvenience-safety trade off: use of pedestrian subways (UK)	1987£400,000
Jones-Lee (1977)	Wealth-safety trade off: frequency of tyre replacement (UK)	1987£1,830,000
Blomquist (1979)	Time-inconvenience-safety trade off: use of car seatbelts (USA)	1987£400,000
Dardis (1980)	Purchase of domestic smoke detectors (USA)	1987£280,000
Portney (1981)	House price-air pollution trade off (USA)	1987£150,000
Ippolito & Ippolito (1984)	Cigarette smokers' response to health hazard information (USA)	1987£390,000
Acton (1973)	Small non-random sample survey (n=93) of WTP for heart attack ambulance (USA)	1987£50,000
Melinek et al (1973)	Non-random sample survey (n=873) of WTP for domestic fire safety (UK)	1987£250,000
Melinek et al (1973)	Non-random sample survey (n=873) of WTP for hypothetical safe cigarette (UK)	1987£80,000
Jones-Lee (1976)	Small non-random sample survey (n=31) of WTP for airline safety (UK)	1987£8,250,000
Maclean (1979)	Quota sample survey (n=325) of WTP for domestic fire safety (UK)	1987£2,480,000
Frankel (1979)	Small non-random sample survey (n=169) of WTP for elimination of small airline risk (USA)	1987£11,700,000
Jones-Lee et al (1985)	Large random sample survey (n=1,150) of WTP for transport safety (UK)	1987£1,860,000

Table 3.1 highlights a range of some of the most credible VSL studies that have been conducted in the UK and the USA between 1973 and 2005. These studies have used a

[221] Jones-Lee, "The Economics of Safety and Physical Risk", p. 91-93
[222] Miller, "Variations between Countries in Values of Statistical Life", p. 177: Average of values for Australia, Austria, Canada, Denmark, France, Japan, New Zealand, South Korea, Sweden, Switzerland, Taiwan, UK, US in 1995 US dollars.
[223] Value in 1990 $ and is the mid-point between the study estimate range of 4,000,000-5,000,000: Costa & Kahn, "Changes in the Value of Life 1940-1980", p. 13
[224] Value in 2000 $ and is the mid-point between the study estimate range of 5,500,000-7,600,000: Viscusi & Aldy, "The Value of a Statistical Life: A Critical Review of Market Place Estimates throughout the World", p. 45
[225] Value in 2000 $ and is the mid-point between the study estimate range of 5,000,000-6,200,000 and is the average value for: Australia, Austria, Canada, Japan, UK, Hong Kong, India, South Korea, Taiwan: Viscusi & Aldy, "The Value of a Statistical Life: A Critical Review of Market Place Estimates throughout the World", p. 45
[226] Value in 2005 $ and is the mid-point between the study estimate range of 4,700,000-4,800,000: Kniesner & Viscusi, "Value of a Statistical Life: Relative Position versus Relative Risk", p. 143

variety of methods, references and samples (across the UK and the USA) to try and derive a VSL. Table 3.1 highlights that these studies also report their VSL results in a variety of currencies. This is not a problem because the key value of the table is to highlight the range of existing VSL estimates, which Table 3.1 highlights is so broad, even without a standardised currency. Therefore, Table 3.1 indicates that the collection of even the most credible VSL studies lack precision, which has led sceptics to claim that "*the variation in VSL estimates raises such doubts about their reliability that they are virtually redundant"* (for example, for a time this sceptical view was adopted by the UK Department of Transport)[227]. A more positive and preferable approach (on philosophical, scientific and practical grounds) is to identify the reasons for the large variation in empirical estimates and try to define what constitutes a reliable study and subsequent estimate of the VSL[228].

Although it is not possible to claim that the VSL selected for use in the thesis is a definitively precise estimate, it does supersede many of the shortcomings inherent in numerous VSL estimates. The thesis utilises the VSL result from one of the most credible and detailed studies available. Miller (2000) provides a summary VSL 'best estimate' derived from applying a detailed and robust statistical analysis to the most reliable existing VSL studies (for the UK)[229]. The initial stage of Miller's analysis was the identification of reliable VSL studies which are candidates for his aggregate VSL. These are shown in the table below.

Table 3.2: Range and estimate of statistical life values from the most credible studies for United Kingdom as selected by Miller (in thousands of 1995 $)[230]

Author / Year	**Type of VSL study**	**Estimated VSL**
Ghosh et al (1975)	Consumer Behaviour	1704
Jones-Lee et al (1983)	Contingent Value	3568
Jones-Lee et al (1995)	Contingent Value	2691
Maclean (1979)	Contingent Value	2446
Marin & Psacharopoulos (1982)	Wage-risk	2497
Melinek (1974)	Wage-risk	1457
Melinek (1974)	Consumer Behaviour	1608

[227] Ibid
[228] Ibid
[229] Miller, "Variations Between Countries in Values of Statistical Life", p. 172
[230] Miller, "Variations Between Countries in Values of Statistical Life", p. 176 -177

Hence, the studies in Table 3.2 were used by Miller to estimate the most plausible aggregate VSL value. This was achieved through a process of statistical analysis, which optimises the credibility of Miller's VSL results.

At the outset it was necessary for Miller to standardise all of the studies. This was achieved through ensuring that they were all measuring the same features (unique to each type of VSL derivation study) through regressing any inaccurate or inconsistent variables whilst maintaining the necessary features of the study, e.g. through identifying occupation specific risk from industry wide risk data. Moreover, all VSLs used in the Miller estimate have been standardised for currency, the differing value of currencies over time and tax, such that all units of value are in the same convertible format.

After standardising the studies Miller remedied one of the most persistent and obvious flaws in numerous VSL studies, which is that they account for actual risk rather than perceived risk, and catered for this through including an adjustment for risk misconceptions in his regressions. Along a similar vein, numerous labour market studies tend to utilise 'all-cause fatality risk' rather than 'work place fatality risk by occupation' and/or use 'fatality risk by industry' without including 'occupational indicator variables', which is often further exacerbated by use of average wage data. Miller's regression model estimates the impact of these problems and subsequently eliminates their influence. This is desirable as these flaws tend to artificially influence the value of the VSL: using 'all-cause mortality risk' tends to underestimate the VSL and failure to use 'occupational mortality risk' or occupational dummies (in regression analysis) tends to overestimate the VSL.

In addition to compensating for the problems that are evident in labour market revealed preference VSL studies, Miller applies similar considerations to revealed preference consumption VSL methodologies. The key problem here is that consumer behaviour studies tend to underestimate the VSL because these studies are based on (consumer) risk, which is typically under-perceived. Hence, labour market and contingent valuation studies tend to yield VSL estimates in a similar range, whereas consumer behaviour VSL results tend to be lower as a result of misunderstood risk. This is shown in Table 3.2, where, with the exception of the Melinek (1974) wage-risk estimate, behaviour studies yield the lowest VSL. This artificial influence is amended in the weighting of the estimates in Miller's aggregate best estimate VSL, e.g. Miller's best estimate VSL (which is a weighted average

of the VSL results in Table 3.2 [see Table 3.3] = 2,750 versus 1,608 and 1,704 which were yielded in the consumer behaviour VSL studies [see Table 3.2]).

Lastly, in a best effort to include the broadest range of credible VSL studies whilst yielding the most plausible aggregate VSL estimate for the UK, Miller has weighted the VSL studies contained within the overall estimate. Hence, the best estimate that is developed from the seven studies (outline in Table 3.2) has weighted these studies according to their type of VSL study, methodologies within the study and overall results. This optimises the plausibility of Miller's best VSL estimate as it ensures (as far as possible) that any artificial influences have been minimised.

The outcome of Miller's efforts to consider the greatest number of existing VSL studies and include them with optimal accuracy in an aggregate or 'best estimate' VSL yields results that are desirable for the thesis. This is largely because the objective of the thesis is to utilise a VSL which is roughly plausible, for twentieth century England. The provision of a weighted average of studies is desirable because it provides a broader consideration about what the VSL might be. The subsequent application of this broad range of studies to detailed statistical analysis is also desirable for the thesis because it helps to eliminate any sources of inaccuracy and artificial risk which would in turn yield a distorted VSL value. Finally, the inclusion of a representative for all three types of VSL study is also desirable as it goes some way in balancing out the advantages and disadvantages inherent in each type of study. Moreover, a probable reason for the difference in estimates yielded by the three types of VSL study is that they are measuring different risk functions and therefore having all of these represented (and weighted accordingly, by Miller) is desirable for the accuracy of the thesis' VSL.

A final appeal of adopting the VSL results of the Miller study is the versatility of the VSL results. It is possible to present the VSL as either an aggregate monetary estimate (shown in the table below as 2,759,000 [1995 dollars]) or as a VSL multiple (shown in the table below as 101, 128 or 154), which is then multiplied by GDP per capita, which yields the VSL estimate. The mechanics and appeal of using Miller's VSL multiple approach are outlined below.

Therefore, Miller's unique and credible efforts of finding an aggregate 'best estimate' VSL will be utilised in the thesis as a result of the likely accuracy of Miller's estimates. As a

direct result of Miller's procedure and analysis, the best estimate VSL used in the thesis is representative (as it was derived from a broad base of studies) and accurate (as far as possible due to detailed statistical analysis) which makes it ideal for the thesis' methodology. The best estimates that this intricate statistical analysis has derived are shown in the table below, which presents Miller's 'best VSL estimate' and a range of 'best VSL estimate: GDP multiples', which will be explained below.

Table 3.3: Miller's best VSL estimate (in thousands of 1995 $) and according VSL multiple (of GDP per capita) range for United Kingdom[231]

	Best VSL estimate	**Best VSL estimate: VSL multiple**		
Range		Low	Mid	High
United Kingdom	2750	101	128	154

Table 3.3 provides the results to Miller's analysis. There are two ways of thinking about the VSL (both of which are considering the same features). First, as an aggregate monetary estimate, which is shown in the table above as 2,750,000 (1995 dollars). The second way in which the VSL can be considered is as a function of i) a VSL multiple and ii) GDP per capita. In this second method, the VSL multiple (shown above as 101, 128 or 154) is multiplied by GDP per capita (precisely, GDP for the mid point of the period under consideration, is the value prescribed by this methodology to account for the average economic wealth during the period under consideration) which yields the VSL estimate. For example, for the period 1900 to 2000, 1950 GDP per capita will be multiplied with: 101 (low VSL), 128 (mid VSL) or 154 (high VSL). Or, for the period 1900 to 1925, the thesis will multiply 1913 GDP per capita by 101, 128 or 154. The key point here is that both types of VSL are accurate and identical in what they are measuring, although, when considering long and/or historical time periods the latter (VSL multiple) approach is superior. The reasons for this and the subsequent appeal of using the VSL multiple approach (which is the strategy adopted by the thesis' methodology) will be outlined below.

Therefore, in relation to Table 3.3 the VSL(s) that will be used throughout the thesis will be those that are derived by using the Miller VSL multiple, either 101 (low estimate), 128 (mid estimate) or 154 (high estimate), which will be multiplied by the associated GDP per capita number (which will be the level of GDP per capita [GDP pc] for the mid point of the

[231] Miller, "Variations Between Countries in Values of Statistical Life", p. 180

era under consideration), in order to identify the VSL for the different eras of the twentieth century analysed by the thesis.

When considering the value of the VSL that will be utilised in the thesis, it should be noted that the precise value of the VSL is not fundamental in influencing the overall findings of the thesis methodology. The thesis will apply a range of VSL estimates ('low' [101*midpoint GDP pc], 'mid' [128*midpoint GDP pc] and 'high' [154*midpoint GDP pc]) in order to reinforce that even if a low value for the VSL is considered, the twentieth century health gains have been extremely valuable and pronounced and contribute to the same overall conclusions as the high VSL estimate. Hence, because of the nature of the VSL in the thesis methodology combined with the magnitude of improvements in the death rate a VSL estimate that is in the correct region will be sufficient to provide an accurate approximation about the value of twentieth century mortality improvements in England.

When considering the validity of the thesis' VSL what is important is that the key issues of contention are recognised and that a range of estimates are chosen accordingly. Although it is possible to supersede some aspects of inaccuracy in VSL studies (as was achieved by adopting Miller's estimates, as outlined above) there are still inherent problems associated with estimating the VSL. The most prominent sources of VSL inaccuracies and contention are considered below.

A universal problem with estimating the VSL is the likelihood that individual rates of marginal substitution – and hence the VSL – will almost certainly differ from one cause of death to another and therefore it may not be appropriate to find one VSL for all situations[232]. For example, the UK Department of Health have estimated that willingness to pay for reductions in involuntary and poorly understood risk can exceed that of voluntary, familiar risk by a factor of up to two or three[233].

A problem associated with evaluating labour market risk is the underlying notion of this revealed preference approach: that riskier occupations can be expected to carry clearly identifiable wage premiums as compensating for risk. However, this cannot be accepted as a universal occurrence. For example, workers in high risk employment may simply be less

[232] Blomquist, "Estimating the Value of Life: Recent Developments", p. 28 in Jones-Lee, "The Value of Life and Safety"
[233] Department of Health, "Economic Appraisal of the Health Effects of Air Pollution", p. 3

risk opposed than average, or lack other economic opportunities[234]. Hence, some of the differences in the VSL are surely due, in part to different relative demands for occupational safety among the groups studied[235].

The extent to which wage premiums are aggregated for labour market conditions often reflects non-fatal as well as fatal risks. Lalive (2003) argues that industry aggregation of risk is responsible for the wide range in VSL estimates[236]. This shortcoming is particularly problematic from the perspective of the thesis because it creates the potential for double counting issues when the VSL (valuation for improvements in fatal risk) is combined with the VSHLY (valuation for improvements in non-fatal risk) in the overall QALE methodology. However, it should also be noted that this weakness has been amended as far as possible by Miller's statistical analysis which attempts to identify, convert and measure accurate risk.

There are also problems concerning model misspecification and risk data sets that are inaccurate for estimating the wage differential. For example, the omitted variables problem and the over aggregation of data are likely to produce estimates that are not precisely accurate. The inability to keep all variables separate (and avoid multicollinearity) is one of the greatest problems when trying to estimate the VSL[237]. Hence, it is necessary, but not always possible, to control for other factors in order to isolate the pure wealth risk trade off and the subsequent VSL[238]. Miller has adhered to these problems and overcome them as far as possible through his regressions.

A final limitation of the revealed preference approach is that it is inherently incapable of generating estimates at an individual level. The questionnaire approach is useful as it is able to avoid these major difficulties encountered by revealed preference studies. The questionnaire method is also a straightforward procedure for computing the VSL without distributional weighting schemes, an exercise that is extremely difficult under the compensating wage differential approach. However, contingent valuation (CV) estimates are viewed as less indicative than revealed preference, largely because survey respondents may have inadequate opportunities or incentive to accurately determine their trade-off

[234] Hodgson & Meiners, "Cost-of-Illness Methodology: A Guide to Current Practices and Procedures", p. 443
[235] Blomquist, "Estimating the Value of Life: Recent Developments", p. 28 in Jones-Lee, "The Value of Life and Safety"
[236] Lalive, "Did we Overestimate the Value of Health?"
[237] Hudson, "History by Numbers: An Introduction to Quantitative Approaches", p. 162
[238] Jones-Lee, "The Economics of Safety and Physical Risk", p. 54

between income and mortality risk[239]. The Miller VSL estimation process used in the thesis recognises this short coming of the CV method and weights CV VSL results accordingly. It should be noted that, when considering Table 3.2 and 3.3 the best estimate VSL (=2750) shown in Table 3.3 seems very close to the results of CV studies shown in Table 3.2 (=2691, 2446, 3568), which is a coincidence as the final VSL number is a function of a very detailed statistical analysis which consolidates a large number of weights and estimates.

An additional source of inaccuracy is generated through utilising a constant VSL value over time. This problem is exacerbated by the contention that exists in the literature regarding the nature of the VSL over time. Costa & Kahn (2003) have highlighted that as an economy develops, income, the quantity of safety and the health and well-being of the population also increase along with the demand for safety and the subsequent compensating wage differential[240]. For example, they estimate that between 1940 and 1980 the VSL increased by 300 to 400 percent, rising from roughly 1 million (1990 $) in 1940 to 5 million (1990 $) in 1980 in the USA, which indicates a VSL income elasticity of between 1.5 and 1.7 (estimated here as 1.6, see Table 3.4)[241]. Conversely, some studies have shown that there is an inelastic relationship between income and the VSL. Viscusi & Aldy (2003) also consider wage risk studies and conclude that the income elasticity for the VSL is less than 1 and they estimates income elasticity as being between 0.5 and 0.7 (estimated here as 0.6, see Table 3.4)[242]. There have been no studies, to date, for the historical VSL in the UK and it is therefore impossible to provide estimates which indicate the income elasticity and subsequent VSL over the twentieth century for England.

However, the thesis makes some progress in addressing this issue through utilising Miller's VSL multiple which is applied to GDP for the mid-point of the era being considered and is therefore dynamic to an extent, because changes in the wealth of an economy are accounted for with GDP per capita. Moreover, in utilising a constant VSL multiple the thesis is estimating income elasticity as unitary, which provides a mid-point between the divergences (of 1.6 elasticity and 0.6 inelasticity) in the literature.

[239] Hammitt, Lui, Lui, "Survival is a Luxury Good: The Increasing Value of a Statistical Life", p. 2
[240] Costa & Kahn, "Changes in the Value of a Statistical Life, 1940-1980", p. 1
[241] Costa & Kahn, "Changes in the Value of a Statistical Life, 1940-1980", p. 13
[242] Viscusi & Aldy, "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout The World", p. 44

The implication of these different elasticities is the variance of the VSL relative to GDP. Costa & Kahn's results imply that as an economy develops, increases in longevity or the VSL become more valuable (as the VSL rises 60 percent more than GDP or income, because the VSL income elasticity they propose is 1.6 percent, on average). This is in direct contrast with Viscusi & Aldy's result, which implies that earlier increases in longevity were more valuable (as the VSL rises approximately 60 percent less than GDP or income, because they propose that the VSL is inelastic by 0.6 percent). The implication of these theories (which are summarised in Table 3.4) is the relative magnitude of the VSL: if the VSL is income inelastic (Viscusi & Aldy), the value of the VSL is relatively large in earlier time periods, and the opposite is true for an income elastic VSL (Costa & Kahn), where the VSL becomes increasingly valuable as the twentieth century unfolds. The unitary elasticity (Miller utilised in the thesis) VSL estimates will lie between inelastic and elastic, respectively.

The table below provides a summary of the ramifications these different elasticities have upon the VSL. In Table 3.4 the VSL is derived from Miller (unitary elasticity), this VSL is calculated as the VSL multiple times GDP per capita for 1950 (mid point of 1900 to 2000). The alternative VSL values (for elasticity = 1.6 and inelasticity = 0.6) are derived through considering the Miller 2000 VSL value and adjusting this accordingly: identifying a VSL that has risen by 60 percent and 160 percent relative to income over the twentieth century.

Table 3.4: VSL value when assuming different levels of VSL income elasticity: Costa versus Viscusi, 1900-2000

Study	**VSL income elasticity**	**1900-2000 (= 1950) VSL value (millions)**
Costa & Kahn[243]	1.6	0.64
Viscusi & Aldy[244]	0.6	1.18
Thesis (Miller)[245]	1	0.88

In Table 3.4 the thesis VSL assumes a unitary VSL income elasticity. This is calculated through multiplying the VSL multiple (128) with GDP per capita for the mid-point of 1900 to 2000 (1950 GDP per capita = 6907, hence 6907 * 128 = 0.88 million = the VSL for 1900-2000 using unitary VSL income elasticity), in order to identify the VSL (this is the

[243] Costa & Kahn, "Changes in the Value of a Statistical Life, 1940-1980", p. 1
[244] Viscusi & Aldy, "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout The World", p. 44
[245] The Miller multiple used for the year 2000 was used as the base in deriving the VSL for alternative income elasticities as this is considered to be the most accurate VSL estimate, derived from Miller, "Variations Between Countries in Values of Statistical Life", p. 180

process of calculating the VSL using a VSL multiple approach, which has been outlined in Table 3.3).

The other VSL estimates in Table 3.4 are calculated by considering the Miller VSL for 2000 and adjusting this accordingly for elasticity of 0.6 and 1.6, in order to identify the VSL for these alternative elasticities, in 1950, which will make these results comparable with the thesis VSL shown above in Table 3.4. The Costa and Kahn estimates is the product of a VSL income elasticity of approximately 1.6 (average of 1.5 and 1.7), which indicates that the Miller VSL for the year 2000 estimate needs to be adjusted (in accordance with the Costa VSL change) in order to estimate a historical (1950) VSL with 1.6 VSL income elasticity (i.e. Miller 2000 VSL = 128 [VSL multiple] * 18714 [GDP per capita in the year 2000] = 2,395,392; 2.395.392 / 3.735 [percentage change in the Costa 1.6 income elasticity VSL] = 0.64 million). The same process is necessary in order to adjust the Viscusi and Aldy estimate so that the VSL has income inelasticity of about 0.6 (average of 0.5 and 0.7). Hence, the Miller VSL for 2000 estimate needs to be adjusted in accordance with the Viscusi VSL change in order to estimate a historical (1950) VSL with 0.6 VSL income inelasticity (i.e. Miller 2000 VSL = 128 [VSL multiple] * 18714 [GDP per capita in the year 2000] = 2,395,392; 2,395,392 / 2.026 [percentage change in the Viscusi 0.6 income inelasticity VSL] = 1.18 million). These results are shown in Table 3.4.

Another noteworthy point about Table 3.4 is the relationship between the thesis VSL and the alternative elasticity VSL results. The relationship between Costa & Kahn, Viscusi & Aldy, and the thesis VSL displays the properties that would be expected: income inelastic (Viscusi & Aldy) VSL estimates are higher in earlier periods than unitary elastic and income elastic (Costa & Kahn), respectively. This is a result of the VSL relationship with income, implied by these elasticities, such that for income inelasticity, earlier gains in VSL are more valuable and for income elasticity, later gains become more valuable.

Table 3.4 highlights that the VSL is influenced by the elasticity that is adopted and therefore the subsequent result (derived in the thesis) about the value of twentieth century improvements in mortality will also be a function of the VSL (and its elasticity to twentieth century income). However, as has been stated above, this does not cause an overruling concern for the thesis because the precise value of the VSL is not fundamental in changing the aggregate results, largely as a result of the magnitude of improvements in mortality

(and to a lesser extent GDP). I.e. whichever elasticity is adopted the results and conclusions of the thesis hold weight. This claim will be justified in Chapter 8.1 sensitivity analysis about the VSL (see Table 8.1.11), when the range of elasticity VSL values (shown in Table 3.4) are applied to the thesis methodology. Hence, the key value of Table 3.4 is to highlight the range of VSL elasticity theories in the literature and indicate how and why these will not overly influence the conclusions of the thesis.

An additional source of inaccuracy associated with a constant VSL, which is more consistent across all the literature and a problem for the thesis is the change in the age profile of the twentieth century English population. Hence, a dynamic VSL multiple is desirable to account for changes in the VSL during an individual's life cycle and the overall effect that this phenomenon has upon twentieth century increases in the proportion of elderly in the population. VSL studies that are based on hedonic wage model estimates, age-specific hedonic wage estimates and a minimum distance indicator estimate, have highlighted the effect of age upon the VSL through their findings that workers' VSL exhibits an inverted U-shaped relationship over their life cycle, such that the VSL for a 60 year old is likely to be less than half the value for 30 to 40 year olds[246]. The thesis overcomes this to an extent through applying Murray's age-weighting function (to the aggregate results) which also operates on an inverted U-shaped function (this will be explained later).

The aforementioned problems associated with deriving the VSL are significantly heightened when trying to assess the morbidity equivalent, namely the value of a statistical health life year (VSHLY).

3.2.3 Value of a Statistical Healthy Life Year (VSHLY)

The VSHLY follows the same rationale as the VSL, as it tries to establish the amount that a group of people (society) would be willing to pay for a reduction in the current period probability of ill health (instead of a death that which is estimated by the VSL). The VSHLY will estimate the value of illness risk reduction in the expectation of saving one healthy life year (of an undefined person) in the current period, and therefore indicate society's willingness to pay for improved morbidity (= VSHLY).

[246] Aldy & Viscusi, "Age Variations in Worker's Value of a Statistical Life", Abstract

Methodology

In contrast to the VSL literature, very little has been estimated about the value of a healthy life year. Furthermore, the existing initial attempts to consider some form of VSHLY tend to be abstract and generalised and do not consider the array of different illnesses.

Cameron and DeShazo (2004) who coined the term 'Value of Statistical Illness' (VSI), consider this as the rate of substitution between consumption and mortality/morbidity risk through evaluating the willingness to pay to avoid five (and only five) generalised mortality/morbidity states[247]. Although this provides an acceptable first effort to provide a more accurate VSI, there are noteworthy shortcomings. For example, the very general nature of the illness states and the void of considerations about the quality of life mean that the VSI is too generalised. Of greater concern is that some of the assumptions in their illness model seem incorrect, e.g. the 6 year survival profiles are arbitrary[248]. Finally, there is no indication about the change in the VSI, i.e. the associated trade-off costs as medical technology has advanced and the resultant improvement in the health/welfare quality of life associated with illness which is likely to have increased the value of the VSI over time.

A more general drawback of the majority of existing VSI methodologies is the assumptions that individuals are in one of two mutually exclusive states while alive: healthy or ill. The thesis WTP methodology will provide a much more detailed and bespoke evaluation about the burden of illness.

The thesis will provide a much more precise VSI estimate through the following process. The VSHLY used throughout this thesis will be a function of the VSL adjusted for the QALY (for the associated illness and era). The VSL considers a life year with 100 percent health and the QALY equals the necessary deduction of a healthy life year and hence the VSHLY will deduct the according QALY fraction for the burden of illness (blindness, cancer [breast and stomach] or tuberculosis in the context of this thesis). E.g. if the morbidity burden of tuberculosis in 1950 was 40 percent, an individual would only have gained 60 percent of a healthy life year (1 [full healthy life year] – 0.4 [morbidity burden] = 0.6 or 60 percent). The value of this healthy life year has been established above (it is the VSL) and this will need to be reduced by 40 percent for the morbidity burden in order to provide the VSHLY value. This calculation is summarised below.

[247] The five mortality/morbidity states that are considered: 1) shorter term morbidity with recovery, 2) longer term morbidity with recovery, 3) a combination of shorter term morbidity and premature mortality, 4) a combination of longer term morbidity and premature mortality, 5) immediate mortality. Cameron & DeShazo, "An Empirical Model of Demand for Future Health States when Valuing Risk-Mitigating Programs", p. 5

[248] For example, their rates of recovery do not seem to be consistent with the data concerning survival rates for certain cancers. I.e. the breast cancer (five year) survival rate in the USA (where the study was conducted) is greater than 60 percent, which is the recovery rate (six year) used in the study.

Equation 3.1: Summary definition of VSL and VSHLY used in the thesis methodology

$$VSL = Multiple_{VSL} * \text{[mid point]}\, GDP_{pc}$$

$$VSHLY = VSL * [1 - QALY]$$

Where, $Multiple_{VSL}$ = 101 (low) or 128 (mid) or 154 (high)

GDP_{pc} = the level of GDP_{pc} at the mid point of the period under evaluation

The VSHLY will also be considered as a range, containing 'low', 'mid' and 'high' VSL and QALY variables. Finally, it is important to reiterate that these indices do not have to be estimated with complete precision. The thesis will include a range of VSL and VSHLY estimates. All of these estimate values yield similar results that are acceptable for justifying the conclusion about the value of improved health in twentieth century England.

3.3 Methodology Application

As a result of the complexities associated with measuring morbidity and creating a model capable of doing this and the level of detail that is necessary to generate meaningful results, only a limited number of illnesses can be evaluated. However, these results will ultimately be extended in order to provide a lower bound estimate for the value of all health improvements in twentieth century England.

3.4 Illnesses Considered for the Morbidity Component

The morbidity component of the thesis will provide a catalogue of illnesses that have been selected within the rationale of the epidemiological transition, in an effort to optimise the results of the thesis. To this end, the thesis will provide a detailed (qualitative and quantitative) analysis of an infectious disease, tuberculosis and of a debilitating disease, cancer (represented by breast and stomach cancer). Blindness will also be considered in order to provide an indication about the twentieth century trends in disability.

Tuberculosis was selected because it represents one of the most important infectious diseases that declined during the twentieth century in accordance with the epidemiological transition. The prominence of tuberculosis in the twentieth century enables more detailed

quantitative considerations due to the availability of better data than alternative infectious diseases, for example, whooping cough and especially influenza.

One of the reasons for selecting breast cancer is the same as the appeal mentioned above for tuberculosis. Breast cancer was the most funded and researched cancer in twentieth century England, which facilitates a deeper insight into the quality of life features of this disease. Stomach cancer was utilised as a control for breast cancer and also to represent a more generic cancer, as far as possible. Breast and stomach cancer were also selected as their burden accords with the epidemiological transition, where debilitating diseases have increased to replace (and supersede) the decline in infectious diseases. This cancer trend is in contrast to the most prevalent twentieth century debilitating disease, circulatory or cardiovascular disease. The reason this has not been used is because of the decline of this disease from 50 percent of deaths in 1971 to 45 percent in 2000 (see Table 2.3.2). This contradicts the (epidemiological transition) framework of the thesis and would skew the results.

Blindness was selected because it represents a significant twentieth century disability. All disabilities, including blindness, have inherent measurement problems for the QALE (largely because they are not usually resolved in death and therefore prevalence is harder to estimate compared to diseases). However, blindness has been well defined and documented over the twentieth century which makes it a preferable proxy disability compared to alternatives like paraplegia or being deformed.

3.5 QALY Estimating Process

The QALY needs to be established in order to provide a standardised, numerical indication about the burden of morbidity (from the illnesses considered in the thesis) during different eras of the twentieth century. Essentially, the QALY is considering the portion of a healthy life year lost as a result of illness. The QALY will be presented as a number that is a fraction of one: where one represents a full healthy life year and anything between zero (which represents no healthy life year, i.e. death) and one is the fraction of a healthy life year lost to illness. The QALY number will move closer to one as the burden of disease is alleviated. This can occur for numerous reasons, which will be outlined below.

The QALY is fundamental in the methodology of the thesis because it provides the index for morbidity. The QALY forms the foundation of the morbidity and value of a statistical

healthy life year (VSHLY) variables, which will amend the mortality and value of a statistical life year (VSL) data. Therefore, without the QALY it would be impossible to gauge and estimate the numerical/monetary value of improved health and the thesis' aggregate health measure, QALE.

The QALY, in the context of the thesis, considers the quality of life associated with blindness, tuberculosis and cancer (breast and stomach) related to health and welfare. In order to establish this number it is necessary to provide a detailed and consistent analysis of all pertinent literature and data (relevant for the key health and welfare variables in the thesis). These considerations will also be made consistently across all the illnesses and eras considered in the thesis in order to provide a standardised series of QALYs. This is also necessary so that the QALE results for all illnesses can be combined to provide aggregate QALE results for twentieth century England. This will be achieved through applying the EuroQol standardised spectrum of development, which is explained below.

3.5.1 Key Variables

The key variables for consideration for each illness in the morbidity chapters of the thesis have been selected for their power as the most indicative and relevant aspects of quality of life related to health and welfare for sufferers.

Table 3.5: Key variables considered for all disease states in the thesis methodology

Key Variable	**Description/Significance**	**Example**[249]
Government initiatives and help	All government legislation, initiatives and benefits (financial and non-monetary) directed towards sufferers.	National Health Service Act 1948 and specific Acts of Parliament, e.g. 1922 Cancer Bill
Recognition and awareness	All factors related to public (i.e. non-governmental) sources of help. This largely includes the work of charities raising awareness and help for sufferers and also preventative campaigns.	Chest Clinic Samaritan Funds for the treatment of tuberculosis sufferers in Sanatoria before the introduction of government aid.
Health developments	Provides an aggregate score for the combination of the three most applicable health sub-variables and contains facets such as understanding, treatment, cure and prevention	The introduction of mammography screening for breast cancer and antibiotic therapy to treat tuberculosis.
Pain and discomfort	Provides a summary indication of the symptoms of a disease in conjunction with the associated medical and (to a lesser extent) welfare aid available to abate the condition.	The transformation of twentieth century therapy for tuberculosis.
Ability to lead a normal life	Provides an aggregate score for the combination of the three most applicable health and welfare sub-variables. These are more subtle and peripheral although equally as important as the aforementioned categories, e.g. depression and anxiety, physical ability, financial burden and social difficulties experienced by disease sufferers	The conditions of daily life associated with the prognosis and treatment of stomach cancer.

Each of these variables will be consistently evaluated from the perspective of tuberculosis and cancer sufferers in order to provide a detailed and standardised estimate about the quality of life burden of these illnesses for all eras of the twentieth century. For all of the variables in the above table the consideration point will be matched for tuberculosis, breast cancer and stomach cancer, with the exception of the sub-variables contained in 'health developments' and 'ability to lead a normal life'. These will be disaggregated for tuberculosis and cancer as outlined in the table below.

[249] It should be noted that the examples provided here are not exhaustive.

Table 3.6: Sub-variables for ‘health developments’ and ‘ability to lead a normal life’ variables in the thesis methodology

Morbid Condition	Key Variable	Sub-Variable	Sub-Variable Description
Cancer (Breast and Stomach)	Health developments	Prevention	Medical knowledge about potential predisposing factors for cancer, e.g. smoking.
		Screening	Ability to monitor individuals for cancer before they display symptoms, e.g. mammography.
		Treatment	Ability to abate (if not cure) the spread of cancer, e.g. chemotherapy.
Cancer (Breast and Stomach)	Ability to lead a normal life	Depression	The unhappiness associated with having such a potentially fatal illness and the distraction to normal life this can cause.
		Anxiety	The worry about the effects of cancer and treatment options.
		Physical ability	The disabling effects of therapy (whether successful or not) for the treatment of cancer.
Tuberculosis	Health developments	Understanding	The biological recognition and medical agreement about what the tubercle bacillus is, how it is spread and ultimately how it can be treated/prevented.
		Treatment	The discovery of successful therapy for relieving, curing and preventing tuberculosis and the burden that these different twentieth century therapy regimes had upon tuberculosis sufferers.
		Cure	The contribution of having a cure for tuberculosis and the associated boost to quality of life this yielded.
Tuberculosis	Ability to lead a normal life	Depression/Anxiety	The unhappiness associated with having such a potentially fatal illness.
		Social difficulties	The problems that arose out of isolation in the pre tuberculosis antibiotic era.
		Financial burden	The problems associated with the discontinuation of work that were caused by the tuberculosis disease.

It should also be noted that for blindness these variables are slightly different because of the different nature of disease versus disability. This is essentially because the emphasis on certain quality of life variables is different for a disability than it is for a disease. Although it would be possible to consider blindness in the context of the variables above (as it is still a form of health burden and therefore compatible), it would not be strictly accurate. The key variables for blindness are: government initiatives and help, recognition and awareness, health developments (prevention, treatment), status, ability to lead a normal life (education, employment, wages). The first two variables are identical to those evaluated for diseases (and shown as the first two rows in Table 3.5) and ‘health developments’ is

also comparable, just with slightly different sub-variables. The final three are more unique to disability and subsequently differ from disease. These are summarised below.

Table 3.7: Key variables considered for the disability state in the thesis methodology in addition to relevant variables in Table 3.5

Key Variable	**Description/Significance**	**Example**
Health developments	Prevention	Medical knowledge about potential predisposing factors for blindness, e.g. the effect of syphilis when giving birth. Antibiotic treatment for eye trauma that prevent eventual blindness, e.g. cortisone
	Treatment	Ability to cure causes of blindness, e.g. cataract surgery.
Status	Provides a summary indication of the actual effectiveness of blind legislation and recognition initiatives across all aspects of blind health and welfare related quality of life.	The extent to which anti-discrimination legislation was implemented and offenders prosecuted
Ability to lead a normal life	Education	The opportunities for the blind to be educated and instilled with an equal foundation to the able bodied for later employment and wages.
	Employment	The extent to which the blind experienced equal treatment with the able bodied in the labour market.
	Wages	The extent to which the blind experienced equal wages with the able bodied in the labour market.

Therefore, the above table highlights that the general considerations being made by the thesis are uniform across all illnesses, but that the most fundamental variables differ slightly between disease and disability.

After these key variables have been analysed in qualitative detail they will be transformed into a series of index numbers, which are representative of the quality of life associated with these conditions during different eras of the twentieth century. The first stage of this qualitative to quantitative transformation will be conducted on a standardised spectrum, referred to as EuroQol. For the actual transformation of the qualitative analysis, through applying EuroQol to the key variables for different illnesses and eras, into a quantitative (QALY) index, see Chapter 7.

3.5.2 EuroQol Grid

EuroQol has been used in numerous studies that try to yield QALY weights for a variety of medical conditions[250]. The appeal of EuroQol is the simple ranking spectrum that it

[250] EuroQol (2005). "What is EC-5D?", Retrieved 9 October 2005, from: http://www.euroqol.org/web/users/whatis.php

facilitates and the subsequent transparent comparison it provides, through establishing a two dimensional information medium. The first set of EuroQol dimensions is the five key variables (see Table 3.5). The second set of EuroQol dimensions is the values generated by a ranking scale, which, in the context of the thesis, assess the performance of these variables from a perspective of quality of life for the sufferer. For all illnesses and eras in the thesis there are six possible health and welfare ranks. By applying these rankings to the key variables for health and welfare the thesis will provide a standardised comparative analysis of all the illnesses and eras compared in the thesis. To summarise, the table below provides the (two dimensional) fundamentals of the EuroQol grid.

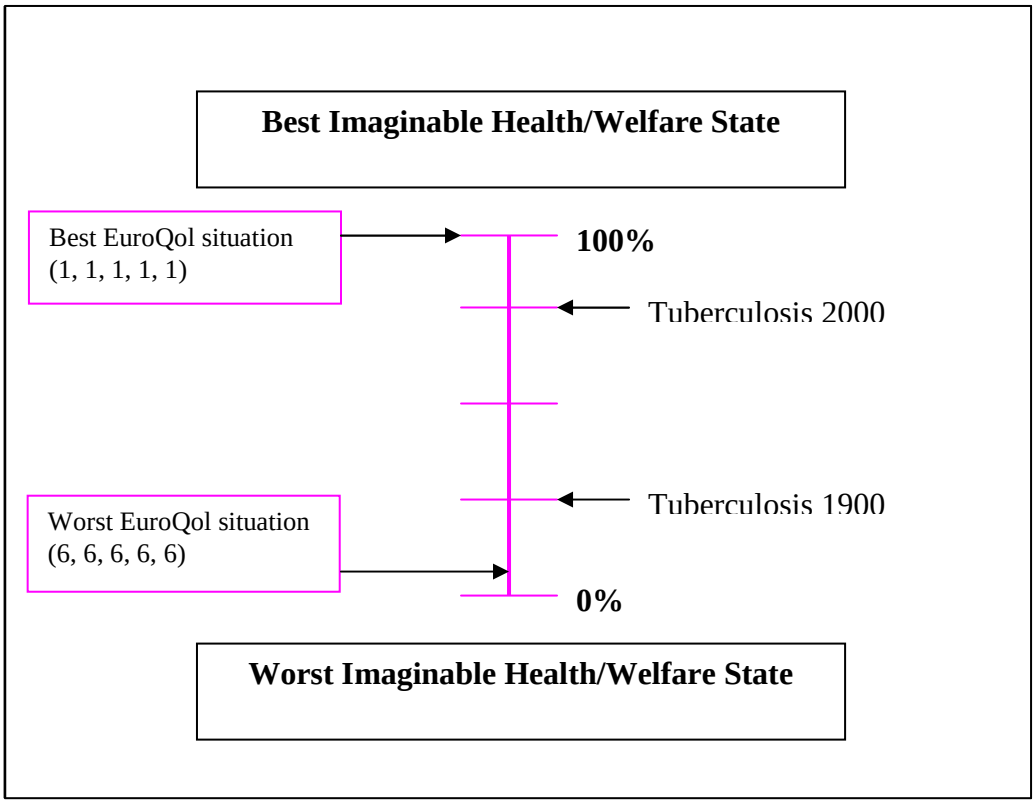
Table 3.8: EuroQol standardised spectrum: two dimensional ranking scale of the thesis methodology: all possible EuroQol variables, health states and ranks

EuroQol Variables (= EuroQol health and welfare variables)	
Government initiatives and help	
Recognition and awareness	
Health developments	
Pain and discomfort	
Ability to lead a normal life	
Status (disability only)	
EuroQol standardised spectrum **(= EuroQol evaluations of health and welfare variables)**	**EuroQol Rank**
Complete quality of life	1
Good quality of life	2
Fair quality of life	3
Some quality of life	4
Poor quality of life	5
No quality of life	6

Therefore, when considering the illnesses in the thesis, there are five key variables (top part of Table 3.8) and six possible (whole number) rankings that each of these can achieve (bottom part of Table 3.8), i.e. there are 5^6 or 15,625 different states of health and welfare, where the worst situation (of no quality of life for any of the key variables) would be 6, 6, 6, 6, 6 and conversely the best situation (or complete quality of life for all the five variables) would be 1, 1, 1, 1, 1. Once these scores have been established it will be possible to convert them into QALY numbers (a fraction between 0 = death and 1 = complete health

or 0.1 percent to 100 percent), which forms a key component of the QALE methodology and eventual thesis results. This is achieved through indexing the EuroQol 1 to 6 range of possible values into the equivalent QALY 0 to 1 range (this conversion and the corresponding EuroQol and QALY values are presented in Table 7.1). A simplified conceptual illustration of these considerations is shown in the figure below.

Figure 3.2: Simplified QALY ranking grid for tuberculosis 1900 and 2000



In the above example, which considers the improved quality of life associated with tuberculosis at the beginning and end of the twentieth century, the possible burden of tuberculosis (and any other illness as the core grid is standard throughout the thesis) can be anywhere between about 0.1[251] and 100 percent of a healthy life year. In 1900 the burden of suffering from tuberculosis was considerable and, in the hypothetical context of the diagram, an individual only experienced 25 percent of a healthy life year. By the year 2000, medical and welfare improvements had been considerable and improved the quality of life for tuberculosis sufferers to 75 percent of a healthy life year. These types of simplified ranking considerations will be made for all illnesses and all time periods studied in the thesis.

[251] The reason that the lowest score is 0.1 and not 0 is a result of the underlying inherent assumption in the thesis that any living state, regardless of how severe the burden of morbidity, is better than death. Hence, death is equal to 0 and perfect healthy life is equal to 100, it is theoretically possible to have a perfect healthy life but it is not possible to have an illness state that is worse than or equal to death.

3.5.3 Periodisation

The period that the QALE methodology will be applied to is the twentieth century, because during this era there were unprecedented increases in life expectancy and also when the epidemiological transition occurred in England. Although this era could be extended back in time, e.g. to the late nineteenth century, this would not change the overall findings or add any detail that is not captured in the early twentieth century.

In order to estimate how the health and welfare related quality of life evolved over the twentieth century it is necessary to analyse the key variables during different periods between 1900 and 2000. Because of the pace of health and welfare improvements, charting the changes for consecutive years would provide an excessive level of detail. The ideal approach is to consider health and welfare related quality of life at reference points throughout the twentieth century. To this end the twentieth century has been divided into quartiles: 1900, 1925, 1950, 1975 and 2000. The qualitative analysis will chart the improvements and breakthroughs that were necessary to change the quality of life at these points in time.

The actual process of this appraisal is much more dynamic, as the thesis analysis and results will consider the evaluation of health and welfare related quality of life between these reference points that contain the eras that are listed below.

1. 1900 to 1925
2. 1925 to 1950
3. 1950 to 1975
4. 1975 to 2000
5. 1900 to 2000

3.6 QALY Value Accreditation

The thesis utilises an unorthodox method for eliciting quality of life values for illnesses and it is therefore necessary to identify the conventional methods for deriving QALYs in conjunction with the reasons and appeal for the divergence between this and the thesis' approach. It is also desirable to highlight that the thesis QALY results do not bias the overall thesis findings, and show that in most instances the thesis QALYs are more conservatively estimated than those in mainstream measures.

Methodology

There is no uniformity in how a QALY should be measured and the most common methods for evaluating health are: expert ranking and individual self-rating of perceived health status, both of which can be aided by the use of tools such as a visual analogue scale (VAS), standard gamble (SG) and time trade off (TTO)[252].

The most established studies are Cutler and Richardson (1997, 1998, 1999)[253], who use a combination of expert judgement and individual self-rating to evaluate *"health capital"* and Murray's (1996)[254] global burden of disease study, which derives DALY (disability adjusted life years, which are essentially an inverse QALY) through an expert study that utilises VAS and TTO. The majority of credible studies which include the value of a QALY are based upon these methods and often provide refinements, e.g. age influence.

The most obvious incompatibility with these studies and the thesis is that they all tend to be based on modern national health questionnaires, e.g. the National Heath Interview Survey (NHIS) in America from 1969 onwards and the British Household Panel Survey in Britain from 1996. The QALYs generated through such methods are recent and therefore inadequate for considering the changing QALY between 1900 and 2000. This issue eliminates all types of QALY measures that rely on any form of self-reporting.

The next obvious alternative is the Murray type approach, of conducting an expert study to determine (through a series of revealed preference exercises) the likely QALY burden of different illnesses in different eras of the twentieth century. Although this approach has numerous attributes, it was felt that for the time and effort required to conduct an unbiased expert study was not productive, especially given the unavoidable drawbacks of such an approach, e.g. relativity among experts, participant biases which detract from subjectivity, and avoidance of issues about adaptation and fist hand understanding of the quality of life burden of illnesses. Moreover, although the expert study has 'consensus' as the compensating feature, it was felt that this did not add enough value for the thesis to conduct major research along the lines of an expert study to yield historical QALY values.

Therefore, as a first approximation, the QALY for the thesis illnesses and eras will be as well established by the author summarising the consensus of a detailed literature review,

[252] Praag & Ferrer-i-Carbonell, "Age Differentiated QALY Losses", p. 3
[253] Cutler & Richardson, "The Value of Health 1970-1990" and "Measuring the Health of the US Population" and "Your Money and Your Life: The Value of Health and What Affects It"
[254] Murray, "Rethinking DALYs", in Murray & Lopez, "The Global Burden of Disease", pp. 1-98

while appreciating all the potential factors of bias. The major sources of contention of this method are: author biases which detract from subjectivity and avoidance of issues such as adaptation and first hand understanding of quality of life burden of illnesses. Hence, these issues are very similar to those which arise from an expert study.

Because the thesis QALY is derived by the author's consistent review of all relevant sources these estimates escape other problems associated with numerous quality of life studies that are based on Cutler and Richardson methods, namely adaptation and relativity. Adaptation is defined by Heyink (1993) as *"an intra-psychic process in which past, present, and future situation and circumstances are given such cognitive and emotional meaning that an acceptable level of wellbeing is achieved"*[255]. As the prevalence of disease increased during the twentieth century, so did the biasing from 'adaptation'[256]. I.e. distortions arise because people's perception is standardised by their surroundings and experiences of themselves and those they know[257]. This is avoided by the thesis because the author is eliciting QALYs from unemotional, unphysical and unconnected experience, which eliminates the scope for adaptation and enhances the consistency (and subsequent reliability) of the thesis' QALY estimates.

In an additional effort to consider the validity of the thesis' unorthodox QALY generating process (which can be considered as a definitive survey of all existing relevant sources) it is desirable to consider the results that the Cutler and Richardson and Murray studies have derived for comparable periods as this will help to indicate the acceptability of the thesis results.

[255] Heyink, "Adaptation and Wellbeing", p. 1332
[256] Groot, "Adaptation and Scale of Reference Bias in Self-Assessments of Quality of Life", p. 406
[257] Ibid

Table 3.9: Comparison of QALY results from leading studies versus thesis (Hickson) estimates, for all available periods; 1970-2000

Illness	**Study**	**Range**[258]	**Year**					
			1970	**1975**	**1980**	**1990**	**1996**	**2000**
Blindness	Cutler & Richardson[259]		0.73		0.80	0.87		
	Murray[260]						0.60	
	Hickson	Mid		0.67				0.67
		Low						0.50
Cancer[261]	Cutler & Richardson		0.70		0.70	0.70		
Breast Cancer	Hickson	Mid		0.67				0.83
		Low						0.67
Stomach Cancer	Hickson	Mid		0.67				0.67
		Low						0.50
Heart Disease	Cutler & Richardson		0.64			0.70		
'Heart Disease'[262]	Hickson	Low						0.50

[258] For the purposes of the thesis; Mid represents what Hickson deems to be the most likely QALY value and Low represents the most conservative estimate that will be utilised for the extended results lower bound estimate
[259] Cutler, D. & Richardson, E. (1998) "The Value of Health: 1970-1990"
[260] Murray, "Rethinking DALYs", in Murray & Lopez, "The Global Burden of Disease"
[261] Cutler and Richardson's cancer estimate is obtained from the 'National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) database'.
[262] In the Extended Results of the thesis, all non-infectious and classifiable diseases are given the low profile of stomach cancer, and therefore this can be applied to heart disease for exemplary purposes. The rationale of this will be elaborated in Chapter 9: Extended Results.

The above table highlights the validity of the QALY weights yielded in the thesis, for the comparable periods of the twentieth century. The key evidence for this claim is two-fold. Fist, the similarity in estimates between the thesis and Murray and Cutler and Richardson, which indicates that the thesis QALY is making similar quality of life considerations and measurements. Second, the lowest bound QALY estimates yielded by the thesis add weight to the eventual conservative estimates of the thesis. However, it should be noted that this comparative substantiation is not available for the years prior to 1970, as these types of consideration had not been made. In an attempt to eliminate the challenges caused by this void in the literature, the thesis QALY has been developed from a detailed and consistent review of the literature in order to derive the most universally accurate QALY estimates. Moreover, this estimation process was consistent across all illnesses and eras considered in the thesis in order to yield the most robust summary of the QALY from all relevant historical sources.

Hence, in every comparable instance the thesis QALY weights are less favourable than Murray's and particularly Cutler and Richardson's. This is evident for the 'Mid' thesis estimates, which will be used in the Quantitative Results to provide a range and is deemed to be the most likely value by the author. The only exceptions to this are Hickson: breast cancer: 2000 versus Cutler and Richardson: cancer: 1990 and Hickson: blind: 2000 versus Murray: blind 1996. However, these are marginal and also there is the potential for the Cutler and Richardson and Murray estimates to have closed this QALY gap by the year 2000.

A final issue which adds extra validity to the thesis QALY estimates is the additional research related to QALY measurement a la Cutler and Richardson. These studies have attempted to add more precision to existing methods, for example, Praag and Ferrer-i-Carbonell (2001)[263] consider the effects of age upon QALY and Honda and Ohkusa (2001)[264] have developed a more rigorous model for defining quality of life, which includes economic consideration variables. All such studies have found a more optimistic value for the QALY than Cutler and Richardson, which implies that when more accuracy is attained the QALY burden declines, although estimates of magnitude are still lacking in the literature.

[263] Praag & Ferrer-i-Carbonell, "Age Differentiated QALY Losses"
[264] Honda & Ohkusa, "International Comparison of Subjective Health Evaluation – USA, UK and Japan"

Hence, the above analysis highlights that there is no mainstream literature that generates results and conclusions that are inconsistent with the thesis. Although the methodology used in these studies differs from that in the thesis, this is a result of the unique objectives of the thesis and the final QALY results of the thesis are in consensus with those yielded in established studies. Although it is not possible to justify in the pre 1970 era, every reasonable process has been employed to estimate the most accurate QALY values. Furthermore, compared to the estimates yielded by Cutler and Richardson and Murray between 1970 and 2000 the thesis results are conservative.

3.7 QALE Methodology

The measurement of mortality and morbidity will be combined in the quantitative methodology in order to provide an aggregate measurement for the value of improved health in twentieth century England. This overall (QALE) measure will provide an indication of the value of the decline in mortality in conjunction with the improvements in quality of life when ill.

Once this has been achieved for the thesis sample of illnesses the findings will be developed in order to provide a lower bound estimate about the value of improved morbidity in its entirety, which will be combined with all twentieth century mortality improvements to provide an aggregate estimate for the value of improved health or QALE for the entire twentieth century in England. This is facilitated by the creation of the thesis methodology, which provides the thesis' most significant contribution to knowledge, through facilitating estimates about the value and contribution (to a more rounded notion of economic welfare development) of improved QALE. This methodology is summarised below.

Methodology

Equation 3.2: Summary of the thesis methodology: quality adjusted life expectancy (QALE)[265].

Willingness to Pay Morbidity (MB):

WTP_{MB} Considers increased quality of life with an illness or disability →

WTP_{MB} = VSHLY * population weighted fall in the burden of disease / disability

For WTP_{MB} this would have to be calculated for each type of illness and disability and their associated QALY (λ)

This will then be combined with the equivalent information for mortality improvements

Willingness to Pay Mortality (MT):

WTP_{MT} Considers increased life expectancy →

WTP_{MT} = VSL * population weighted fall in the death rate

Quality Adjusted Life Expectancy (QALE):

Such that WTP_{MB} + WTP_{MT} = $QALE$ improvement:

$$\frac{dc^*}{d[\mu+\lambda]} = \frac{-u(c^*)}{(\rho+[\mu+\lambda])}$$

$u(c^*)$ = the goods value of life,

c^* = consumption or income,

ρ = the pure rate of individual time preference,

μ = set of mortality rates,

$\lambda = (\sum \Pr[\text{ Condition D at t + k}] * [\text{QALY for D at t + k}])$

Hence, the willingness to pay mortality represents the monetary value estimate for improvements in mortality and will be estimated through combining the decline in the death

[265] For detailed methodological algebra see Appendix 12.16.

rate with a value of this decline, which is represented here as the value of a statistical life (VSL). The willingness to pay morbidity represents the monetary value estimate for improvements in morbidity (through an improvement in quality of life associated with illness) and will be estimated through combining the decline in the burden of illness with a value of this decline, which is represented here as the value of a statistical healthy life year (VSHLY). These two measures will be combined to provide an overall indication about the (extended) willingness to pay for improved health which is depicted here as the quality adjusted life expectancy (QALE).

3.8 Methodology Sensitivity Analyses

Because of the numerous assumptions and estimates that were necessary to produce the overall results of the thesis it is desirable to try and accommodate for this through providing a range of possible results. The thesis will apply two broad categories of sensitivity analysis: the provision of a range of estimates, which will then be applied to an age-weighting function. These processes will provide an additional, more sophisticated set of QALE estimates.

3.8.1 Sensitivity Analysis: Range of QALE Gain Results

This sensitivity analysis will provide a range of aggregate estimates in order to overcome some of the uncertainty associated with estimating the QALY, VSL and VSHLY (which is a function of the VSL and QALY values) and subsequently add authenticity to the results.

The QALY will be considered with a degree of error of one EuroQol category in either direction. This will help to highlight the robustness of the overall conclusions. For example, the overall rank for tuberculosis in 2000 was 2 ('good' quality of life) on the scale of 1 to 6[266]. In the final results, the values 1 and 3 will also be considered. This range of (low [3], mid [2] and high [1]) QALY estimates will highlight that even if the QALY is not precisely 2, the results and overall implications of the results still hold weight. The VSL and VSHLY will also be subject to a similar range of estimates. Because the VSHLY is a combination of the QALY and VSL this will be subject to both of the aforementioned sensitivity analyses. Hence, the thesis will provide a range of VSHLY estimates which reflect low, mid, high VSL and QALY estimates.

[266] For the possible EuroQol ranks, see Chapter 3: Section: 3.5.2 *'EuroQol Grid'*

The above analysis has provided a detailed explanation about why there is such wide variance between VSL estimates and has identified a subsequent best estimate for the VSL. Although the value that has been selected avoids many of the associated problems, additional sensitivity analysis (the provision of a low and high as well as a mid VSL value) will be applied in an effort to further overcome inaccuracies. As well as providing a range of results, this analysis also recognises the likelihood that there is no definitively correct VSL estimate, and highlights that, regardless of the value utilised, the findings remain the generally unchanged.

The results of this sensitivity analyses process will be an extremely broad range of estimates, for example, instead of providing twenty QALE results (one QALE gain estimate for five eras and four illnesses) the thesis' sensitivity analyses will extend this series of QALE results to more than one thousand. The QALY, VSL and VSHLY sensitivity analysis will yield 27 estimates for each of the 4 illnesses and 5 eras (= [27*4]*5 = 540) the age-weighting will double the number of estimates for the three illnesses (= {[27*4]*5}*2 = 1080) and therefore there will be an aggregate number of 1080 estimates about the aggregate QALE gain in twentieth century England, all of which contribute to the same conclusion.

3.8.2 Sensitivity Analysis: Results Application: Age-Weighting

There is evidence that the VSL is not constant across all age groups and therefore a more valuable approach for estimating society's willingness to pay would be a methodology that considers the potential for different ages to have varying values. Barnum (1987) has indicated that relying on calculations of health effects that are un-weighted for different ages is not wholly satisfactory[267].

There are numerous perspectives associated with these calls for age-weighting. Some have highlighted that age-weighting can be conducted from an equity standpoint, where age-weights reflect the feeling that everyone is entitled to some normal span of life[268]. Anyone failing to achieve this has been cheated, while anyone getting more than this is 'living on

[267] Barnum, "Evaluating Healthy Days of Life Gained from Health Projects", p. 837
[268] Williams, "Intergenerational Equity: An Exploration of the 'Fair Innings' Argument", p. 119

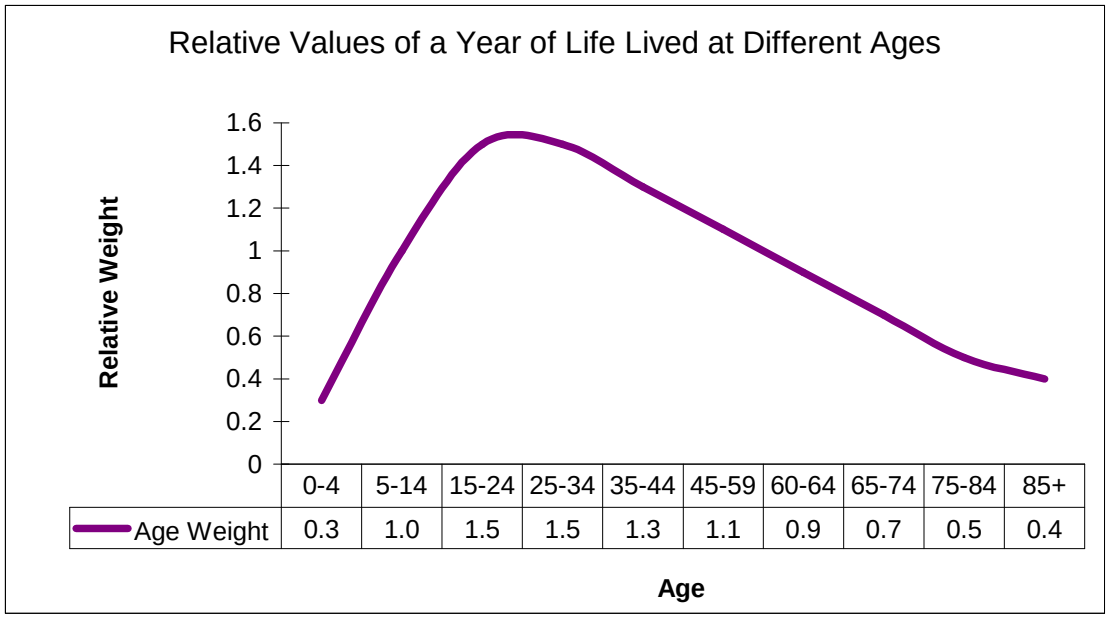
borrowed time'[269]. In this type of 'fair innings' argument, the younger population receive a higher age weight as they have completed a smaller portion of their normal life span[270].

At the opposite end of the spectrum, certain authors have identified the need to consider more indicators of economic contribution than just age. Cooper and Rice (1976) adopt a human capital approach to include income differentials of workers, where higher income groups who contribute more to total economic product are given a greater weighting, i.e. income weighted years of life[271]. Others have studied the extra contribution of more educated workers, and emphasised the importance of including differentiations between high school and college graduates[272].

The preferable – and more commonly utilised – method of age-weighting considers the relationship between age and efficiency, by reflecting an individual's social role, where people in general are supported by others during infancy and old age but support others during adulthood[273]. This form of age-weighting is provided by Murray (1996) in his Global Burden of Disease Study for the World Health Organisation (WHO) where he considers the age weight function of Disability Adjusted Life Years (DALYs). Murray provides values with which to calculate an age weighted value of death averted, supported by the notion that greater importance needs to be attached to years of productive adult life[274]. The continuous age weight function outlined by Murray is presented in the graph below.

[269] Ibid
[270] Ibid
[271] Cooper & Rice, "The Economic Cost of Illness Revisited" and Rice, "Estimating the Cost of Illness"
[272] Rosen, "A Theory of Life Earnings", p. S65
[273] Tsuchiya, "QALYs and Ageism: Philosophical Theories and Age-weighting", p. 61
[274] Murray, "Rethinking DALYs", in Murray & Lopez, "The Global Burden of Disease", p. 57

Figure 3.3: Murray's age weight function of DALYs: relative value of a year of life lived at different ages[275]



The appeal of this particular study is its versatility in conjunction with its power to highlight that improvements in health at different ages have different values to society. Furthermore, Murray's model is detailed and accurate enough to provide the necessary indication for the purposes of the thesis.

Despite the appeal of Murray's methodology there are some areas of unavoidable conflict which need to be recognised. In the context of the thesis there are evident problems with the static nature of Murray's age-weighting model. The economic contribution from different ages and genders is likely to have altered considerably over the twentieth century. Therefore, it would be ideal to have a different set of age weights for different eras of the twentieth century. As this option is not feasible, the Murray age-weighting will be utilised in order to provide a more accurate indication, rather than a definitive estimate, about the value of improved health, particularly compared to not making any age-weighting considerations.

[275] Murray, "Rethinking DALYs", in Murray & Lopez, "The Global Burden of Disease", p. 60

Methodology

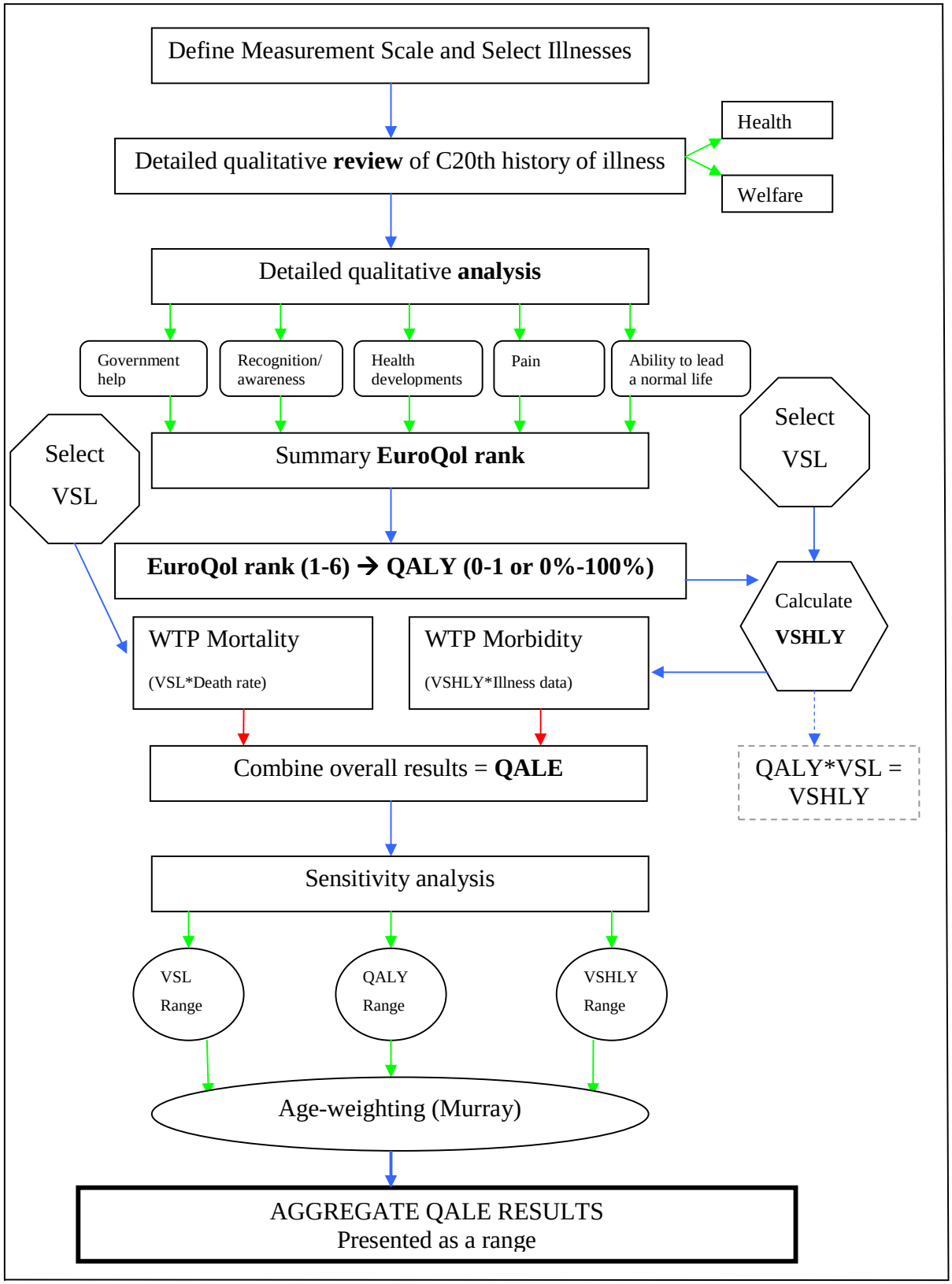
The age-weighting represents the final aspect of the thesis' QALE methodology. This process is summarised in the flow chart overleaf. The commentary below also provides a summary to the QALE methodology, outlined in detail above.

In the context of the methodological series in the flow chart below: defining a measurement scale was achieved through constructing the QALE and (its measuring tool) the two dimensional ranking scale, EuroQol. The illnesses that were applied to this measure were chosen within the rational framework of the epidemiological transition (the appeal of which has been outlined in Part I of the thesis). The detailed qualitative review will be conducted in Part II of the thesis in order to provide a transparent analysis (about the key EuroQol variables) for the reader. This analysis will also provide the justification for the EuroQol rank which will subsequently be transformed into an aggregate QALY for each illness and corresponding era (in Chapter 7).

Concurrently, the thesis has also identified the most credible VSL estimate. This process has been outlined previously in this chapter. This VSL can be combined with the change in the death rate in order to establish the WTP or value of improved mortality. Along a similar vein, the VSL can be combined with the previously identified QALY in order to provide the VSHLY. This can then be combined with data about the burden of illness (which is established through considering the prevalence of an illness in conjunction with the QALY for the corresponding illness and era) in order to identify the WTP morbidity, or value of improved health. The WTP mortality can then be summed with the WTP morbidity in order to identify the QALE gain.

For completeness and in an effort to provide the most robust results, this QALE gain will then be subject to a series of sensitivity analyses. The first will recalculate the QALE gain for lower ('low') and higher ('high') QALY, VSL and VSHLY values. This will provide a wide range of results which will then be applied to Murray's age-weighting function (see Figure 3.3).

Figure 3.4: Flow chart summary of the thesis' methodological process



Therefore, once this new health measure has been applied to the data it will enable a simultaneous evaluation about the value of improvements in the quality of life associated with morbidity (from a health and welfare perspective) and mortality. These considerations will be made for different points of the twentieth century in order to identify the extent and value of improvements in health and welfare related quality of life.

3.9 Extended Results

Once the methodology has been applied to the thesis illnesses (Chapter 8) the analysis will be extended in order to optimise the results of the thesis. The findings for individual (proxy) illnesses will be developed to provide an aggregate lower bound estimate about the value of improvements for the entire twentieth century morbidity (and mortality) burden (Chapter 9). This will provide the fullest evidence for the importance and meaningfulness of the claims of the thesis.

3.10 Methodology Location

A fundamental component of the quantitative thesis results is the qualitative evaluation of different illness states and the subsequent estimation of the quality of life burden (QALY) of the illness for different eras of the twentieth century. This will be achieved in detail in Part II of the thesis, after which it will be possible to summarise the key qualitative results and subsequently utilise this in the quantitative chapters, which form the results of the thesis.

PART II

4. Blindness

"Blindness is a disability that has far-reaching social, economic and developmental implications. When visual disability occurs at birth or in childhood it provides a challenge to development and learning. When it is apparent in working age individuals it lowers productivity and capability. Across the lifespan it increases the scope for inequality, poverty, discrimination, poor status and ultimately devaluation in the quality of life"[276]. Consequently the recognition and help provided by the government and society have very important implications for the overall quality of life of the blind, as do improvements in medical technology.

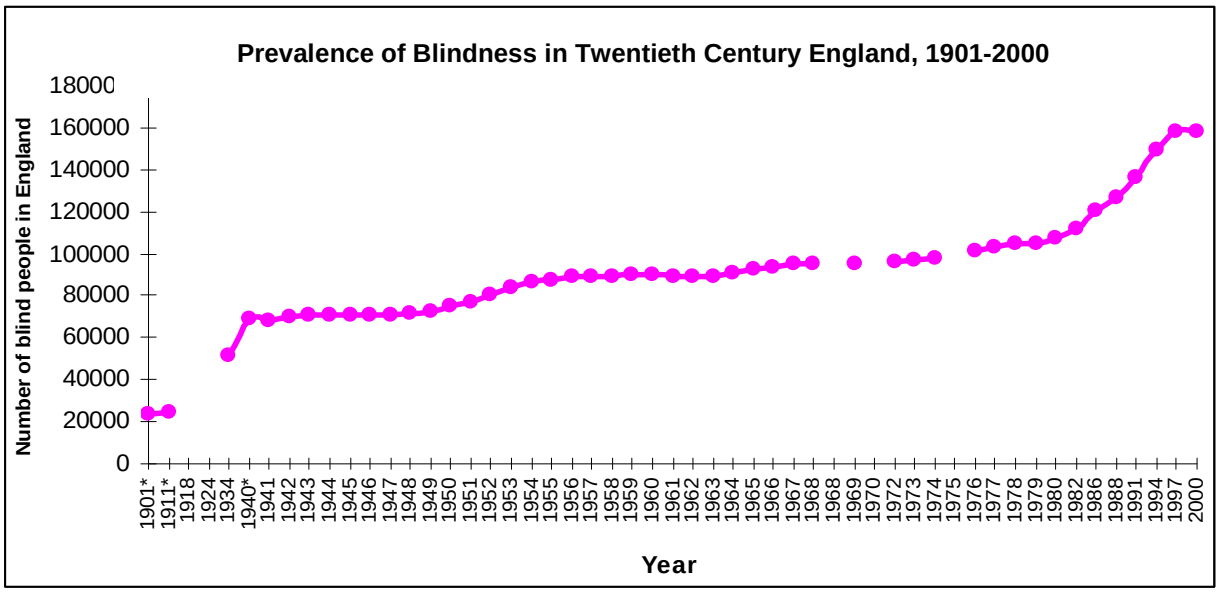
The number of blind people has been counted in Britain since 1851, starting with a simple declaration of blindness on census returns. This was discontinued after 1911, but after the 1920 Blind Persons Act, with its statutory benefits to the blind, a register of blind persons was established. This was refined during the 1930s with the introduction of the BD8 forms, which were accepted only if they had been signed by a recognised ophthalmologist. The legal definition of blindness, which has been used throughout the twentieth century, was *"so blind as to be unable to perform any work for which eye sight is essential"*[277]. This is usually considered as 3/60 vision or worse in better eye or 6/60 or worse in better eye with markedly restricted fields[278].

[276] WHO (2002) Article for world blindness day: World Health Organisation (2002), "Article for World Blindness Day". Retrieved 2 March 2003, from**:** www.who.int/dg/speeches/2002/geneva
[277] The National Archives: MH 55(74): 1933-1935 De-Certification of Alleged Blind Persons; this interpretation is based on the advice of the Ophthalmologic Section of the Royal Society of Medicine and from the Prevention of Blindness Committee
[278] Evans, J. (1995) "Causes of Blindness and Partial Sight in England and Wales 1990-1991", p. 57

Illnesses: Blindness

The number of registered blind people in England continually increased during the twentieth century, from approximately 23,907 in 1901[279]* to 75,420 in 1950[280] and peaking at 157,820 by 2000[281]. For the first half of the twentieth century this increase was a likely result of improved registration and not a genuine increase (at least not entirely). For the second half of the century the increase in the number was strongly affected by the growth of the elderly population (70 years and over).

Figure 4.1: Prevalence of blindness in twentieth century England, 1901-2000[282]



There was also a change in the age distribution of the blind population. The most common age of blindness increased so that by 1975 and especially 2000 this disability was mainly prevalent in old ages. This is an improvement over the first 50 years of the twentieth century when blindness was most common in working aged individuals and therefore hindered quality of life for a greater number of years. These types of consideration will be made at the end of this chapter (Table 4.9) and in the blind results chapter (Chapter 8.2). Before this is achieved, this chapter of the thesis will consider the key quality of life variables for blindness (see Tables 3.5 and 3.7) throughout the twentieth century.

[279] Census of England and Wales 1901
* NB: It has been highlighted (by the Parliamentary report of the departmental committee on the welfare of the blind 1917) that census returns do not provide a reliable indication of the total number of persons who are blind for practical and economic purposes.
[280] Government Papers: Local Authority Blind Returns
[281] Department of Health and National Statistics, Registered Blind Year Ending 31 March 2000
[282] Census of England and Wales and RNIB "Local Authority Blind Returns"

Hence, this chapter will highlight the welfare conditions faced by the blind during the twentieth century and identify the improvements in blind quality of life by examining three fundamental areas: legal, socio-economic and health.

The legal history encompasses government acts and monetary aid to assist the blind and also various other state initiatives to help compensate the blind for their handicap. These questions are important because legislation had the potential to help the blind obtain equal quality of life in most socio-economic domains of life and facilitate an improvement in their quality of life, despite their disability.

The socio-economic history will be evaluated in order to highlight the actual conditions faced by the blind in the major domains of their lives. This will include the following: employment and wages, education, and the status of the blind and the major issues of blind discontent during the twentieth century.

Medical developments will be considered in order to determine how effective and valuable preventative and curative technological breakthroughs have been for blind quality of life.

4.1 Government Initiatives towards Blindness

Table 4.1 provides a summary of the legal legislation that was provided to help the blind during the twentieth century. Table 4.2 provides a summary of the average income received by the blind (independently and due to government welfare). Many of these policies were insufficient for improving the blind quality of life. However, others were more far reaching and these will be discussed in more detail after the tables.

Illnesses: Blindness

Table 4.1: Government legislation for the blind 1906-1999[283]

Year	Title of government legislation	Provisions of government legislation
1906	Blind Aid Act	Established technical schools, workshops and home-workers schemes. Also provided limited financial maintenance for the unemployable blind.
1920	Blind Persons Act	The blind were recognised and helped more explicitly, welfare payments increased, pensions were introduced and numerous charities established.
1929	Local Government Act	Forced Local Authorities to donate funds to voluntary associations providing services for the welfare of the blind and to provide funds for other blind services – workshops, home teachers, hostels, libraries, etc. Local Authorities were also expected to augment central spending on the blind.
1934	Old Age Pensions	A reinforcement of pension policies under the 1920 BPA.
1938	Blind Persons Act	Forced Local Authorities to promote blind welfare. Improvements in aid for the blind. Pension age for the blind was lowered: from 50 to 40 years of age. The pension levels were the same as under the 1920 BPA.
1942	Scheme to employ suitable blind persons	Forced Local Authorities to encourage and facilitate the employment of blind persons in normal industry.
1948	National Assistance Act	Removed the blind from the Poor Law. Further provision for the disabled, in the form of increased welfare payments.
1966	Travel Concessions Act	Provided monetary concessions for the blind when using public transport.
1970	Chronically Sick and Disabled Persons Act	Extended the powers of Local Authorities to help disabled people and forced Local Authorities to publicise services to help the disabled.
1981	Education Act	Required the provision of special education for blind children to be conducted in normal schools (as far as possible).
1986	Disabled Persons Act	Promoted the inclusion of disabled people on committees and bodies, especially for issues concerning the interests of disabled people.
1992	Disability Living Allowance	Provided financial payments to the blind (£15 to £30 per week in 1992 and this increased since its introduction).
1992	Disability Working Allowance	Provided a 'top-up' for low earning disabled people who worked 16+ hours per week.
1995	Disability Discrimination Act	Made it illegal to discriminate against disabled people and introduced a quota system which forced large companies to employ a certain number of disabled workers.
1999	Disability Rights Commission Act	Established the disability rights commission, which helped to promote equal opportunities for the disabled.

[283] Information on the government legislation in Table 4.1 can be found in the corresponding Parliamentary Papers, The National Archives, and Beacon references in the bibliography.

Illnesses: Blindness

In addition to the provisions outlined in Table 4.1, there were also financial measures within some of the Acts. These are summarised below in Table 4.2, which highlights the average income of the blind from different sources during different eras in twentieth century England.

Table 4.2: Comparison of blind and able bodied sources of weekly income (per person), all values in new money, 1900-2000[284]

			1920-1921	1924	1933	1944	1948	1988	1992-1993	2000
Pension	Blind[285]		5p-50p							£40.40[286]
	Blind Soldiers[287]		94p-£6.00							£116[288]
	Able Bodied[289]			25p						£67.50[290]
Benefits	Blind		(>£1.38)[291]			£1.05-£1.75[292]	£1.25-£2.75[293]		£15-£30[294]	£35.80[295]
	Able Bodied								£44.65[296]	
Wages	Blind (mainstream)		50p-60p[297]			£3.49[298]		£134 (all disabled)[299]		
	Blind (blind industry)[300]	Industry		13p-£1.20	31p-£1.37	£3.27[301]				
		Home		65p	71p					
	Able Bodied	Semi-skilled[302]		£2.25	£2.35			£159 (all employed)	£240[303]	£385
		Un-skilled[304]	£2.11[305]	£1.99	£2.09		£5.44[306]			

[284] For elaboration on the figures provided in Table 4.2 see Appendix 12.13.3
[285] Parliamentary Bills, "Allowances under the 1920 Blind Persons Act"
[286] Non contributory pension: Department of Statistics (2006): "Weekly Rates of Social Security Benefit: Great Britain" Retrieved 18 May 2006, from: http://www.statistics.gov.uk/STATBASE/Expodata/Spreadsheets/D3989.xls
[287] Government Papers, "Reports of the Advisory Committee on the Welfare of the Blind"
[288] Department of Statistics (2006): "Weekly Rates of Social Security Benefit: Great Britain" Retrieved 18 May 2006, from: http://www.statistics.gov.uk/STATBASE/Expodata/Spreadsheets/D3989.xls
[289] Glennerster et al, "One Hundred Years of Poverty and Policy", p. 69
[290] Contributory pension: Department of Statistics (2006): "Weekly Rates of Social Security Benefit: Great Britain" Retrieved 18 May 2006, from: http://www.statistics.gov.uk/STATBASE/Expodata/Spreadsheets/D3989.xls
[291] Parliamentary Bills, "Allowances under the 1920 Blind Persons Act": the minimum amount of weekly income that the low level (£1.38) was supposed to exceed, although evidence suggests that it did not.
[292] The National Archives: MH 55(1098): 1943-1944 Blind Persons Treatment under Beveridge. The figures provided here represent a rough national average.
[293] Beacon 1948: National Assistance Rates
[294] The New Beacon 1995: Insight; Disability Working Allowance
[295] Department of Statistics (2006): "Weekly Rates of Social Security Benefit: Great Britain" Retrieved 18 May 2006, from: http://www.statistics.gov.uk/STATBASE/Expodata/Spreadsheets/D3989.xls
[296] Ibid
[297] Beacon 1919: State Aid for the Blind and Beacon 1930: A Record of Useful Work
[298] The National Archives: MH 55 (1089) : 1943-1946 Employment of the Blind: Placement in Industry
[299] This represents all disabled, and the blind only value is likely to be lower: Martin, & White, "The Financial Circumstances of Disabled Adults Living in Britain"
[300] Beacon 1924: Home Industries for the Blind and Beacon 1933: The Social and Economic Value of Home working Schemes
[301] Beacon 1924: Home Industries for the Blind and Beacon 1933: The Social and Economic Value of Home working Schemes
[302] Blind home worker wage data: Beacon 1924 and 1933 and mainstream industry wage data; Routh, "Occupation and Pay in Great Britain 1906-79"
[303] 1993 and 2000 wage: able bodied: Department of Statistics (2006): "Average Gross Weekly Earnings" Retrieved 18 May 2006, from: http://www.statistics.gov.uk/STATBASE/Expodata/Spreadsheets/D7937.xls
[304] Ibid
[305] Relative Values of Sums of Money (2006), "Average Minimum Wages paid to Ordinary Agricultural Labourers for Basic Hours, 1914-1980". Retrieved 7 March 2006, from: http://privatewww.essex.ac.uk/~alan/family/N-Money.html
[306] Ibid

Illnesses: Blindness

1920 Blind Persons Act

With the introduction of the 1920 Blind Persons Act, aid for the blind became, for the first time, widespread and specific. The provision of assistance under this Act was designed to ensure that no blind person was deprived as a result of their disability. The conditions of this groundbreaking Act were generally regarded – among the blind population – to be *"exceedingly wide and comprehensive"*[307].

Under Section 1 of this Act pensions were provided for blind men and women aged between 50 and 70 years of age. The rate of the pension ranged from 5 to 50 pence per week, dependent upon existing income. Additionally, the vast majority of Local Authorities provided another 50 pence, which provided an extra boost for blind pensioners' quality of life[308].

Although the National Institute of the Blind (NIB) did not regard the maximum under the Act as *"nearly adequate for the proper maintenance of those who were completely dependent upon the pension"*, they still recognised that it was a useful beginning[309]. The Advisory Committee on the Welfare of the Blind (hereafter the advisory committee) adopted a more positive view with their claims that the provision of pensions *"has provided a great boon to the unemployable blind, and has secured for them a greater degree of comfort than previously…and has done much to alleviate stress among unemployed blind people"*[310].

A further credit for Section 1 of the Act was the wide coverage from the onset of this legislation. In 1922 it is estimated that 93 percent of the blind aged between 50 and 70 were receiving the full pension[311]. Furthermore, for the first four years of the 1920 Blind Act pension, the blind were especially privileged compared to the rest of the population as there was no state pension before the Royal Assent in 1924.

Under Section 2 of the 1920 Blind Persons Act the scale of blind assistance *"was to be provided by Local Authorities; as it was thought to be a matter which should be determined by the local councils as representatives of the ratepayer who will meet the costs, and the Minister*

[307] Beacon 1920: The Editor
[308] Ibid
[309] Beacon 1920: The Editor
[310] RNIB, "Fourth Report of the Advisory Committee on the Welfare of the Blind 1923"
[311] Beacon 1922: Report of the Advisory Committee on the Welfare of the Blind

of Health would probably only criticise the scale if it was excessively low or high"[312]. The 1920 level of weekly income that the low amount had to exceed was £1.38[313]. The actual level of weekly payouts for the blind, around the midpoint of the twentieth century (1944), was between £1.05 and £1.75, with a median of roughly £1.35[314]. These payments also took into account the existing means (wages, pensions, charitable grants, investments, etc) of blind individuals who were applying for benefits. This represents an average blind welfare payout which was about 26 percent[315] to 40 percent[316] of semiskilled and unskilled wages, respectively, in 1944. In addition to these blind welfare payments, some Local Authorities also provided allowances for winter, rents, fuel, and other paraphernalia[317]. The government also provided a grant for the maintenance of blind services.

[312] The National Archives: MH 55(76): 1930-1934 Declaration of Schemes by Local Authorities: Blind Persons Act 1920
[313] Parliamentary Bills, "Allowances under the 1920 Blind Persons Act": the minimum amount stated is one pound, seven shillings and sixpence (this has been converted into new money)
[314] The National Archives: MH 55(1098): 1943-1944 Blind Persons Treatment under Beveridge: provides detailed lists of the amounts each Local Authority paid in blind relief. The figures provided here represent a rough national average. (£1.75 (35/-), £1.05 (21/-) and £1.35 (27/-) were converted from old money).
[315] Daily money wages of building craftsmen and labourers in Southern England, from "The Relative Value of Sums of Money", taken from Brown & Hopkins 1955: Wirksworth (2003), "Daily Wages of Building Craftsmen and Labourers in Southern England in The Relative Value of Sums of Money", taken from Brown & Hopkins 1955. Retrieved 10 March 2003, from: www.wirksworth.org.uk
[316] Average minimum wages paid to ordinary agricultural labourers, from "The Relative Value of Sums of Money", taken from Department of Employment and Productivity 1981 and Lund 1982: Wirksworth (2003), "Average Minimum Paid to Ordinary Agricultural Labourers in The relative Value of Sums of Money", taken from Department of Employment and Productivity 1981 and Lund 1982. Retrieved 10 March 2003, from**:** www.wirksworth.org.uk
[317] The National Archives: MH 55 (1098): 1943-1944 Blind Persons Treatment Under Beveridge

Illnesses: Blindness

Table 4.3: Grants from the Ministry of Health in respect of services provided for the welfare of the blind 1921 to 1930 (£s)[318]

Services	**1921 – 2**	**1922 – 3**	**1923-4**	**1926-7**	**1927-8**	**1929-30**
Counties Associations	6844	7002	7169	8428		
Workshops	31476	33356	35809	42212		
Home workers	6117	9212	10838	20007		
Homes	6673	6705	7409	7628		
Hostels	739	786	842	1001		
Home Teaching	12978	14753	16667	23209		
Tools and equipment	147	187	213	418		
Books and production	4912	3414	4440	6552		
Capital expenditure	-	30	63	3035		
Miscellaneous	-	-	20	20		
TOTAL	**69,886**	**75,445**	**83,470**	**112,510**	**120,550**	**131,368**

The advisory committee have highlighted that the total amount paid under the 1920 Blind Persons Act was: *"satisfactory, in as much as it represents the considerable addition in the number of services for the blind and also indicates that additional blind persons were constantly being brought within the scope of the services"*[319]. Table 4.3 highlights that nearly all the services show progressive increase during the twentieth century and the value of grants had nearly doubled between 1921 and 1930[320]. Another achievement of this Act was *"the efforts made by the workshops to keep blind workers fully employed during the times of economic depression"*[321].

[318] Beacon 1930: "Report of the Advisory Committee on the Welfare of the Blind 1923 – 1928"
[319] Ibid
[320] Calculation: 100-([69886/1313680]*100) = 47
[321] Ibid

This Act also catalysed the formation of numerous regional and national blind charities. Although the National Institute of the Blind (known as the Royal National Institute for the Blind since 1953) was established in 1899 (and since has represented the main vehicle for blind promotion and help) many more specialist and regional charities evolved out of the 1920 Act. These charities and voluntary agencies were generous with the support they donated to the blind population, and consequently they played a key role in enhancing the quality of life of the blind population during the twentieth century.

1942 Scheme to Employ Suitable Blind Persons

In 1942 the government introduced a scheme to employ suitable blind persons This government initiative forced Local Authorities to facilitate the blind joining normal employment. Within four years (by January 1946) *"this policy had been so well received by blind persons that there was a need to employ additional staff for the placement of blind workers in normal industry"*[322].

This enabled blind employment in normal industry, which represents one of the most important contributions towards blind equality with the able bodied population. *"There can be no doubt about the benefit to the individual blind person who has been found a place in industry. Many were now able to work for the first time since being diagnosed as blind, while others were being given the opportunity of doing work previously regarded as beyond their capacity"*[323].

1948 National Assistance Act

The blind population was not removed from the Poor Law until 1948 when the National Assistance Act was introduced. This Act made further provisions for the disabled, sick and aged. The scales of assistance of this Act ranged from £1.25 to £2.75 per blind person, dependent upon age. In addition to these basic rates, blind individuals also received financial help for their rents and mortgages.

[322] The National Archives: MH 55 (1089): 1943-1946 Employment of the Blind, Placement in Industry
[323] Ibid

Illnesses: Blindness

1981 Education Act

The 1981 Education Act required that *"the provision of special education of the blind be conducted in mainstream schools as far as possible"*, but did not abolish schools for the blind[324]. This indicates a step towards equality and integration of the blind. Although the effectiveness of normal schools compared to special schools for the education of the blind is contentious, the blind were still provided with the opportunity for equality, which facilitated an improvement in their quality of life.

1992 Disability Living and Working Allowances

The introduction of the Disability Living Allowance was an important development in welfare payments for the unemployable blind and in legal recognition and help for the blind in general. The Disability Living Allowance replaced the Attendance Allowance and Mobility Allowance, which were introduced in the 1970s but did not cater for the blind. This allowance paid blind individuals a weekly sum of between £15 and £30 (which increased later in the 1990s), regardless of any other income. This highlights increased recognition of the blind and improved financial aid for the needs of their disability, which would have enhanced their quality of life[325].

Also in 1992 the government introduced the Disability Working Allowance (DWA), which was designed to 'top-up' low earnings of disabled people who worked at least 16 hours per week. This indicates a further effort to recognise and cater for the needs of the blind in the labour market. Unfortunately *"when this policy was first introduced, the number of disabled people who received DWA was far lower than the official projections"*[326].

1995 Disability Discrimination Act & 1999 Disability Rights Commission Act

"The blind community experienced another milestone on the path to full equality and civil rights with the introduction of the 1995 Disability Discrimination Act"[327]. This legislation made it illegal to discriminate against blind people. It also forced companies who employed more than 20 workers to hire a certain number of disabled (including blind) individuals. Although this Act provided important rights for the blind, some areas were not covered and

[324] Government Papers, "1981 Education Act"
[325] The New Beacon 1995: Insight; Disability Working Allowance
[326] Ibid
[327] The New Beacon 1995: Statement from the RNIB

other aspects of the Act were poorly designed, which often left the blind without comprehensive and enforceable rights. For example, the Act did not apply to manufacturers or cover transport vehicles. The provisions of this Act were formalised through the creation of the Disability Rights Commission Act in 1999. Both forms of legislation provided an improvement in quality of life.

4.2 Socio-Economic Initiatives towards Blindness

The analysis below draws heavily on the reporting in the Beacon magazine, as a source of evidence about the standards of living experienced by the (overall) blind population. The Beacon was published monthly by the RNIB and was aimed at people with sight problems and also those who were involved with the blind. The typical content of a Beacon magazine would be a collection of topics that were relevant to contemporary blind issues. The tone of the beacon was upbeat, which is a likely result of the underlying objective of the Beacon, which was to empower, inspire and inform the blind. The Beacon should be considered as a reliable source, although some of the claims need to be interpreted with cautions of journalism and bias. Journalism in the language and possible over enthusiasm and bias concerning features related to the contribution of the RNIB and conversely, the government.

4.2.1 Employment

1900 → 1925

Blind employment initiatives were developed many centuries before the twentieth, however during this century the blind experienced the most significant developments towards equality with their able bodied counterparts. Home working began in the 1850s, as welfare schemes with aid from charitable funds. *"In the industrial circumstances of the time, very few blind people were ever able to become self supporting and so the pattern of supported employment was established. This pattern was redrawn on employment lines in the early twentieth century as a result of war time needs, which saw the development of workshops and the government established its factories for disabled ex-servicemen. Such arrangements continued to be envisaged as welfare or therapy rather than commercial employment and genuine rehabilitation"*[328].

[328] Royal London Society for the Blind & RNIB (1998) Supported Employment: Towards a National View

This attitude changed in around 1920 when: *"the great aim of those who had the welfare of the blind as their objective campaigned to get them into normal life, so that they could work shoulder to shoulder with their sighted colleagues"*[329]. The leader of the NIB had pointed out in 1918 that *"workshops throughout England and Wales employed less than 3,000 blind out of a possible 34,000"*[330].

Furthermore, the wages that blind individuals were earning at this time were insufficient. For example, there were 700 blind workers existing in London on less than 50 pence per week[331]. This is compared to the recommendation of the advisory committee that blind workers should be earning about one pound per week[332].

During the 1920s conditions improved somewhat for the blind who were seeking employment. There was an increase in the number of blind employed, for example by 1923 there were 8,246 blind people in employment (compared to 6,391 in 1918)[333]. Blind employment opportunities were further helped by the 1922 Ministry of Health grant of £20 per head for each home worker, to assist with: provision of material, sale of produce, and also to enhance the scheme so that home working could become a good career option for the blind. Although the average wages for home workers did not reach the recommended level they still provided the blind with employment and some form of economic compensation[334]. The extent to which this applies varied widely between occupations: in 1924 the highest weekly earning were £1.20 (piano tuners) versus the lowest weekly earnings, 13p (boot repairers). This gap was evident, although reduced, in 1933 when the highest weekly earnings were £1.37 (piano tuners) versus 31p (hand knitters). Full details of the range of weekly wages are shown in Appendix 12.13.3.

1925 → 1950

There was a slight increase in the wages of home workers during this time, however relative to the wages of comparable mainstream industries the blind were still not close to equality. This is highlighted by comparing the average wage of blind workers in home industries with the average wage for semi-skilled and unskilled mainstream industry, shown in Table 4.2, which

[329] Beacon 1920: Scope for the Blind in General Occupations
[330] Beacon 1919: State Aid for the Blind
[331] Beacon 1919: State Aid for the Blind
[332] Ibid
[333] Beacon 1924: Welfare of the Blind
[334] Beacon 1921: A Minimum Income for all Blind Workers

highlights that the blind only earned as much as 34 percent of their sighted counterparties between 1924 and 1935. This unequal situation motivated criticism from blind supporters, e.g. *"I have examined in the closest detail most of the systems of remuneration obtaining in the institutions for the blind in Great Britain, and, in my judgement, none of them can be regarded as entirely satisfactory"*[335]. However the home worker scheme is still thought to have been a success in other ways. *"Because of the increase in the number it employed and the extension of this service, many blind were capable of working and obtained training"*[336].

The advisory committee had stated that by the 1930s many blind people had been employed in ordinary factories and workshops. Furthermore, *"between 1910 and 1935 nearly every workshop for the blind had been enlarged or rebuilt and many more have been introduced"*[337]. As well as developing existing blind industries this helped foster new blind industries. For example, in 1900 flat machine knitting was a practically unknown industry for the blind and by 1932 it was one of the main industries for the employment of blind females. These developments contributed to the valuable 49 percent increase in the number of blind in employment between 1919 and 1930[338]. Wages also improved during this period where the average weekly wage paid to blind men in workshops increased from 60p in 1910 to £1.24 by 1935[339].

However, even though many blind persons had been employed in workshops, there were evident problems in finding employment for the younger working age blind. Furthermore, *"many of these workshops were too small to be efficient and about 60 percent of the blind workers would not have been employed if strict standards of productivity were enforced"*[340]. Furthermore, blind employment levels were geographically very uneven, for example the proportion of employed blind persons in 1931 varied from about 14 to 21 percent between different regions[341]. Also, out of the blind who were employed during the 1930s (and this is generally true for all times during the twentieth century), less than half were in workshops or under home workers schemes, which suggests that many of the blind were only casually or

[335] Purse,"The Blind in Industry", p. 76
[336] Beacon 1924: Home Industries for the Blind
[337] Beacon 1935: 1910-1935
[338] Beacon 1930: A Record of Useful Work
[339] Ibid
[340] Beacon 1924: The Employment of the Blind; A Survey of the General Problem
[341] Beacon 1931: Report of the Advisory Committee on the Welfare of the Blind

partially employed. Another area of inequality was evident between male and female employment of the blind, whereby women experienced discrimination.

Despite the problems with employment opportunities for the blind, the situation with wages continually improved during this era. Between 1938 and 1955 there was a substantial increase in normal industry wages and blind wages appear to have also experienced this increase, which marks a valuable achievement for the blind. This comparison is shown in Table 4.2.

1950 → 1975

In 1963 a new comprehensive service for placing blind people in industrial employment was introduced by the Ministry of Labour. During the first year of this scheme 921 blind people were placed in industry, of which 761 were placed in open employment, which the RNIB considered a *"good start"*[342].

Table 4.4: Employment of the blind: number and percentage of working age blind in employment and areas of employment, 1968-1975[343]

	1968	**% blind age working population employed**	**1969**	**% blind age working population employed**	**1970**	**% blind age working population employed**	**1975***	**% blind age working population employed**
Total Employed	9143	24	8969	24	8683	24	7837	31
In open employment	6269	17	6241	17	6123	17	5687	23

Table 4.8 considers the total number of blind in employment and the percentage of the blind working age population that this represents. This table highlights that despite the efforts of the Ministry of Labour, the blind employment rate failed to improve before 1975. This is best highlighted by there never being more than 24 percent of the blind working age population in

[342] The New Beacon 1965: Placement
[343] Ibid
* 1975 = average of 1974 and 1976 (1974 = 24933 and 1976 = 24843, aged between 16 and 64 → approximately 24888 at working age in 1975)

employment. However, the percentage of blind employed in general and in open industry by 1975 was a considerable increase from the previous levels.

1975 → 2000

"*Between 1991 and 2000 there is not thought to have been very much improvement in the percentage of the blind population who* [were] *employed"*, and between 1975 and 1991 the data show a considerable decrease in blind employment[344]. The RNIB suggest that "*the problem is one of discrimination and inadequate government help to train the blind and place them in open employment"*[345]. This was exacerbated by the decline in blind workshops (from 4,000 in 1949 to 1,500 in 1975).

Between 1985 and 1991 the RNIB conducted a major survey of blind and partially sighted adults in Britain, which identified that only 17 percent of the working age blind were in employment[346]. This is considerably worse than what the blind employment rates had been for the rest of the century.

Therefore, the above data indicates that there was still deep seated discrimination against the blind in employment. This is partly because "*there* [were] *still many situations where disabled people [could] be discriminated against legally"*, despite government legislation[347]. Most noteworthy is that only direct discrimination was prohibited which enabled the more subtle forms of oppression that were sustained by in-built or institutions patterns of inequality[348]. An additional source of inequality was between the blind and the able bodied population was that "*visually impaired people are less likely to be in professional jobs than non-disabled and disabled people in general"*[349]. This is summarised in Table 4.5.

[344] RNIB, "Work Matters"
[345] Bruce & McKennell, "Blind and Partially Sighted Adults in Britain: The RNIB Survey"
[346] Ibid
[347] French, "Disabled People and Employment A Study of the Working Lives of Visually Impaired Physiotherapists", p. 29
[348] Borsay, "Disability and Social Policy in Britain since 1750", p. 1
[349]French, "Disabled People and Employment A Study of the Working Lives of Visually Impaired Physiotherapists", p. 29

Illnesses: Blindness

Table 4.5: Occupational status of visually impaired people, disabled people and non-disabled people as a percentage of the workforce, 1990-2000[350]

	Professional	**Semi-skilled and Unskilled**
Visually impaired people	**14%**	**36%**
Disabled people	25%	31%
Non-disabled people	34%	23%

These problems in employment are exacerbated by labour market changes which should have boosted the ability of the blind to obtain employment. Throughout the twentieth century there was a shift in Britain's employment structure away from industry and towards manufacturing and the service sector, where the blind would have been more able to engage in employment. The increase in the size of the labour market between 1979 and 1999 from 24.5 million to 27 million should have helped provide employment for a higher proportion of the blind population[351]. During this time there was an increase in part-time temporary jobs, which suited the employment needs of the blind more favourably, and should have boosted blind employment. Finally, developments in workplace technology should have facilitated additional employment.

Despite the efforts of the government and charities the blind did not experience any significant reductions in the adverse effects of blindness in employment. *"In 1990, 53 percent of the visually impaired were living on less than half the national average income"*[352]. This is a strong indication of the poor quality of life that the majority of the blind consistently faced in twentieth century England.

4.2.2 Education

1900 → 1950

The provision of education was the most developed socio-economic aspect of blind welfare at the start of the twentieth century.

[350] Aggregate disabled population includes all disabilities. French, "Disabled People and Employment A Study of the Working Lives of Visually Impaired Physiotherapists", p. 34
[351] RNIB, "Work Matters"
[352] RNIB, "Blind in Britain: The Employment Challenge"

Illnesses: Blindness

The education of the blind has traditionally been conducted in special blind schools, which were funded by the government. In 1918 the government grant (which was increased during the twentieth century) was £10 per unit of average attendance in a certified day school for the blind and £16.50 in a boarding school, both per annum[353].

At this time it was generally considered that the special blind schools were the best place to educate the blind and that these schools were satisfactory at this task. Some even highlighted how *"blind schools were superior to a blind child attending a normal school"*[354]. There had been a continual improvement in the standards of these schools and by 1930 there was *"ample provision for the education of blind children and the facilities to educate them to the same extent as seeing children"*[355].

An alternative method for educating the blind was through home teachers. During the 1920s the number of home teachers began to expand rapidly: in 1924 there were 270 home teachers and by 1928 there were 390[356]. Despite the merits of home teaching, as technology advanced and special schools for the blind improved there was an increase in the number of blind children who attended school and a decline in the number who were taught at home.

There was also a decrease in the number of blind children who did not receive any kind of tuition. In 1921, 15.7 percent of normal blind children were not attending school. By 1930 only 10 percent of blind children were not attending school.

1950 → 2000

These improvements in education for the blind continued and are likely to have inspired the Warnock Report and the 1981 Education Act, which encouraged greater integration for children with disabilities into mainstream schools[357]. However, the blind were slow to enter mainstream schools and instead remained at special schools for the blind. *"In the year 2000 in England, Wales and Scotland, 59 percent of primary aged children and 46 percent of*

[353] Beacon 1918: The Board of Education
[354] Beacon 1922: The Blind and Education; a letter from the Chairman of the Standing Committee and Honorary Treasurer of the NIB
[355] Claims of a headmaster of a blind school in Beacon 1930: Helping the Blind
[356] Beacon 1924: The Welfare of the Blind, Beacon 1925: Advisory Committee on the Welfare of the Blind, Beacon 1928: Welfare of the blind Ministry of Health Report
[357] The New Beacon 1982: Integration of the Educationally Blind

secondary aged children with visual impairments attended mainstream schools"[358]. The majority of blind children who were attending mainstream schools were at infant schools and it is likely that they would have graduated to blind schools for their further education. The other type of blind pupil at mainstream schools was one-off placements.

Despite these developments in equality for educating blind children, the number of blind university graduates provides a depressing contrast as there was never more than 3 percent of the blind population (1969-1985) and usually only about 0.5 percent (1901-1985, mode average) attending university[359].

4.2.3 Status

1900 → 1950

The first improvement in the status of the blind was delivered under the 1920 Blind Persons Act. This was the first time that the blind had been exclusively recognised and helped for their disability. However, it was not until the Second World War (and after) that the blind experienced their most significant improvement in status. This boost in blind persons' standing was a result of their inclusion in the war effort, which continued throughout the rest of the twentieth century with an increased proportion of the blind workforce (albeit small) working in open industry. However, the blind standing, particularly in employment, was still not overly impressive (see Table 4.4).

1950 → 2000

As a result of continued problems in gaining employment and the failure of legal legislation to completely cater for the needs of the blind, their status did not reach optimum levels during the second half of the twentieth century. In the areas of recognition and help the status of the blind had improved considerably. These developments indicate that some of the burden of blindness had been alleviated. However, much of the help and treatment of the blind still seemed to be aimed more at welfare rather than rehabilitation, which was undesirable for the status related quality of life of the blind.

[358] Mason, H. & McCall, S. (1997) Visual Impairment, Access to Education for Children and Young People, p. 16
[359] Calculated from: Butler, "Visually Handicapped Studies: A Survey"

Illnesses: Blindness

An improvement in status was experienced by blind children with the introduction of the 1981 Education Act.

The 1986 Disabled Persons Act promoted the inclusion of disabled persons on committees, especially for issues concerning the interests of disabled people. This highlights another small improvement in the standing of the blind population. The 1995 Disability Discrimination Act provided equality between the blind and the able bodied populations. However, in actuality this did not provide equality, to the detriment of the status of the blind.

Therefore, as a result of better education, employment opportunities and various social improvements *"the status of the blind has improved such that the word blind no longer has unpleasant connotations, in the way it might have done at the start of the century"*[360]. However the blind population had not reached a point where their opportunities and treatment were equal to able bodied individuals. One of the key frustrations for this achievement was the experiences of the blind with regard to policy. As well as the government introducing more far reaching measures (which had been stated) it could also be argued that the blind charities could have taken a more involved and influential role in order to reiterate the precise legislative needs of the blind to policy makers.

4.3 Blind Discontent

Regional Inequalities

The organisation of blind welfare differed between Local Authorities. For example in 1944 there was nearly a 100 percent difference between the highest and the lowest levels of welfare payments to the blind[361]. The provision of other services for the blind were also unevenly distributed: in some areas there were many more provisions for home working, workshops and schools. The densely populated urban areas tended to lead with the provision of blind aid and the sparse rural areas were able to provide comparatively very little to their blind residents.

[360] The New Beacon 1980: Visually Handicapped or Blind?
[361] The National Archives: MH 55 (120): 1934-1935 Proposed Review of Circumstances of Unemployable Blind

Illnesses: Blindness

Preferential Treatment of Blinded War Veterans

Another area of discontent was the treatment of blinded ex-service men, who received substantial state pensions (shown in Table 4.2) and comprehensive retraining at St. Dunstans, which was the rehabilitation and training centre for blinded ex-servicemen.

There were approximately 2,000 of these war veterans who received this special treatment that was generally considered to be excellent[362]. Their benefits were considerably more extensive than the welfare benefits received by the rest of the blind population, for example, the maximum veteran pension was £6, which was twelve times greater than the 50p maximum for the normal blind, who interpreted this as a very unfair situation, and demanded *"an equal standard of living for all blind persons, the standard of living that is presently enjoyed by soldiers and sailors blinded in the Great War"*[363].

Perceived Inadequacy of State Help

Perceived inadequacy of state treatment of the blind is best measured by the demonstrations and marches of the blind. During the twentieth century these started in around 1915 as a result of demands for aid, which culminated in the 1919 NIB demonstration, which was supported by numerous trade unions, cooperatives and labour bodies. Their demands were to secure decent conditions of life and labour for every sightless person in Britain[364]. This process was re-enacted in 1920 when there was a march of about 200 blind people who were campaigning *"to highlight and improve the unsatisfactory social and industrial conditions faced by the blind and to demand that the government shall, without delay redeem its promises by providing the necessary financial arrangement"*. In 1936 there was another march of the blind, which was very similar in nature to the 1919 campaign. Finally, in 1990 there was a major demonstration by over 250 disabled groups and charities: of this group the RNIB were present on behalf of the blind. They were campaigning for a new system of benefits for disabled people, in order to bridge the gap between the living standards of the able bodied and disabled.

[362] Ibid
[363] The National Archives: MH 55 (607): 1936 Amendment to the Blind Persons Act 1920: Demands shouted by 500 or so blind protesters
[364] Beacon 1920: March of the Blind to London

Illnesses: Blindness

Also insightful is the stigma that was associated with blind welfare payments, which was evident throughout the twentieth century. Hence, as well as welfare payment levels being low there was a stigma in take-up, which meant that the welfare state failed to guarantee the blind against poverty and financial exclusion[365].

4.4 Medical Developments

1900 → 1925

During the first quarter of the twentieth century there was no knowledge about the causes and treatment of blindness, although it was known that much of childhood blindness was caused by venereal disease. The known causes of blindness and associated childhood prevalence of these causes are shown in the table below.

Table 4.6: Major causes of blindness in blind children as a percentage of the blind children population, 1913 -1991 (%)[366]

Cause	**1913**	**1920**	**1950**	**1991**
Ophthalmia Neonatorum	24	18	16	-
Syphilitic inflammation	30	31	-	-
Optic Atrophy	-	-	8	16

The table above highlights the proportion of children suffering from blindness caused by known congenital defects during the twentieth century.

Table 4.6 highlights that ophthalmia and syphilitic inflammation were both major causes of blindness at the beginning of the twentieth century and that by 2000 (and even the 1950s) these causes had been completely eliminated, which was achieved through the treatment of venereal disease. This was helped by the Local Government Board in 1914, when they made ophthalmia neonatorum a notifiable disease. The discovery of safe and effective antibacterial treatment in the 1930s eliminated venereal diseases. This decline was partially substituted with an increase in congenital optic atrophy.

[365] Borsay, "Disability and Social Policy in Britain since 1750", p. 168

[366] This list is not exhaustive. Beacon 1921: The Cause and Prevention of Blindness and Beacon 1923: Departmental Committee on the Causes and Prevention of Blindness and 1950 Privy Council Medical Research Council Memorandum Number 24: The Causes of Blindness in England and Wales and 1988-1991 DOH Statistics Bulletin

Illnesses: Blindness

1925 → 1950

By 1950 the key causes of blindness had started to change, from infectious diseases (e.g. syphilitic keratitis) to old age degenerative health problems (e.g. macular degeneration).

Table 4.7: Major causes of blindness in all ages of blind as a percentage of the blind population, 1922-1991 (%)[367]

Cause	**1922**	**1950**	**1991**
Cataract	16	25	3
Congenital defects	6	10	-
Glaucoma	9	13	12
Macular degeneration	-	6	49

The above table considers the proportion of blindness distributed among major causes. The most noteworthy is the decline in congenial defects. There was a significant increase in blindness due to macular degeneration, which is not surprising if it is seen within the context of the epidemiological transition. Also noteworthy in the context of the epidemiological transition is the trend in blindness experienced by cataracts and glaucoma: both of these are related to the change in mortality and the burden of disease. These both worsened around the middle of the century and improved by the end, which is a direct result of medical technological improvements that arrived by 2000 but not 1950, when these types of blindness associated with a degenerative disease environments became more prevalent.

1950 → 2000

Increasingly from the late 1950s the introduction of cortisone diminished the risk of blindness from numerous causes, from chemical accidents in industry to Iritis and Iridocyclitis (inflammation of the iris).

Also at this time, surgical treatment for cataracts was introduced and continued to develop throughout the second half of the twentieth century so that by the year 2000 curing cataracts was a very simple procedure, especially as by 1960 it had become possible to remove a cataract before it was fully mature (and causing complete vision loss). This development

[367] This list is not exhaustive. Beacon 1923: Ministry of Health Departmental Committee on the Causes and Prevention of Blindness and 1950 Privy Council Medical Research Council Memorandum Number 24: The Causes of Blindness in England and Wales and 1991 DOH Statistics Bulletin

explains the – inverted 'U' shaped – trend of the prevalence of cataracts during the twentieth century. In 1922 16 percent of the population (25 percent of the blind old aged population) suffered from cataracts. At this time there was no medical procedure that could alleviate the problem and eventual blindness was inevitable. The prevalence of cataracts peaked in 1950, at 25.4 percent (43 percent for old ages), caused by the ageing of the population without the medical developments to eliminate cataracts. By 1991, only 3.5 percent of the blind old aged population suffered from cataracts. This is a direct result of modern cataract surgery, and indicates the significant contribution of medical technology to blind quality of life.

Table 4.8: Blindness (caused by cataracts, glaucoma and diabetic retinopathy) in old aged blind as a percentage of the old age blind population, 1922-1991 (%)[368]

Cause	**1922**	**1950**	**1991**
Cataract	25.4	43.0	3.5
Diabetic Retinopathy	-	-	2.2
Glaucoma	29.8	18.0	12.9

Developments similar to those for cataracts have been made in the treatment of glaucoma, which also tends to be an old age disease. Blindness caused by glaucoma is a result of increased intraocular tension on the eyeball, which causes changes in the optic disk and affects vision. The solution to this disease is early diagnosis and adoption of appropriate treatment: eye drops and/or laser surgery. This can be achieved with regular screening, which was provided by the National Health Service (NHS) during the last quarter of the twentieth century for relatives of glaucoma sufferers who were are aged 40 years or older.

Finally, increased awareness about diabetic retinopathy (when the blood vessels in the retina enlarge and leak fluid which eventually causes blindness), evident during the last decade of the twentieth century, also provided a contribution towards the reduction of blindness. In the same way as glaucoma, retinopathy can be managed through regular screening, which is free on the NHS for diabetics (because of the close correlation of diabetes and vision disorders). However, there was still no curative treatment for glaucoma or diabetic retinopathy at the close of the twentieth century.

[368] Ibid

These developments created an increase in the average age of onset of blindness and a subsequent reduction in the average number of life years spent in blindness. This represents a significant improvement in the quality of life associated with blindness that is especially important to measure because it is a more subtle development.

However, estimating the average number of years in blindness is very difficult, due to a void of detailed blind data. There is no information about the age of onset of blindness and the only remaining alternative data about the prevalence of blindness only exists in 10 (sometimes 5) year blocks, which makes it impossible to determine the precise age of onset of blindness (particularly as prevalence is not equal to the contraction of blindness and the lower the frequency of data points for prevalence, the more pronounced this difference becomes between prevalence and contraction). Moreover, unlike the other illnesses considered in the thesis, blindness is not considered with survival rates and it is therefore impossible to determine the average number of years spent with blindness (per case of blindness). The combination of these problems means that the thesis is only able to provide a rough impression about the average number of blind years at different times during the twentieth century. Hence, the results yielded in Table 4.9 should only be considered only as a general indication about the likely number of years in blindness.

These estimates are derived through the following methodological process, which entails numerous assumptions that are also outlined below. The most common age of onset of blindness was identified for 1900, 1925, 1950, 1975 and 2000. This was achieved through reviewing the percentage of the population that were blind at different ages (the illustration of this is shown below in Figure 4.2.i and 4.2.ii). This often contained two (and three in 1950) reference points in an attempt to be more representative about the most common ages of onset of blindness. The fundamental assumption here is that a peak of blindness represents an increase in blindness and another subsequent peak will imply an additional increase in the number of blind. These assumptions are coupled with another, even more tenuous assumption, which is the estimate about the average duration of blindness from these peaks, in order to identify the average number of blind years in different eras. Because blindness was (generally) resolved in death, the age of onset of blindness in these peaks is considered relative to life expectancy at that age.

Illnesses: Blindness

Figure 4.2.i: Prevalence of blindness by age, 1900, 1925, 1950, 1975 and 2000[369]

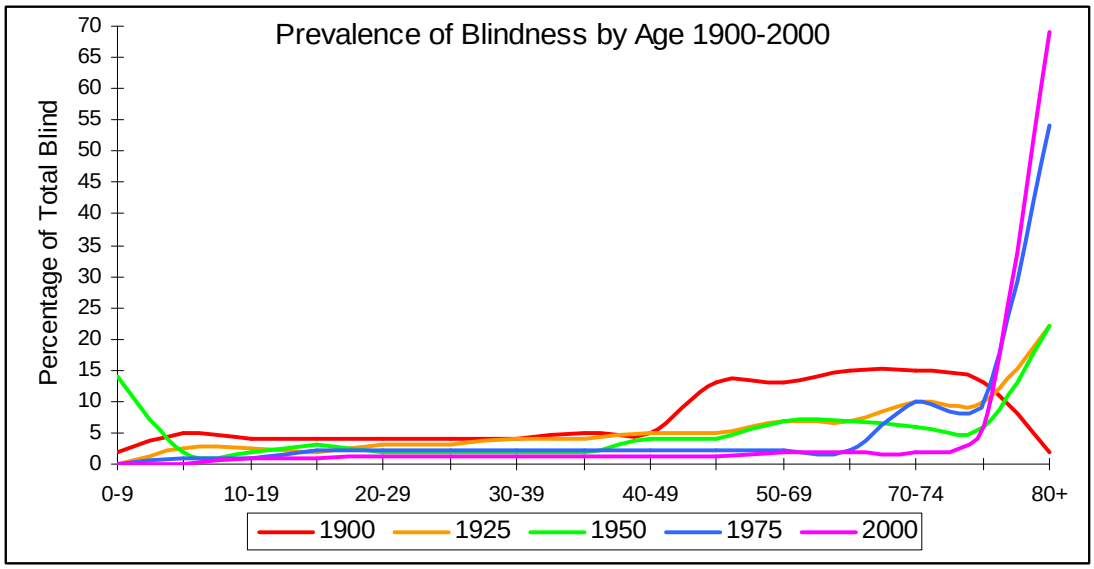
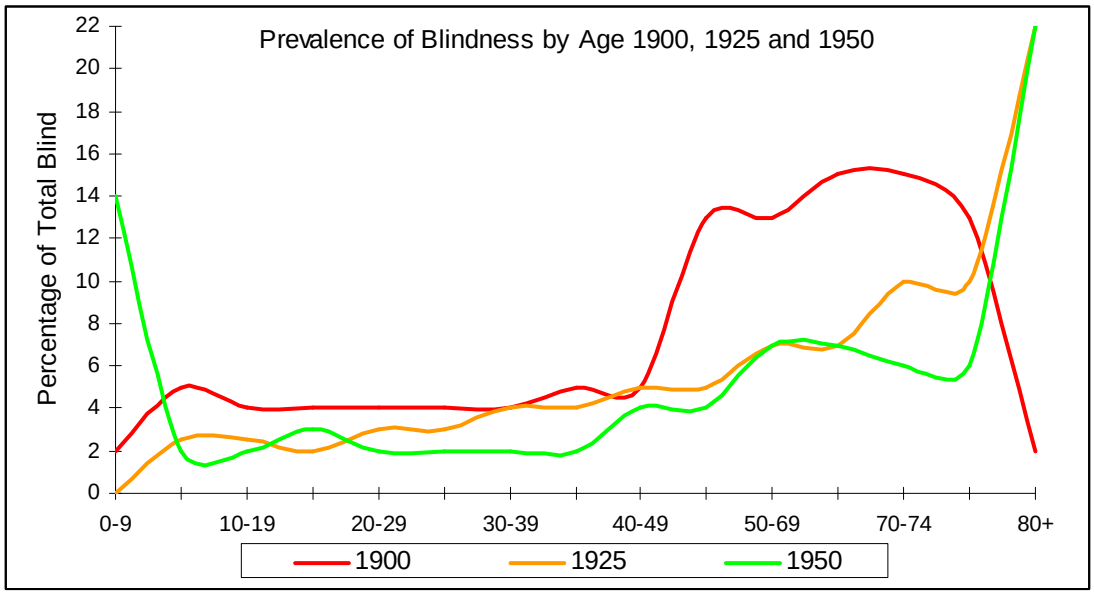


Figure 4.2.ii presents the same data but on a preferable scale.

Figure 4.2.ii: Prevalence of blindness by age, 1900, 1925 and 1950[370]



Hence, once these most common ages of onset of blindness had been identified this information was considered in conjunction with life expectancy by age. This was necessary in order to estimate the end point of blindness (through death) and subsequently identify the

[369] For a more detailed graphical analysis see Appendix 12.13.1
[370] For a more detailed graphical analysis see Appendix 12.13.1

average number of blind years. This methodological process identified the most common ages of onset of blindness, which is combined with life expectancy at the age of onset of blindness in order to provide an estimate for the average number of years in blindness. These results are shown in Table 4.9.

For completeness it is also desirable to consider the results in Table 4.9 in their wider context. This will be achieved in Table 4.10, which considers the proportion of the blind population that is represented in the most common age of onset of blindness. Table 4.10 will also develop this information one stage further through identifying the number of blind years that Table 4.9 implies. This will provide an indication about the average number of blind years during different eras of the twentieth century.

Table 4.9: Average number of years spent in blindness during the twentieth century[371]

Year	**Average most common age of onset of blindness 1 (Age)**	**Average most common age of onset of blindness 2 (Age)**	**Average most common age of onset of blindness 3 (Age)**	**LEx[372] Life Expectancy at age of onset of blindness (Years)**			**Average number of years of blindness 1 (Years)**	**Average number of years of blindness 2 (Years)**	**Average number of years of blindness 3 (Years)**
1900	35-55 → 45	65-77 → 71		LE_{45} = 23	LE_{71} = 8		23	8	
1925	55 → 55	70+ → 70		LE_{55} = 19	LE_{70} = 9		19	9	
1950	0 → 0	40-60 → 50	70+ → 70	LE_{0} = 68	LE_{50} = 24	LE_{70} = 10	68	24	10
1975	50 → 50	75+ → 75		LE_{50} = 26	LE_{75} = 9		26	9	
2000	75+ → 75			LE_{75} = 11			11		

[371] Calculated from; 1901: Census, 1925, 1950, 1975, 2000: Local Authority Blind Returns. See Appendix 12.13.1 for detailed calculation of the results in Table 4.11.
[372] Life expectancy by age data: Figures based on the 'Office of National Statistics/Government Actuaries Department' England and Wales mortality database. This is still subject to revisions. Extract was provided by Mita Saha (Office of National Statistics) on March 17 2006. See Appendix 12.15 for a copy of exert that was used here.

Table 4.10: Proportion (%) and number of the blind population represented in Table 4.9 and subsequent number of blind years from most common ages of onset of blindness

Year	**Blind at common age of blindness onset 1**		**Blind at common age of blindness onset 2**		**Blind at common age of blindness onset 3**		**Total number of blind years (from life expectancy at most common ages of onset)**
	Percent	**Number**	**Percent**	**Number**	**Percent**	**Number**	
1900	39	10069	33	8520			(10069*23=231587) + (8520*8=68160) = **299,747**
1925	26	10956	43	18120			(10956*19=208164) + (18120*9=163080) = **371,244**
1950	13	10572	24	19517	22	17890	(10572*68=718896) + (19517*24=468408) + (17890*10=178900) = **1,366,204**
1975	25	24900	54	53783			(24900*26=647400) + (53783*9=484047) = **1,131,447**
2000	69	108896					(108896*11) = **1,197,856**

Therefore the above tables have considered the prevalence of blindness by age for 1900, 1925, 1950 and 2000 and have reported on the peak episodes in column two, three and four, i.e. the 'common age of onset of blindness' in both tables. These have then been compared with life expectancy in order to produce the results in columns seven, eight and nine, 'average number of years of blindness' in Table 4.9. This last part of the calculation is derived through considering the average age of onset of blindness with the average length of life (life expectancy at age of onset of blindness - average age of onset of blindness = average number of years of blindness) in order to determine how many years are spent in blindness. These considerations in Table 4.9 enable Table 4.10 to generate more aggregate indications about the 'total number of blind years (from most common ages of onset)'. This table also indicates the depth of these calculations through identifying the percentage of the blind population that are included in the most common ages of onset.

Once this has been achieved, it is possible to illustrate that, although the trend has not been uniform, and although the trends are based on very ambiguous data and assumptions, there is an indication that the burden of blindness, in terms of the average number of blind years endured by the average blind person, improved between 1900 (about 16 years on average), the peak in 1950 (about 34 years on average) and 2000 (11 years on average). Conversely, Table 4.10 highlights a substantial increase in the number of people afflicted with blindness. Although this is a set back for improvements in blindness (related to prevalence), it does not detract from: i) improvements in the average durations of blindness and ii) other (aforementioned) quality of life gains for the blind in twentieth century England.

A final caveat for these findings requires a consideration to be made about cohort effects. This is necessary to add validity to the above claims about the increasing most common age of onset of blindness, as part of this increased average age of blindness is likely to be due to a cohort effect, as some of the 69 percent of the blind population who were aged over 75 in 2000 were also blind at ages 50+ in 1975. In order to provide the most accurate indication of the changes in the dynamics of the burden of blindness it is necessary to identify the extensiveness of these cohort effects. Table 4.11 considers the magnitude of this influence.

Illnesses: Blindness

Table 4.11: Cohort effects of increased average age of onset of blindness[373]

Year (T)	Most common age group	Period T-1	Age in period T-1	Number of blind in age group in T	Number of blind in age group in T-1	Cohort effect (%) % in T that are from T-1
1900	-	-	-	-	-	-
1925	16-64	1900	1-39	24723	8676	35
1950	16-64	1925	1-39	39472	12027	30
1975	75+	1950	50-60	54638	11394	21
2000	75+	1975(6)	50-65	108360	11179*	10

In Table 4.11 the most common ages of blindness are identified (and presented as a broader age group than in Tables 4.9 and 4.10) and are then considered for their relationship with the number of blind in the previous era, T-1. For example, in 1925 the most common age of onset of blindness was between ages 16 and 64 (this has been conservatively estimated as 55 in the tables above). However, this could be a result of earlier generations' blindness, e.g. if blindness was contracted between ages 1 and 39 in 1900. Therefore, to decipher the extent of this effect, the thesis considers the number of blind in aged 16-64 in 1925 (T), relative to the number of blind aged 1-39 in 1900 (T-1), in order to identify the percentage of blind who could be part of the cohort effect.

These considerations about the extent of spill-over (from period T-1 to T), which are made by the thesis in order to assess the accuracy of the average number of blind years as considered in Table 4.9 and 4.10 show that in most eras there was an evident cohort effect but that this was not far-reaching enough to over turn the trend in the reduction of average blind years. The most substantial cohort effects were in 1925, where 35 percent of the blind were also blind in 1900, versus 2000, which had the lowest cohort effects, where only 10 percent of the blind in 2000 had also been blind in 1975. The results of this analysis indicate that although there were evident spill-over or cohort effects, these were not large enough to drastically change the overall findings about the increasing age of onset of blindness, and the contribution that this had towards improving the quality of life of the blind, especially by the year 2000, when these

[373] Calculated from: Census of England and Wales 1901, Government Papers: Local Authority Blind Returns. See Appendix 12.13.2 for elaboration.

* For 1975 there is no data of enough detail. This estimate was derived from estimating the proportion of the blind population aged 50 to 65 out of the 16-65 age groups. In 1950 and 2000 (for which detailed data exists) the 50 to 65 age group = 45 percent of the entire 16 to 65 age group. Applying this to 1976 → 16 to 64 age group = 24843*0.45 = 11179

cohort effects were least significant. The consistently declining magnitude of the cohort effect also adds weight to earlier indications about a decline in the average number of years of blindness. Finally, the key value of Table 4.11 is that it substantiate earlier judgements related to the quality of life associated with (the average number of years of) blindness in twentieth century England, which marks an important improvement and a valuable development, which will be analysed further in Chapter 8.2, blind quantitative results.

4.5 Summary

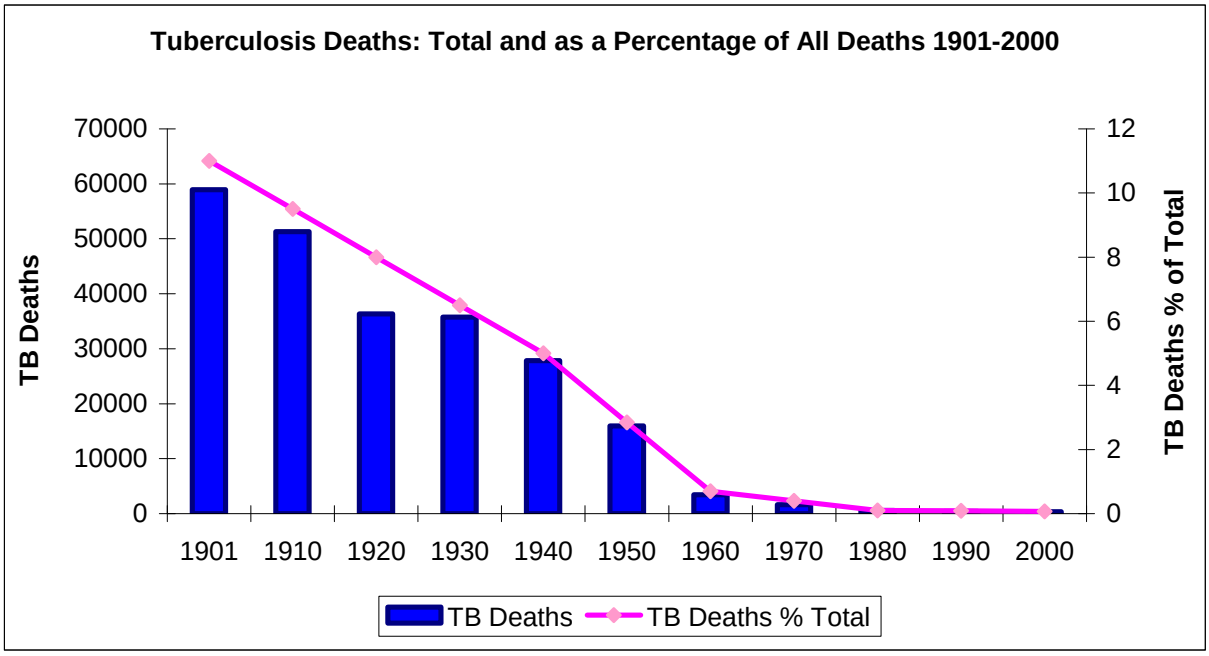
By the end of the twentieth century socio-economic standards of living for the blind had improved to levels which were regarded as good by some and adequate by most, and consequently the burden, discomfort and unpleasantness of blindness had been somewhat alleviated. The result of this was an improvement in the quality of life for the blind during the twentieth century. The developments in government legislation, blind peoples' recognition and status and a move towards equal rights and opportunities in employment and education have generated improvements in blind welfare, such that by the close of the twentieth century the blind had a satisfactory quality of life. However, there still remained considerable scope for improvement in the health and particularly welfare related quality of life associated with blindness. The extensiveness and value of these twentieth century changes in blind quality of life will be summarised in Chapter 7.1 and quantified in Chapter 8.2.

5. Tuberculosis

During the twentieth century, Britain experienced a significant decline in the burden of tuberculosis. In 1901 tuberculosis was responsible for 11 percent of deaths and by 2000, only 0.07 percent[374]. This is illustrated below in Figure 5.1, which considers the decline in tuberculosis (blue bars and left-hand side Y axis) and this tuberculosis decline relative to all deaths (pink line and right-hand side Y axis).

[374] Calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

Figure 5.1: Tuberculosis deaths: total and as a percentage of all deaths, 1901-2000[375]



The reduced tuberculosis mortality (shown in Figure 5.1) has been accompanied by a decline in morbidity, as measured by the tuberculosis notification rate. Table 5.1 highlights the course of the 90 percent decline in the prevalence of tuberculosis between 1915 and 2000[376].

Table 5.1: Total number of notifications for respiratory tuberculosis, 1915-2000[377]

Year	Notifications	Year	Notifications
1915	70,000	1955	33,000
1920	58,000	1960	21,000
1925	60,500	1965	12,000
1930	54,000	1970	9,000
1935	45,000	1975	7,000
1940	35,000	1980	6,000
1945	41,000	2000	6,087
1950	42,000		

[375] Ibid
[376] The Standing Medical Advisory Committee for the Central Health Services Council, "Tuberculosis: Epidemiology and Control", p. 3
[377] 1915-1980: Citron et al, "Tuberculosis Today", p. 6. 1990: Watson et al, "Notifications of Tuberculosis in England and Wales, 1982-1989", p. R13. 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 887

Although there is widespread agreement about the significance of this decline, there have been numerous debates about the key features for the virtual elimination of tuberculosis by the close of the twentieth century.

This chapter will highlight the health and welfare conditions faced by tuberculosis sufferers during the twentieth century and identify the changes in these conditions and ultimately the improvements in quality of life associated with this disease. This will be achieved through a detailed consideration of the key features associated with the health and welfare experiences of tuberculosis sufferers.

The chapter will begin by providing a background on this disease, in order to highlight vividly how improvements in the prognosis of tuberculosis affected quality of life. The thesis will provide a detailed explanation of the changing aetiology, pathogenesis and prognosis and the changes in related courses of therapy associated with tuberculosis in twentieth century England. This will be followed by an outline of the statistical detail related to the mortality and morbidity burden of tuberculosis during different eras of the twentieth century.

After this has been achieved this chapter will provide the twentieth century chronology that details the efforts made by government, charities and medical technology in the fight to treat, aid and cure tuberculosis. This section will concentrate on evaluating the outcome of these efforts for their contribution towards improving the quality of life for tuberculosis sufferers.

After the improvements have been analysed it will be desirable to identify the areas where there has been a comparative lack of progress in improving quality of life for tuberculosis sufferers in twentieth century England. This will provide the final necessary details to enable comprehensive conclusions.

5.1 Aetiology and Prognosis

5.1.1 Definition

Tuberculosis is an infection with the bacterium Mycobacterium tuberculosis (hereafter M.tuberculosis), which most commonly affects the lungs (in at least 75 percent of cases),

where it is called pulmonary tuberculosis[378]. Extrapulmonary sites include the pleura, central nervous system, lymphatic system, genitourinary system, and bones and joints. An especially serious form is disseminated or miliary tuberculosis, which is more common in immunosuppressed persons and in young children. Pulmonary tuberculosis can co-exist with extrapulmonary tuberculosis. Additional terminology for tuberculosis are shown in the table below[*].

Table 5.2: Common synonyms for tuberculosis[379]

Synonym	**Rational for synonym**
Consumption	Because tuberculosis seemed to consume people from within with its symptoms of bloody cough, fever, pallor, and long relentless wasting.
Wasting	
White Plague	Tuberculosis sufferers tended to appear markedly pale.
Phthisis	Greek for consumption.
Phthisis Pulmonalis	
Scrofula	Swollen neck glands.
Pott's Disease	Of the spine.
Miliary Tuberculosis	X-ray lesions look like millet seeds.
Tabes Mesenterica	Tuberculosis of the abdomen.
Lupus Vulgaris	Tuberculosis of the skin.
The Common Wolf	

5.1.2 Epidemiology

The epidemiology of tuberculosis is influenced by two important factors: exposure and susceptibility. Exposure is a result of M. tuberculosis being transmitted between an infectious patient and susceptible contacts via droplet nuclei that are expelled by coughing, sneezing and other forceful respiratory activities[380]. In order to develop the disease a victim must have contact with a source case.

The probability of transmitting tuberculosis depends upon: the infectiousness of the carrier (quantity expelled), environment of exposure, duration of exposure, virulence of the

[378] Wikipedia (2005), *"Tuberculosis"*. Retrieved 24 February 2005, from: http://en.wikipedia.org/wiki/Tuberculosis

[*] Hereafter, respiratory and pulmonary tuberculosis will be used interchangeable, and when tuberculosis morbidity, incidence and notification are mentioned tuberculosis is referrs to respiratory/pulmonary tuberculosis only.

[379] Ibid

[380] Johns Hopkins Centre for Tuberculosis Research (2005). Retrieved 24 February 2005, from: http://www.hopkins-id.edu/diseases/tb/tb_class.html

organism, and susceptability of the contact. The chain of transmission can be stopped by isolating patients with the active disease (pre 1950 this was the common solution) and starting effective anti-tuberculous therapy (which became increasingly effective and commonplace from the 1950s).

The outcome of exposure is dependent upon individual susceptibility to disease. A number of conditions are associated with altered host immunity and increase the risk of developing tuberculosis, e.g. HIV infection, extremes of age, immunosuppressive therapy, cancer, end stage renal disease, diabetes, severe malnutrition and some upper gastrointestinal surgeries[381]. In addition, injection drug use is associated with an increased risk of developing tuberculosis (for reasons that are not well described). Close contacts (i.e. persons with prolonged, frequent or intense contact) are at highest risk of becoming infected.

During the twenteith century this infection rate (i.e. the chance of an individual being infected after close contact with a contagious tuberculosis agent) fell to about 22 percent from a level much closer to 100, largely as a result of improvements in standards of living[382].

5.1.3 Pathogenesis

The pathogenesis of tuberculosis is relatively complex as a result of the variety of possible temporary and permanent states associated with an initial exposure to the tubercle bacillus bacteria. Most noteworthy is the distinction between tuberculosis infection and disease. A diagnostic staging system for tuberculosis is shown below.

[381] Ibid
[382] Wikipedia (2005), “Tuberculosis”. Retrieved 24 February 2005, from: http://en.wikipedia.org/wiki/Tuberculosis

Illnesses: Tuberculosis

Table 5.3: Classification of tuberculosis[383]

Classification	Description
TB0	No exposure No infection
TB1	Exposed to tuberculosis Infection status unknown
TB2	Latent infection No disease (positive PPD)
TB3	Active tuberculosis
TB4	Inactive tuberculosis Healed / Adequately treated
TB5	Possible tuberculosis Status unknown ("rule out" tuberculosis)

In those people in whom the tuberculosis bacillus overcomes the immune system defenses and begins to multiply, there is progression from tuberculosis infection to tuberculosis disease (i.e. a move from TB1 to TB3). This can occur soon after infection, primary tuberculosis or post primary, secondary, reactivation tuberculosis disease of dormant bacilli[384]. About five percent of infected persons will develop the tuberculosis disease in the first two years, and another five percent will develop the disease later in life[385]. In aggregate, about 10 percent of infected persons with normal immune systems will develop the tuberculosis disease in their lifetime[386]. However, while only 10 percent of tuberculosis infections progresses to tuberculosis disease, when untreated (in earlier parts of the twentieth century) the death rate was more in the region of 50 percent.

Symptoms of developed tuberculosis include a prolonged cough, chest pain, and hemoptysis. Systemic symptoms include fever, chills, night sweats, appetite loss, weight loss and easy fatigability[387].

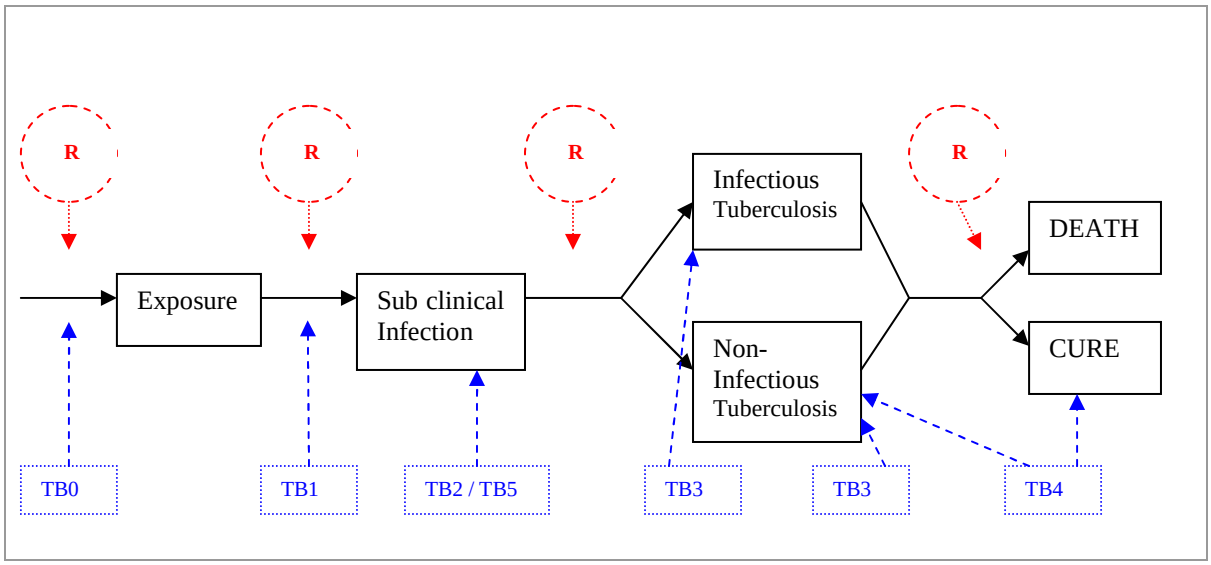
Some medical conditions increase the risk of progression from tuberculosis infection (or latent tuberculosis) to disease. In HIV infected persons with a tuberculosis infection, the

[383] Johns Hopkins Centre for Tuberculosis Research (2005). Retrieved 24 February 2005, from: http://www.hopkins-id.edu/diseases/tb/tb_class.html
[384] Ibid
[385] Ibid
[386] Ibid
[387] Wikipedia (2005), "Tuberculosis". Retrieved 24 February 2005, from: http://en.wikipedia.org/wiki/Tuberculosis

risk increases to 10 percent each year instead of 10 percent over a lifetime. Other such conditions include drug injection, substance abuse, recent tuberculosis infection (within two years) or a history of inadequately treated tuberculosis, a chest X-ray suggestive of previous tuberculosis (fibrotic lesions and nodules), diabetes mellitus, silicosis and prolonged corticosteroid therapy[388].

In an attempt to summarise the process and potential outcomes of tuberculosis, which will be considered throughout the remainder of the chapter the diagram below provides an illustration of the basic pathogenesis associated with tuberculosis. This figure also includes the above stages from Table 5.3 (blue rectangles) and highlights the series of tuberculosis risk factors (red circles).

Figure 5.2: Epidemiological process of the tuberculosis infection[389]



Exposure is defined as occurring in a person who breathes in an environment that contains tubercle bacilli[390]. Infection is defined as a person harbouring viable bacilli but without having any clinical, bacterial or radiographic signs or symptoms of the disease. Infectious tuberculosis is the form of disease that facilitates symptoms and the potential transmission of tubercle bacilli, while the non-infectious form does not. Death or survival from tuberculosis are the final stages in the pathogenesis of tuberculosis. The probabilistic relationship between these final outcomes changed considerably during the twentieth century as did the risk factors for exposure and infection.

[388] Johns Hopkins Centre for Tuberculosis Research (2005). Retrieved 24 February 2005, from: http://www.hopkins-id.edu/diseases/tb/tb_class.html
[389] International Union against Tuberculosis and Lung Disease: http://www.tbrieder.org/
[390] All humans breathe air that contains tubercle bacilli and a more accurate definition would be some form of contact with a tuberculosis patient (even outside and within a few hours of a tuberculosis patient vacating the environment) with a tuberculosis patient.

5.2 Tuberculosis Data

Concerns about tuberculosis were rife at the beginning of the twentieth century, and consequently, there are numerous data, from varied sources which provides detailed information about tuberculosis morbidity and particularly mortality.

Since 1927 tuberculosis deaths have been registered in compliance with WHO regulations, which provides a consistent and reliable mortality index for tuberculosis, although this does not escape the general problems of death registration and also displays some classification inconsistencies.

Tuberculosis morbidity data is more problematic. Although some very vague estimates for the beginning of the twentieth century exist, it was not until the 1950s that this data became more common place and reliable. This is despite continual government efforts to create an accurate index of the prevalence of tuberculosis morbidity. For example, the Public Health Act 1896 legislated the *"provision for the notification (to the medical officer of health of sanitary authorities) of cases of pulmonary tuberculosis occurring amongst the inmates of Poor Law institutions, or amongst persons under the care of the district medical officers…"*[391]. Despite these efforts, under notification was a consistent problem throughout the twentieth century: even after the introduction of the NHS the Ministry of Health recognised that *"notification remained incomplete and delayed"*[392].

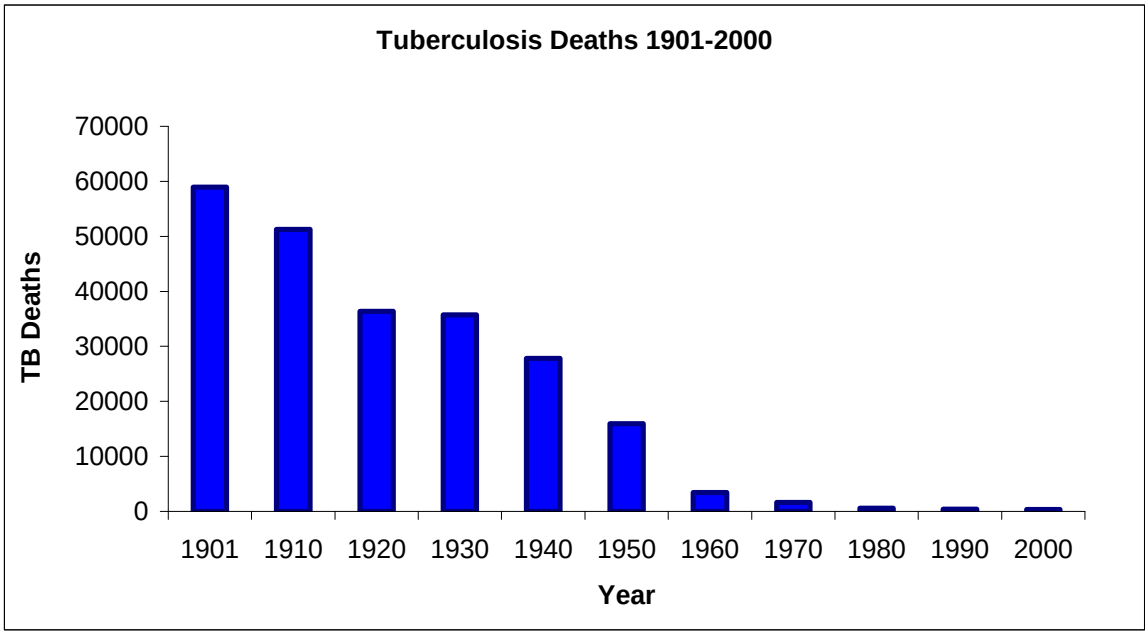
These morbidity data shortcomings before 1950 are not overly problematic because it is not until about 1950 that mortality declined significantly, such that notification rates (which are a proxy for morbidity) became the premier index of the burden of tuberculosis.

5.2.1 Mortality Data

Figure 5.3 highlights the significant twentieth century decline in tuberculosis mortality. This decline began in the late nineteenth century, for example in 1881 the death rate from pulmonary tuberculosis per 10,000 was 18.25, in 1891 it was 15.99 and in 1901 it was 12.64. This trend gathered pace throughout the twentieth century, such that, by 1980 tuberculosis had virtually been eliminated.

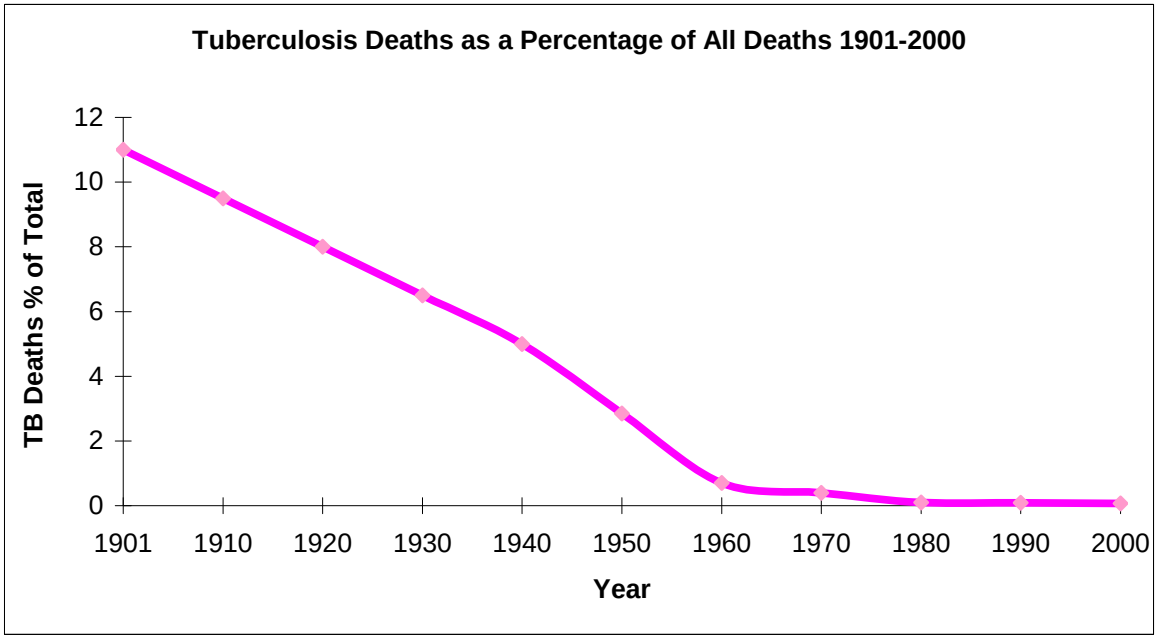
[391] The National Archives: MH55(521): Compulsory Notification of Pulmonary and Non-Pulmonary Tuberculosis: Public Health (Tuberculosis) Act 1912
[392] Coltart et al, "Social Work in Tuberculosis", p. 128

Figure 5.3: Tuberculosis deaths, 1901-2000[393]



The trend in Figure 5.3 is verified when the decline in tuberculosis mortality is considered in conjunction with all mortality. Figure 5.4 highlights that the substantial decline in the level of tuberculosis mortality is genuine, as this trend is maintained when considering the tuberculosis morbidity rate.

Figure 5.4: Tuberculosis mortality rate: tuberculosis deaths as a percentage of all deaths, 1901-2000[394]



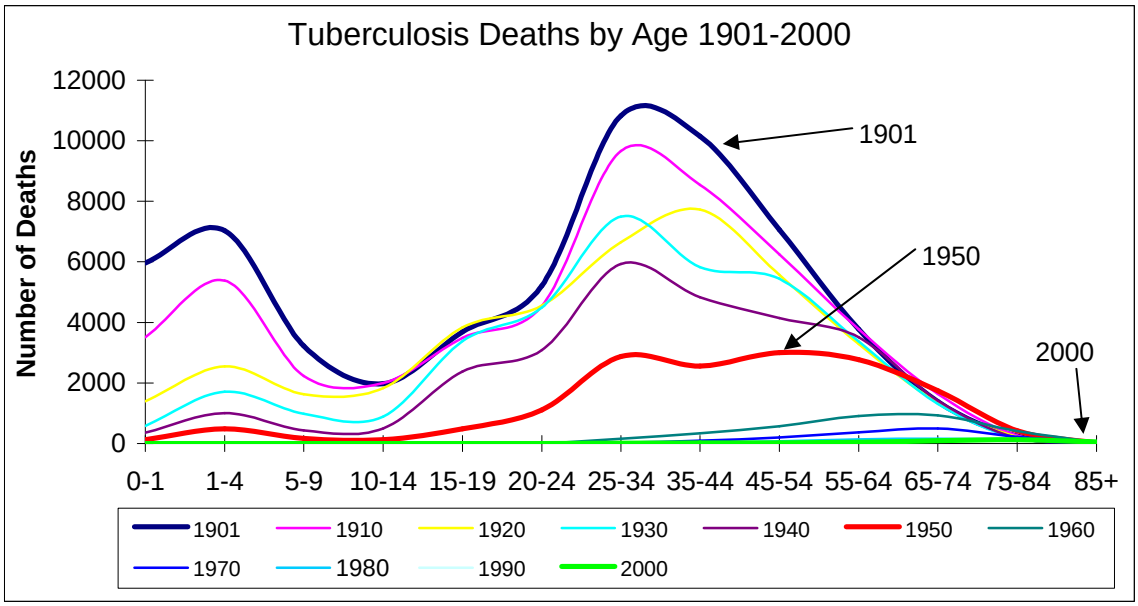
[393] Calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"
[394] Ibid

Illnesses: Tuberculosis

In Figure 5.3 and 5.4 there is a significant decline between 1901 and 1960, after which the pace slows. These figures also show that by the last quarter of the twentieth century, tuberculosis was virtually eliminated.

These improvements are accentuated when the age distribution of tuberculosis mortality are considered. This is shown in the graph below.

Figure 5.5: Tuberculosis deaths by age, 1901-2000[395]



During the twentieth century the age profile of tuberculosis mortality changed considerably. In 1901 the majority (nearly 50 percent) of tuberculosis deaths occurred in ages 25-54, and ages 0-4 accounted for nearly 20 percent of tuberculosis mortality. The age distribution continually improved such that, by 2000 nearly 75 percent of deaths occurred at ages 65+. The only exception to this trend was in 1920, possibly due to reporting issues associated with World War One.

These improvements can be further elaborated by considering age specific tuberculosis mortality in relation to all deaths in the population, i.e. the death rate by age.

[395] Calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

Illnesses: Tuberculosis

Figure 5.6: Tuberculosis deaths as a rate of all deaths by age 1901-2000[396]

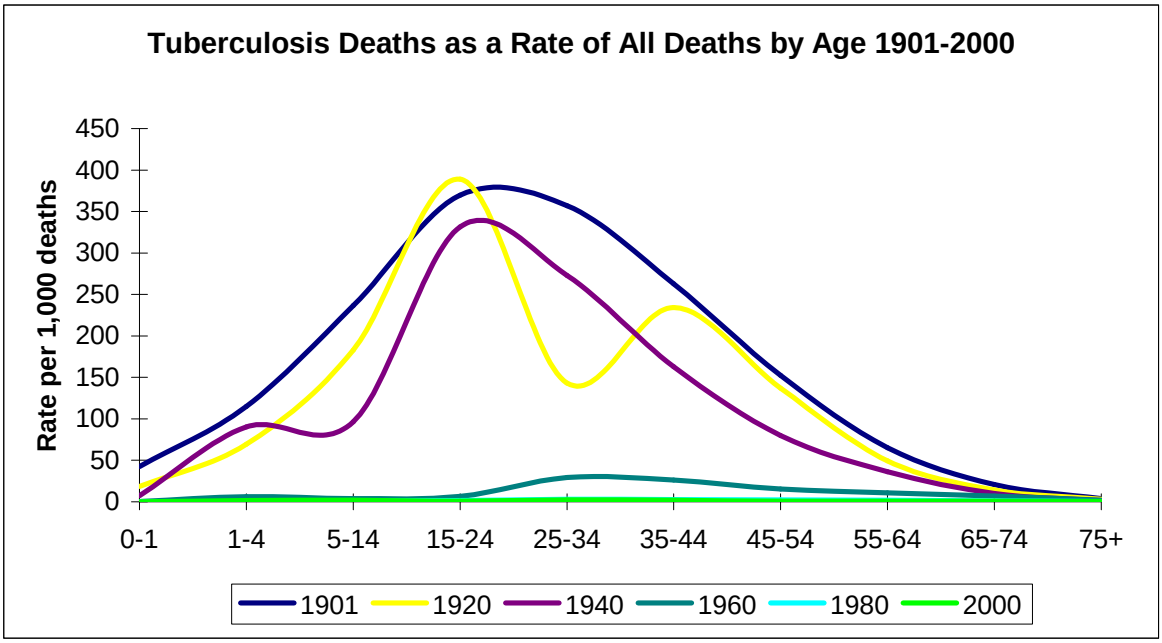
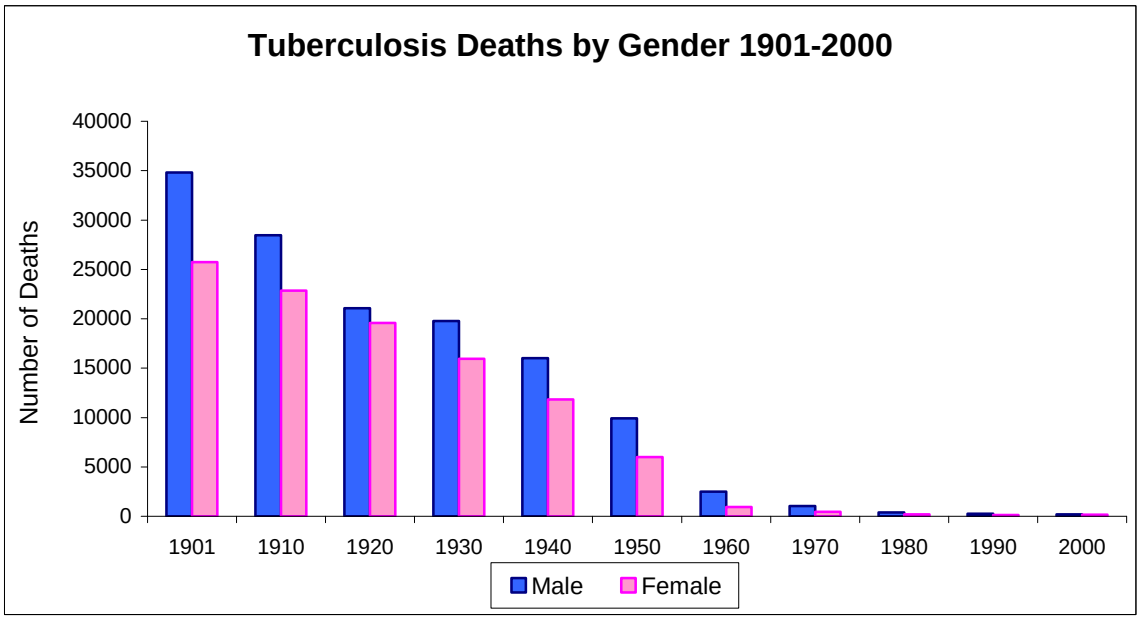


Figure 5.6 shows a consistent improvement in the burden of tuberculosis, which was generally evident throughout the twentieth century (with the exception of the 1920 anomaly).

An additional detail that deserves mention is the distribution of tuberculosis by gender.

Figure 5.7: Tuberculosis deaths by gender, 1900-2000



[396] Calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

Illnesses: Tuberculosis

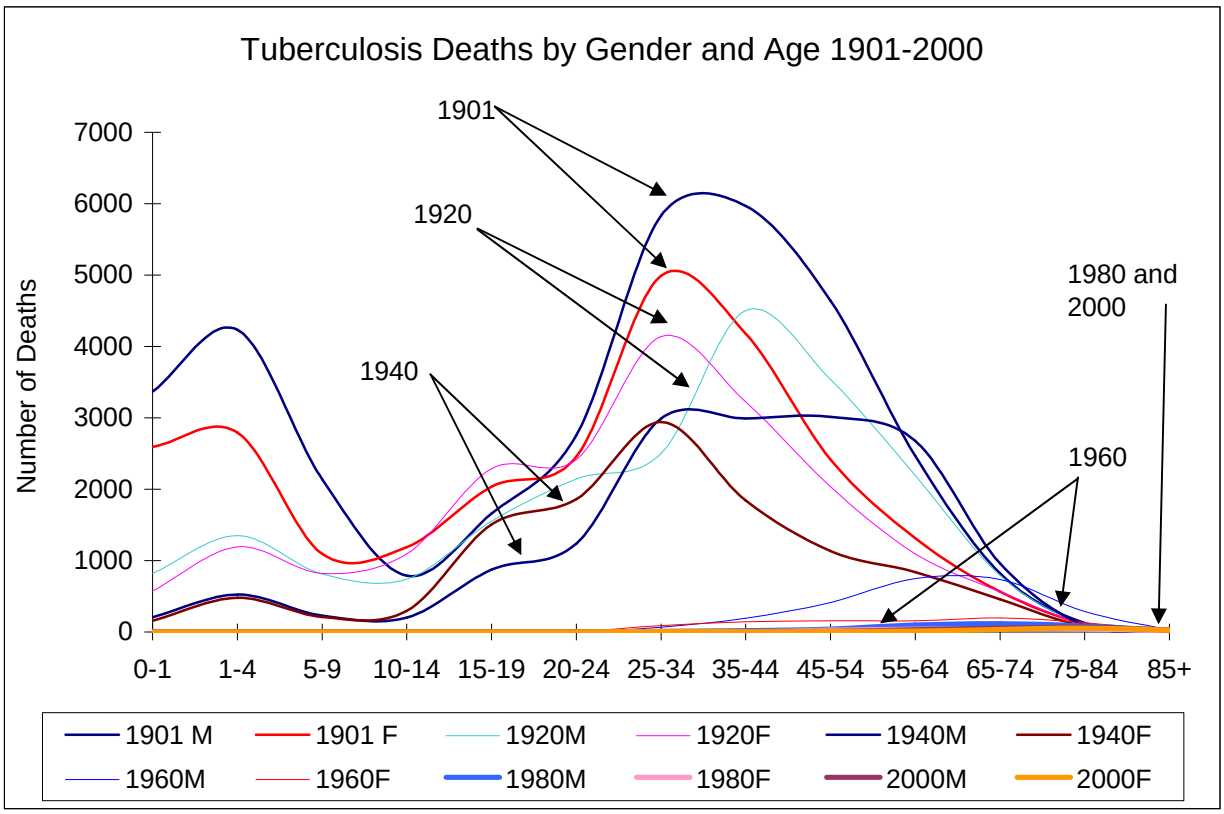
Figure 5.7 highlights that for the entire twentieth century tuberculosis mortality was greater in males than in females for the corresponding decade. The extent to which this was evident ranged from female mortality only being 37 percent of male in 1960 to 93 percent in 1920.

Table 5.4: Tuberculosis deaths: female as a percentage of male, 1901-2000

Year	1901	1910	1920	1930	1940	1950	1960	1970	1980	1990	2000
F as a % of M	74	80	93	81	74	60	37	45	49	45	78

One possible explanation for this is the higher male labour force participation rates, which would have meant that male exposure to the tuberculosis disease was greater than female. This potential explanation is supported by Figure 5.8, which illustrates the age distribution of deaths by gender.

Figure 5.8: Tuberculosis deaths by age and gender, 1901-2000



The most noteworthy feature in Figure 5.8 is the higher male mortality in the oldest ages, which differs from the majority of causes of death. Also evident is the higher prevalence of male mortality in the youngest and working age groups, although decreasingly pronounced

as the twentieth century unfolded. The only other persistent trend is the consistently higher female mortality in ages of about 15 and 20s.

5.2.2 Morbidity Data

A factor that needs to be noted when comparing mortality and morbidity statistics for twentieth century tuberculosis in England is that the mortality data includes all forms of tuberculosis, whereas the morbidity data only considers pulmonary tuberculosis. This is because pulmonary tuberculosis is the only form of tubercle bacillus that is highly contagious and therefore a public health threat that warranted notification. Table 5.5 indicates the extent to which the above aggregate tuberculosis mortality data reflects pulmonary tuberculosis.

Table 5.5: Pulmonary tuberculosis deaths as a percentage of total tuberculosis deaths, 1901-2000[397]

Year	**Total TB deaths**	**Pulmonary TB deaths**	**Pulmonary TB deaths as a % of total TB deaths (%)**
1901	60,556	50,459	83
1910	51,320	40,046	78
1920	40,634	33,853	83
1930	35,748	29,414	82
1940	27,865	23,462	85
1950	15,897	14,076	88
1960	3,427	3,101	91
1970	1,506	1,345	89
1980	587	529	90
1990	378	340	90
2000	366	310	85

During each time period of the twentieth century pulmonary tuberculosis was the major component of aggregate tuberculosis, although the extent to which this was the case varied slightly.

[397] Calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

Illnesses: Tuberculosis

Since the introduction of chemotherapy (in the 1950s) it has become necessary to consider tuberculosis morbidity in order to obtain the most accurate picture of the health related welfare burden of tuberculosis.

The most viable way to assess tuberculosis morbidity is through the notifications made to the medical officer. This data needs to be analysed with much caution due to the strong prospect of under and uneven reporting. Although this distortion is more pronounced in the earlier years it should be regarded as applicable to the entire twentieth century, despite numerous efforts to improve reporting, e.g. through the 1948 National Health Service Act, numerous [Infectious Disease] reporting regulations, and compulsory notification of all smear positive patients in 1973, there were still likely to be many undetected and unreported cases. Although problematic, under reporting notifications is not thought to be significant enough to affect the overall trends in the number of notifications in twentieth century England and Wales, as shown in Figure 5.9.

Figure 5.9: Number of tuberculosis notifications, 1915-2000[398]

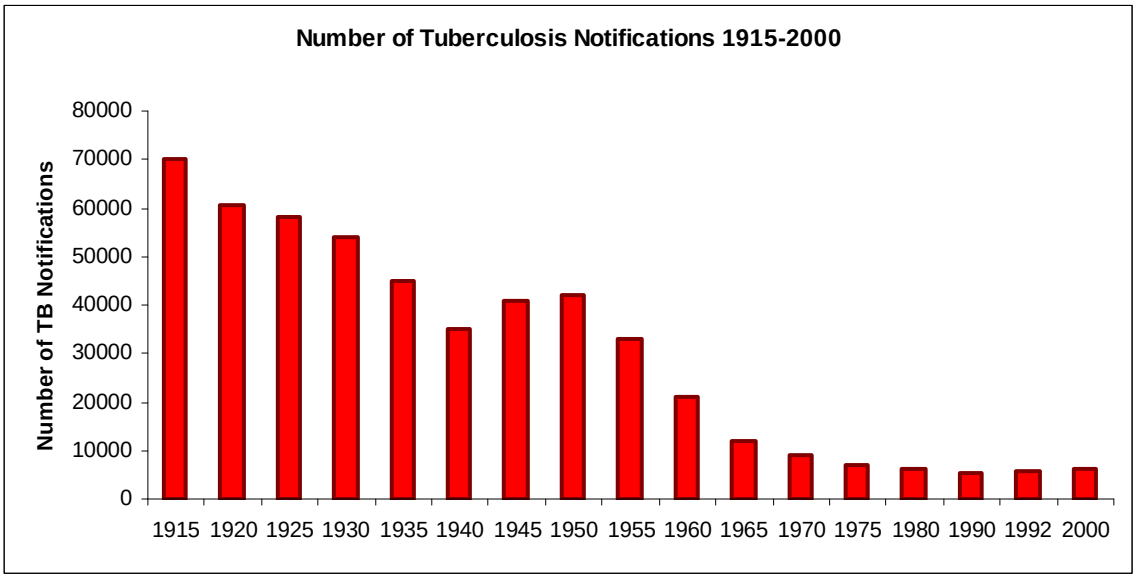


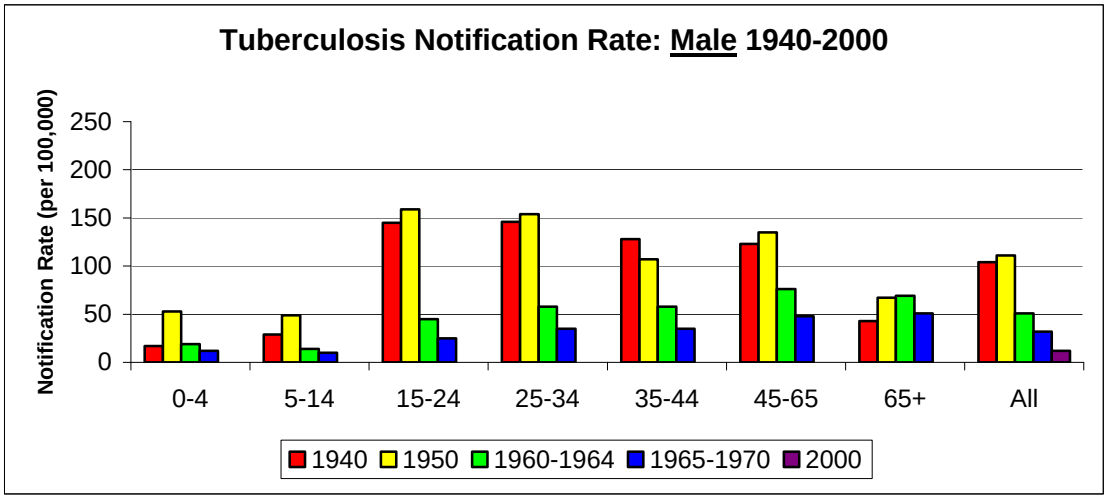
Figure 5.9 illustrates the decline in the incidence of tuberculosis during the twentieth century, which was particularly pronounced between 1915 and 1955 when the prevalence of tuberculosis was halved, largely due to an improvement in environmental factors. The decline between 1955 and 2000 is nearly entirely attributable to medical developments, which will be explained in detail later in this chapter. This fall in prevalence was not

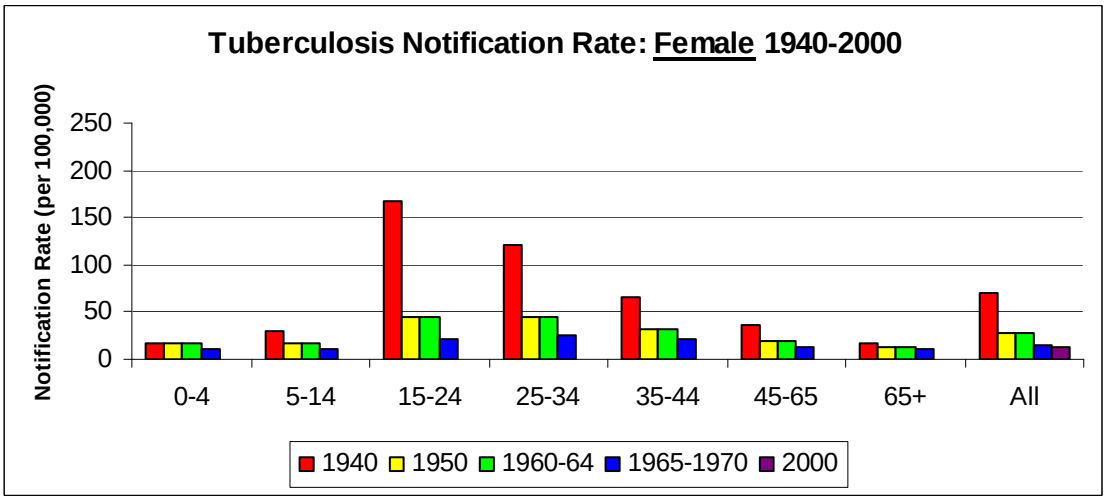
[398] 1920 to 1980: Citron et al, "Tuberculosis Today", p. 6. 1990: Watson et al, "Notifications of Tuberculosis in England and Wales, 1982-1989", p. R13. 1992 and 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 887

consistent because of the increase in 1945/1950, which is thought to be a result of the Second World War[399] and 1990-2000, which is likely to have been caused by increased deprivation particularly among ethnic minorities. E.g. in 1998 the crude incidence rate of tuberculosis per 100,000 populations in indigenous white residents in England and Wales was 4.4 compared with 121 in Indian sub-continent groups and 210 per 100,000 in black African ethnic groups[400].

As with tuberculosis mortality, it is possible to gain a more detailed understanding about the burden of tuberculosis morbidity by considering the age and gender distribution.

Figure 5.10 and 5.11: Tuberculosis notification rate: male and female, 1940-2000[401]





Figures 5.10 and 5.11 highlight trends which are similar to those identified for tuberculosis mortality. In all years except 1940 male notifications were higher than female, except for

[399] Citron et al, "Tuberculosis Today", p. 6
[400] Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 887
[401] 1940 and 1950: Logan & Benjamin, "Tuberculosis Statistics for England and Wales 1938-1955: An Analysis of Trends and Geographical Distribution", p. 12. 1960-1970: Springett, "Tuberculosis Epidemiology in England and Wales", p. 422. 2000

ages 15-24. This heightened level of female tuberculosis morbidity in 1940 could have been a result of increased female participation in factories as part of the war effort[402].

Also noteworthy is the increase in male notifications between 1940 and 1950 versus the significant decline in female notifications during this period. This could be related to reporting inaccuracies associated with World War Two, especially as by 1960, male notification rates had declined to something similar to the female notification levels.

An additional factor which needs to be considered when analysing the incidence of tuberculosis in the population is the size of the population, i.e. the notification rate.

Figure 5.12: Tuberculosis notifications: number and rate, 1920-2000[403]

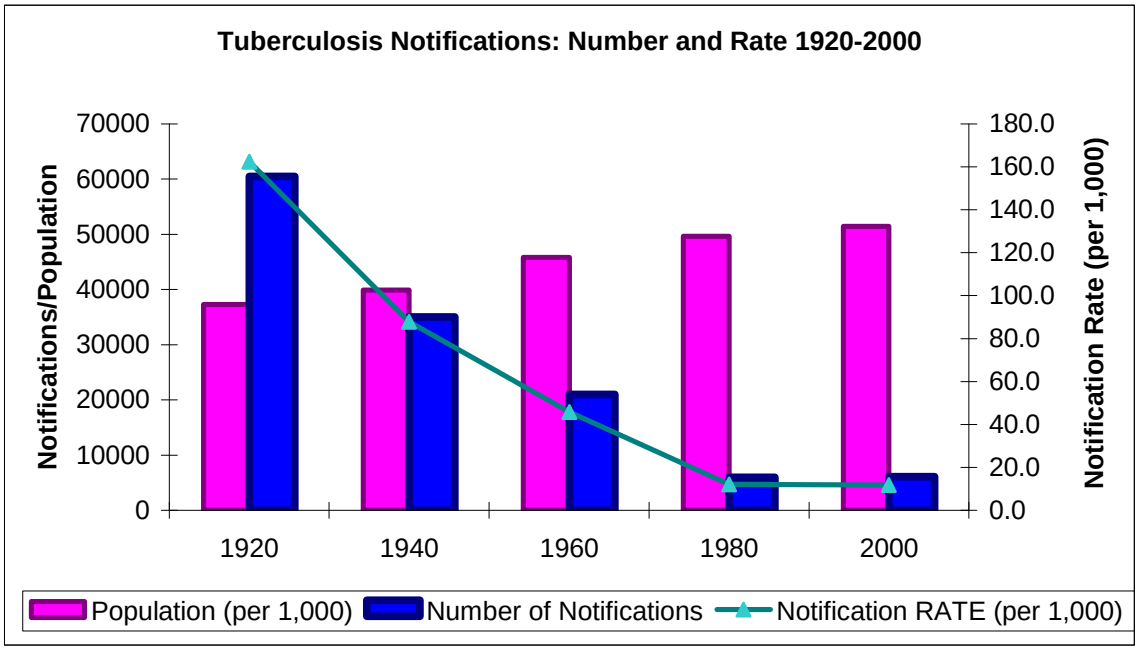


Figure 5.12 highlights that there was a decline in the number of notifications between 1920 and 1980, and that between 1980 and 2000 this trend was reversed when notifications increased from 6000 to 6087 (shown by the blue bars and the left-hand side Y axis).

The trend for the notification rate (shown by the turquoise line and the right-hand side Y axis) is slightly more optimistic. When considering the number of notifications in conjunction with the population (shown by the pink bars and the left-hand side Y axis),

[402] Bryder, "Below the Magic Mountain: A Social History of Tuberculosis in Twentieth Century Britain", p. 111
[403] 1920 to 1980: Citron et al, "Tuberculosis Today", p. 6. 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 887

there is a decline. This is better illustrated by Figure 5.13, which makes the same considerations as Figure 5.12 for 1980 and 2000, but on a magnified scale.

Figure 5.13: Tuberculosis notifications: number and rate, 1980-2000[404]

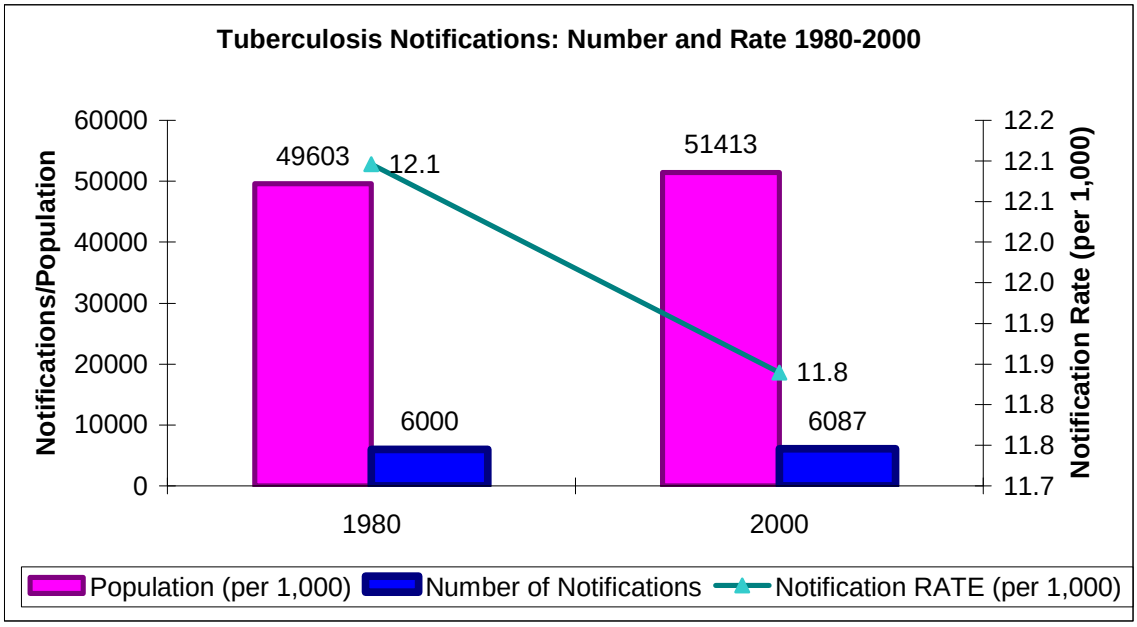


Figure 5.13 highlights that, when the increase in the number of tuberculosis notifications is standardised against the increase in the population there is actually a decline in the tuberculosis notification rate. Although this is not the most positive story about the incidence of tuberculosis (a universal decline in the prevalence would be ideal), it still detracts from the pessimism that was associated with tuberculosis and its re-emergence at the end of the twentieth century.

As a result of this decline in tuberculosis mortality and morbidity, the implications of having tuberculosis improved, such that by the end of the twentieth century tuberculosis was not associated with mortality in the way it was at the beginning. This transformation is further highlighted in Table 5.6.

[404] 1980: Citron et al, "Tuberculosis Today", p. 6. 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 887

Illnesses: Tuberculosis

Table 5.6: Tuberculosis deaths in relation to tuberculosis notifications, 1920-2000[405]

Year	Notifications	Deaths	Deaths: Notifications (%)
1920	60,500	36,504	60
1930	54,000	35,814	66
1940	35,000	27,751	79
1950	42,000	15,953	38
1960	21,000	3,420	16
1970	9,000	1,506	17
1980	6,000	587	10
1990	5,432	378	7
2000	6,087	366	6

Table 5.6 highlights that the severity and fatality of tuberculosis declined considerably during the twentieth century. In 1900 sixty percent of tuberculosis morbidity was resolved in death, versus only six percent in the year 2000. As a result of the improved tuberculosis survival rates the probability of becoming infected with tuberculosis declined significantly. This development has provided contributions to quality of life that are equally as significant as the decline in the prevalence of the disease and therefore ought to be noted, particularly because of their tacit nature. I.e. the counterfactual of 'had the prognosis of tuberculosis not improved, then the prevalence of cases would have remained higher and increased the virus in the environment which would have heightened the risk of tuberculosis infection in the population' is not quantified in the thesis methodology and it is therefore necessary to highlight this here qualitatively.

5.3 Government and Non-Government Initiatives towards Tuberculosis

"As a fundamental destructive social force tuberculosis was rivalled among illnesses only by venereal disease and insanity at the dawn of the twentieth century", and therefore, state intervention had the potential to play a vital role in the control of the spread of tuberculosis[406]. Because of the infectious nature of tuberculosis and the social problems that it threatened, the government faced a greater incentive to intervene in trying to reduce the number of tuberculosis sufferers.

[405] Notifications: 1915-1980: Citron et al, "Tuberculosis Today", p. 6. 1990: Watson et al, "Notifications of Tuberculosis in England and Wales, 1982-1989", p. R13. 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 887. Deaths: calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

[406] Smith, "The Retreat of Tuberculosis 1850-1950", p. 1

As with any illness, for example, cancer and blindness (analysed within this thesis) the government and charities had the potential to improve the quality of life for sufferers, either through economic support, pressure for positive legislation and the provision of research initiatives and motivation for recognition and help for tuberculosis sufferers.

5.3.1 Government: Overview of Twentieth Century Legislation

The seeds of government legislation for tuberculosis were planted in the nineteenth century (under the Public Health Act 1896). The culminations of these early movements towards tuberculosis legislation are shown in the Table 5.7, which provides a brief summary of the twentieth century legislation concerning tuberculosis. The most far reaching legislation in Table 5.7 will be analysed in further detail below.

Table 5.7: Government legislation for the control of tuberculosis

Year	Government legislation	Provisions of government legislation
1907	School Medical Service	The Board of Education established the School Medical Service, which led to the surveillance and detection of tuberculosis in children and promoted the removal of causes of its occurrence[407].
1908	Public Health (Tuberculosis) Act	Regulation for the notification to the medical officer of health of sanitary authorities (within 48 hours) of cases of pulmonary tuberculosis occurring amongst the inmates of Poor Law institutions, or amongst persons under the care of the district medical officers[408].
1909	Housing and Town Planning Act	The building of back-to-back houses was forbidden for the first time[409].
1911	Finance Act	£1,500,000 was made available for the construction of sanatoria[410]. Additional funds were made available for tuberculosis related research[411].
1912	Notification of Infectious Diseases Act (1889)	All forms of tuberculosis were added to this Act, which created the compulsory duty of notification of infectious disease upon the head of the family or the medical practitioner, with a penalty of forty shillings for default[412].
1912	Health Insurance Act 1912	This Act provided insurance against ill health and unemployment and applied to the majority of employed persons who received less than a stated remuneration. The Act provided funds for the treatment of tuberculosis and thus stimulated the building of sanatoria[413].
1916	Tuberculosis (Domiciliary Treatment in England) Order	Provided for treatment in the patient's own home and was designed as a remedy to the shortage of sanatorium beds[414].

[407] Coltart et al, "Social Work in Tuberculosis", p. 120
[408] The National Archives: MH55(521): Compulsory Notification of Pulmonary and Non-Pulmonary Tuberculosis: Public Health (Tuberculosis) Act 1912
[409] Coltart et al, "Social Work in Tuberculosis", p. 121
[410] Coltart et al, "Social Work in Tuberculosis", p. 124
[411] The National Archives: MH 81(44): Tuberculosis Treatment and Applications by Various Bodies for Funds 193-1914
[412] Coltart et al, "Social Work in Tuberculosis", p. 119
[413] Coltart et al, "Social Work in Tuberculosis", p. 123
[414] Coltart et al, "Social Work in Tuberculosis", p. 127

Illnesses: Tuberculosis

1921	Public Health (Tuberculosis) Act	Gave the minister of health power to make arrangements himself in areas where the county council or county borough council had failed to set up a satisfactory scheme of treatment. This Act also authorised councils to arrange after-care for tuberculosis sufferers[415].
1925	Public Health Act	Made additional provisions with regard to tuberculosis, e.g. this Act provided for compulsory segregation of infectious patients, where risk was being caused to others[416].
1929	Local Government Act	Transferred the function of the Poor Law authority to the county council and county borough council. It became the duty of the Local Authority to finance the costs of tuberculosis treatment and recover the expense of treatment if the patient could not reasonably pay. This demand for payment (from tuberculosis patients) was often not made as many local authorities thought it to be in the interest of the community to provide treatment without fee[417].
1929	Public Health (Tuberculosis) Regulations	Consolidated previous tuberculosis regulations of 1912, 1921 and 1925.
1936 (1961)	Public Health Act	Legalised the right to force upon someone treatment / hospitalisation if they appear to have tuberculosis[418]. This Act was re-issued in 1961 for notifiable disease (which includes tuberculosis).
1943	266/T	A scheme of special financial help for tuberculosis. This provided for early diagnosis and financial allowances for sufferers (and their dependents) who had to give up work in order to take treatment[419]. Also introduced x-rays, and implemented mass miniature radiography, for detecting tuberculosis.
1948	National Assistance Act	This replaced 266/T (which was a war time measure) and removed tuberculosis sufferers from the Poor Law. Different provisions were applied to tuberculosis, such that patients who had to suffer loss of income received additional compensation.
1948	National Health Service Act	Responsibility for the entire health service was placed on the Minister of Health. Various additional services were provided for tuberculosis, under Section 28 (which served to make arrangements for the purpose of prevention of illness, the care of persons suffering from illness to after-care)[420].
1969	Public Health (Infectious Diseases) Regulations	Reiterating earlier policy which states that a person should be notified as suffering from tuberculosis and provides the definition of such a person.
1984	Public Health (Control of Disease) Act	Reiterating earlier legislation which states that all forms of tuberculosis are compulsorily notifiable. And compulsory admission to hospitals is only possible where the person has tuberculosis of the respiratory tract. It also re-emphasises that all local authorities should have a written, agreed and integrated policy outlining their policy for tuberculosis control and prevention[421].

Another area of government policy, which was important although not as mainstream as the above listed, legislation was policies concerning milk. The link between cows and tabes mesenterica and other tuberculosis affections in children had long been assumed and increasing awareness that raw milk was a vehicle of human infection inspired legislation.

[415] Coltart et al, "Social Work in Tuberculosis", p. 128
[416] Coltart et al, "Social Work in Tuberculosis", p. 129
[417] Ibid
[418] The National Archives: MH55(2281): Removal of Infectious Persons to Hospitals under Section 172 of Public Health Act 1936
[419] Coltart et al, "Social Work in Tuberculosis", p. 86
[420] The National Archives: MH55(1163): Tuberculosis and the National Health Service: Report by British Medical Association: Nov 1950
[421] Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK", p. 887

Table 5.8: Government legislation for the control of bovine tuberculosis[422]

Year	Government legislation	Provisions of government legislation
1913 and 1938	The Tuberculosis Orders	Provided for the slaughter of cows suffering from tuberculosis and paid compensation.
(1924)	The Milk and Dairies Act 1914	Regulated the production, sale and distribution of milk and prohibited the sale of tuberculosis milk[423].
(1924)	The Milk and Dairies (Consolidation) Act 1915	Provided for the regulation and supervision of dairymen and their premises and for their supervision[424].
1922	Milk and Dairies (Amendment) Act	Permitted local authorities to refuse registration to purveyors of milk. Authorised the official grading of milk.
1923	The Milk (Special Designations) Order	Provided for the establishment of tuberculin tested herds.
1925	The Tuberculosis Order	Made further provisions with regard to tuberculosis in cattle, their inspection and slaughter.
1925	Public Health (Prevention of Tuberculosis) Regulations	Prohibited the employment of persons suffering from tuberculosis of the respiratory tract in various branches of the milk trade.
1926	Milk and Dairies Order	Consolidated the entire provision of previous Orders under the Milk and Dairies Act.
1936	The Milk (Special Designations) Order	For the first time included a pasteurised tuberculin-tested grade in the list of designated milk.

The culmination of these policies enabled Britain's dairy herds to be declared free from the tuberculosis infection by 1960, despite many of the above policies being ineffective due to problems of enforcement and the dominant representation of rural interests[425].

The biggest contribution is likely to have been generated from the pasteurisation of milk. This can be illustrated through a comparison of (abdominal) tuberculosis death rates between different milk consumption environments. No milk was pasteurised until 1924 and less than 15 percent in 1930[426] and nationwide pasteurisation was not evident until 1950[427]. However, in larger cities, such as London, a (unknown) quantity of milk was flash heated to prevent souring (which can be considered as a similar, although less effective, form of pasteurisation). In 1938 as much as 98 percent of the milk consumed in London was effectively (flash heated) pasteurised. Table 5.9 considers the difference in (abdominal)

[422] Coltart et al, "Social Work in Tuberculosis", p. 129
[423] As a result of World War One the full provisions of this Act did not come into force for ten years.
[424] As a result of World War One the full provisions of this Act did not come into force for ten years.
[425] Smith, "The Retreat of Tuberculosis 1850-1950", p. 176
[426] The National Archives: MH96(1920): Treatment of Tuberculosis 1950-1957
[427] Ibid

tuberculosis death rates between London and all other regions of England in order to indicate the importance of pasteurised milk in the elimination of non-respiratory tuberculosis, particularly abdominal.

Table 5.9: Crude death rate from abdominal tuberculosis (per million living), 1921-1950[428]

	1921-1930	**1938**	**1950**
London	20	7	4
Rest of England & Wales	44	15	4

It is impossible to conclusively state the precise influence of pasteurisation (in whatever form) without milk consumption rates. However, if it is accepted that the greater proportion of abdominal tuberculosis is due to bovine type infection, then the above table goes some way in attesting to the benefits of pasteurisation.

1900 → 1925

By 1900 the controversy associated with Koch's announcement in April 1882, that tuberculosis was a communicable disease, had mostly subsided and the mainstream opinion had come into alignment with Koch. This was reflected in the government's approach to combating tuberculosis[429]. (Prior to this discovery it was generally thought that tuberculosis was a hereditary disease)[430]. During the early years of the twentieth century government policy was built upon regulations regarding notification.

Under the 1908 Public Health Act, it was the duty of the medical officer of a Poor Law Institution: *"to within 48 hours of his first recognition of the symptoms of pulmonary tuberculosis in the case of a poor person who is an inmate of the institution, report to the medical officer of health of the sanitary district"*[431]. This approach was enhanced as the twentieth century unfolded. The special methods that the government felt necessary culminated in the 1912 Notification of Infectious Diseases Act, when further regulations were made extending the compulsory reporting system to cases occurring among the in-patients or out-patients of hospitals or other similar institutions for the treatment of the sick. Notwithstanding the strength of the desires of the government to control tuberculosis

[428] The National Archives: MH96(1920): Treatment of Tuberculosis 1950-1957
[429] Smith, "The Retreat of Tuberculosis 1850-1950", p. 47
[430] Latham & Garland, "The Conquest of Consumption: An Economic Study", p. 19
[431] The National Archives: MH55/521: Compulsory Notification of Pulmonary and Non-Pulmonary Tuberculosis: Public Health (Tuberculosis) Act 1912

and the feeling that compulsory notification was an *"indispensable preliminary to its* [tuberculosis] *effective administration"*, these policies were not overly successful[432].

The success of this legislation was mixed. In certain towns compulsory notification had been well established and in others the approach was haphazard and incomplete. For example, between 1915 and 1918 in England and Wales, 19 percent of deaths certified as resulting from tuberculosis had not been notified and 27 percent of tuberculosis deaths had occurred within three months of notification[433]. Even after the Ministry of Health tightened its rules in 1921, 87 percent of tuberculosis deaths in Barnsley were cases previously un-notified or notified only within six months of death[434]. The average duration of life after the commencement of the disease in an individual was about five years[435].

Even more damaging to the success of this legislation was the disregard many practitioners had towards it. Resistance of British physicians to Koch's theory, largely because his findings devalued current therapeutic practices, led them to settle for passive resistance: some notified without telling the patient and others only notified if the consumption seemed active[436].

A more optimistic claim regarding government legislation towards tuberculosis during the first quarter of the twentieth century would be that it provided a foundation for later, more successful legislation.

The 1911 Finance Act initiated provisions for the treatment and cure of tuberculosis. Under Section 16 (2), various organisations were able to apply for research grants, which were underway by 1914[437].

A more far-reaching aspect of the 1911 Finance Act was the funds provided for the extension of tuberculosis treatment (£1,500,000 for the UK and approximately £1,116,000 went to England), which were directed towards providing organised expert treatment of tuberculosis in the form of sanatoria, hospital accommodation and dispensaries[438].

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[432] Ibid
[433] Smith, "The Retreat of Tuberculosis 1850-1950", p. 69
[434] Ibid
[435] Latham & Garland, "The Conquest of Consumption: An Economic Study", p. 11
[436] Bryder, "Below the Magic Mountain: A Social History of Tuberculosis in Twentieth Century Britain", p. 41
[437] The National Archives: MH 81(44): Tuberculosis Treatment and Applications by Various Bodies for Funds 1913-1914
[438] Ibid

Illnesses: Tuberculosis

The 1912 Health Insurance Act has been coined by certain historians as: "*one of the greatest social measures of the century*"[439]. The crux of its contribution to welfare was the provision of insurance for practically all regularly employed men (and their dependents) receiving less than a stated remuneration, at the discretion of the insurance companies[440]. The original weekly contribution (paid half by the employer and half by the employee) was four pence and a government subsidy increased this to nine pence[441]. Under this Act, the state overtook the responsibilities previously carried out (generally sporadically) by charities and organisations, of providing funds for the treatment of tuberculosis, particularly contributions towards sanatorium expenses. This promoted the treatment of tuberculosis and would have helped to reduce the infectiousness, which provided a positive contribution towards standards of living, for the sufferer and population in general. Furthermore, the insurance committee was able to extend sanatoria benefit to dependents of any insured person.

This Act also provided additional powers to the Minister of Health to ensure that local authorities had satisfactory tuberculosis treatment and after-care schemes. Hence, for the first time, after-care was included in the treatment of tuberculosis[442]. However, this legislation only partially met the demands for aftercare, which were persistent until the virtual elimination of tuberculosis in the 1960s.

1925 → 1950

It was not until 1929 that state policies advanced from merely notification to more welfare orientated initiatives although, even by 1950, tuberculosis legislation was by no means comprehensive.

The 1929 Local Government Act provided the first decisive step towards the unification of tuberculosis treatment centres and an improvement in tuberculosis treatment, particularly for sufferers who were uninsured. The Act moved the treatment of tuberculosis from the jurisdiction of the Poor Law authorities to the local authorities and subsequently enabled Poor Law infirmaries to be developed into hospitals, which became better staffed and equipped for the treatment of tuberculosis.

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[439] Coltart et al, "Social Work in Tuberculosis", p. 123
[440] Ibid
[441] Ibid
[442] Coltart et al, "Social Work in Tuberculosis", p. 128

This unification in tuberculosis treatment arrived in 1948 under the National Health Service Act, when responsibility for the entire health service was placed upon the Minister of Health and treatment was administered without insurance. Under Section 28 arrangements were legislated for the comprehensive care of tuberculosis sufferers: from prevention to medical care for sufferers, including after-care. The fundamental benefits for tuberculosis sufferers were the improvements in access to treatment for their condition without the financial burden.

The policy that emerged out of the government's concern about an increase in tuberculosis due to war time conditions (associated with World War Two) provided a significant contribution to the welfare of tuberculosis sufferers, with specific legislation to manage tuberculosis patients, and the recognition that their illness was a social disease that warranted the provision of funds and compensation for ceasing work in order to receive treatment. This was provided under the 226/T Act during war time and was succeeded by the National Assistance Act in 1948, which eliminated the Poor Law and provided sickness benefit for those who contributed to the insurance scheme, which was the majority of the population. The rates which were applicable to pulmonary tuberculosis sufferers (as in the case of the 266/T this was the only form of tuberculosis covered) are shown in Table 5.10.

Table 5.10: National Assistance rate, weekly allowances, 1948[443]

Special scale for "persons who have suffered a loss of income in order to undergo treatment for respiratory tuberculosis"[444].

	Recipient	**Weekly allowance**	
		s.	***d.***
(a)	For a husband and wife:		
	Of whom one is such a person	96	0
	Of whom both are such persons	110	0
(b)	For any other such person, being:		
	Aged 21 years or over	65	0
	Aged 18 years or over but less than 21 years	48	6
	Aged 16 years or over but less than 18 years	40	0
	Aged 11 years or over but less than 16 years	20	0
	Aged 5 years or over but less than 11 years	17	0
	Aged under 5 years	14	6

Although these rates provided a significant improvement in provisions for tuberculosis sufferers (they were more than double the rates provided under 266/T), many felt that these rates only facilitated a minimum standard of subsistence, which did not finance the special needs of tuberculosis sufferers, particularly during after-care or for those who received treatment at home[445]. In the pre antibiotic era the only way to combat tuberculosis was through a strong immune system (and this was the rationale underlying sanatoria treatment), which required a nourishing diet, warm clothing and good living conditions (including adequate fuel supply), and it was felt that these elements were not afforded by the National Assistance allowance. E.g. when the tuberculosis sufferer was the breadwinner, it was thought that his family could get by on only National Assistance payouts for about a year[446].

Therefore government policy during this era was still very much initiated out of desires to prevent the spread of tuberculosis and compared to many other illnesses at the time including the other conditions in the thesis, namely the rights experienced by sufferers of

[443] Coltart et al, "Social Work in Tuberculosis", p. 139
[444] Ibid
[445] Ibid
[446] Ibid

blindness and cancer, tuberculosis sufferers experienced poor levels of recognition and rights.

1950 → 2000

During the second half of the twentieth century the only tuberculosis legislation that was passed was regarding the management of the contagiousness of this disease, through strengthening existing notification and segregation policies, under the 1969 Public Health (Infectious Diseases) Regulations and the 1984 Public Health (Control of Disease) Act. And, between 1950 and 1960 there were considerations about strengthening regulations on immigration policy, although there was never any legislation passed on this issue[447]. Table 5.11 indicates that, although many immigrants were tested, very few of these were refused entry into England.

Table 5.11: London airport tuberculosis in immigrants: number medically examined, x-rayed and refused entry into England and percentage that were refused entry as a percentage of the number medically examined, 1965[448]

Date	**Number medically examined**	**Number x-rayed**	**Number refused entry into England**	**Percent refused entry into England**
12.2.65 – 31.3.65	2,494	165	3	1 %
1.4.65 – 16.5.65	2,547	253	1	0 %
17.5.65 – 11.6.65	1,465	164	5	3 %

Therefore, during the second half of the twentieth century and especially the final quarter there was very little activity by the government to improve the standards of living of tuberculosis sufferers and despite the continued efforts to enforce notification during this period it was still noted *"how much tuberculosis existed in a community at any one time has never been known since many cases do not come to the notice of a doctor or having done so fail to be notified"*[449].

Finally, despite the lack of commitment shown by the government towards improving the standards of living of tuberculosis sufferers, there were other areas that made considerable efforts towards a significant boost to the welfare related quality of life of tuberculosis patients, namely, charities, committees, organisations and the medical profession.

[447] The National Archives: MH 55(2277): Tuberculosis among Immigrants
[448] The National Archives: MH 148(29): X-rays for Tuberculosis
[449] The National Archives: MH 154(53): Tuberculosis: Changing Epidemiological Pattern

5.3.2 Non-Government

The consideration of the roles of charities, committees, societies, medical organisations, etc, is important because they provided an important contribution to the quality of life of tuberculosis sufferers and also because they provide an indication about the shortcomings of the government.

5.3.2.1 Charities

The main role of charities was to provide financial aid to help support tuberculosis sufferers and their families, especially during the early years of the twentieth century, when less government help was available. The growth of sanatoria before 1911 was mainly a result of voluntary efforts[450]. These charities, for example, the British Red Cross Society and the Chest Clinic Samaritan Funds, provided assistance for tuberculosis sufferers' treatment and household income[451]. This assistance was evident even after the introduction of the National Assistance Act in 1948: during this time the focal point of charity initiatives became the augmentation of funds available under this Act.

The type of aid provided by charities was not in the form of a regular allowance provided for the duration of a sufferer's tuberculosis infection, as this was beyond the financial capacity of the charities that helped tuberculosis sufferers. Instead, help was usually applied for special, non-recurring needs. Although, there were some forms of charity which were consistent and ongoing, for example, The Women's Voluntary Services (WVS) which, like the Red Cross, covered the whole country and provided a 'meals on wheels' service at a small charge. The WVS also ran depots for second hand clothing, bedding and furniture[452].

Charity services had practical importance for the sufferer because they provided nursing equipment, food and better living conditions, which were all essential to the restoration of a patient's physical health. Additionally, numerous charities existed to try and help the patient with mental and social problems. E.g. voluntary organisations provided psychiatric help on matrimonial problems and emotional problems brought about by the onset of tuberculosis[453].

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[450] Bryder, "Below the Magic Mountain: A Social History of Tuberculosis in Twentieth Century Britain", p. 36
[451] Coltart et al, "Social Work in Tuberculosis", p. 109 and 112
[452] Coltart et al, "Social Work in Tuberculosis", p. 113
[453] Ibid

Despite the contribution of charities there were some shortcomings, which were largely related to resource allocation. Gerard (1983) among others suggests that resources were not evenly distributed among those who needed them most, whereby the rich and less needy tend to receive more than their fair share[454]. Additional resource allocation problems may have been generated by the number of small tuberculosis charities, whereby greater cooperation and coordination could have generated economies of scale benefits and possibly more impact when campaigning for improved government commitment to helping tuberculosis sufferers.

5.3.2.2 Committees and Associations

Committees (both governmental and charitable) played an important (largely indirect) role in boosting the welfare of tuberculosis sufferers.

The Joint Tuberculosis Council and medical associations (especially the British Medical Association) served to campaign for improved welfare treatment of tuberculosis sufferers. For example, the Tuberculosis Council took issue with the level of funds payable to patients for undergoing treatment and pushed for increases.

The British Medical Association consistently drew attention towards the flaws in the government's approach to tuberculosis treatment. Even in 1950, the BMA's National Health Service Report stated that *"the inadequacy of present provision for the diagnosis, treatment and after-care of tuberculosis in this country is a national scandal which can no longer be viewed with complacency"*[455]. During the 1960s the BMA (along with numerous other regional health boards and charitable tuberculosis committees) was also very active in pushing for medical examinations for immigrants upon entering England, in order to help prevent a worsening in the epidemiological situation associated with this tuberculosis[456].

In alignment with these observations, which highlight the shortcomings of government legislations, the Medical Research Council Committee on Tuberculosis in War-time (World War Two) pointed out that *"in view of the disturbing increase in tuberculosis and in order to effect its control, it is an urgent necessity to provide more efficient detection of*

[454] Gerard, "Charities in Britain: Conservatism or Change?", p. 21
[455] The National Archives: MH 55(1163): Tuberculosis and the National Health Service: Report by British Medical Association: Nov 1950
[456] The National Archives: MH 55(2277): Tuberculosis among Immigrants

cases in order to secure early treatment at an early stage and to reduce the prevalent source of infection"[457]. These types of claims were also voiced by the National Association for the Prevention of Tuberculosis. This organisation was founded in 1891 and throughout the twentieth century, consistently propagated the most prominent issues facing tuberculosis sufferers.

There were also organisations that consistently monitored the progress of contemporary tuberculosis issues. Most noteworthy was the Tuberculosis Advisory Committee, who focused much attention towards the re-housing of tuberculosis sufferers, and more precisely, the consistent shortages (during the late 1950s)[458]. Additionally, the Joint Tuberculosis Council also considered tangential concerns, e.g. the nutrition and treatment of tuberculosis sufferers[459].

A further contribution to quality of life of tuberculosis sufferers that these committees and organisations provided was through increasing community awareness about tuberculosis, which may have in turn perpetuated charitable work.

Hence, non-government organisations were important in helping to improve the standards of living of tuberculosis sufferers and had it not been for the lack of government pursuance in implementing their suggestions, their contribution could have been much more far-reaching. However, improvements in the quality of life associated with tuberculosis did not really originate with the work and policies of charities and government legislation, instead these improvements were primarily yielded from medical developments. Moreover, it could be argued that tuberculosis charity had a new demand at the end of the twentieth century (manifest in the unconventional tuberculosis charity needs of infected AIDS sufferers, poor immigrants and homeless who were largely responsible for the resurgence of tuberculosis in England), and that charities were slow to adapt to this challenge.

5.4 Medical Developments

The twentieth century technological developments that were important for tuberculosis sufferers were those that were experienced in antibiotics, vaccination, radiography and environmental factors. The degree to which the last two facets contributed has been

[457] The National Archives: MH 55(1141): Medical Research Council: Tuberculosis in War Time
[458] The National Archives: MH 55(2280): Priority Housing and Re-Housing for Persons Suffering from Tuberculosis: Information Provided by Local Authorities
[459] The National Archives: MH 57(429): Clause 5(3): Tuberculosis Allowances

debated throughout the twentieth century and the most profound elements of this debate will be considered here.

5.4.1 Antibiotics

Certainly one of the most important achievements of modern medicine has been the development of therapy for tuberculosis[460].

On 20 November 1944 an event of tremendous significance took place in the treatment of tuberculosis, when streptomycin was first successful in curing a patient (who had advanced pulmonary tuberculosis) at the Mayo Clinic in the USA[461]. The streptomycin strain of streptomyces griseus was isolated in September 1943 and the first public announcement of the antibiotic was made in January 1944[462]. By 1947 streptomycin was being distributed in small quantities in the UK[463].

This discovery marked the beginning of a new era in the combat of tuberculosis as it drastically reduced the epidemiological consequences of tubercle bacillus. Moreover, within a few years of the streptomycin revolution, the impetus for further developments in the treatment of tuberculosis had yielded positive results. Part of this impetus was a result of the shortcomings of streptomycin. Despite the fact that it had provided the biggest single improvement in the prospect and subsequent quality of life associated with tuberculosis, it was not potent enough to combat the entire tuberculosis problem, as the tuberculosis organism showed signs of resistance to the streptomycin drug as early as the 1950s. Furthermore, high doses of streptomycin, necessary for the treatment of tuberculosis, produced toxic effects. E.g. streptomycin was known to have adverse effects on the hearing and balance nerves. Additionally, although of less significance, but still potentially minimally damaging for quality of life, was the way in which streptomycin was administered (because of poor absorption when given by mouth), which was through a daily intramuscular injection, which caused significant pain and scarring (bruising) on the area of injection (usually the top legs/bottom)[464].

Fortunately, from the perspective of sufferers' quality of life, two additional medications were discovered shortly after streptomycin. Para-amino-salicylic acid (in 1948) and

[460] Iseman, "Evolution of Drug Resistance Tuberculosis: A Tale of Two Species", p. 2428
[461] Ibid
[462] Waksman, "Streptomycin: Background, Isolation, Properties and Isolation", p. 260
[463] Smith, "The Retreat of Tuberculosis 1850-1950", p. 246
[464] Ross, "Modern Drug Treatment in Tuberculosis", p. 10

isoniazed (in 1952) provided the necessary partners to streptomycin, such that when patients were treated with therapy combining all three antibiotics, not only were they cured of the disease but there was no (initial) emergence of resistance[465].

Despite the significant attributes of these discoveries, when this therapy was first devised, in the early 1950s, its implementation was problematic, due to the uncertainty with the ideal course of therapy: courses were short (four to six weeks), dosages were small (partly because supplies were short) and some initial results were ambiguous and some physicians only viewed this chemotherapy as 'supplemental'[466]. A further drawback of this therapy regime is the continued side effects. Most noteworthy is the potential for hepatitis from the isoniazed component[467].

With the introduction and mainstream utilisation of tuberculosis combined therapy, the development of drug resistance and failure of cure became very rare[468]. This achievement was enhanced by the relatively cheap, coordinated and rapid mainstream utilisation of this therapy. As a result of the National Health Service access to the cure for tuberculosis was facilitated to all tuberculosis sufferers. Furthermore, because of the cheap and largely straightforward nature of tuberculosis therapy the scope for problems was reduced and subsequently tuberculosis treatment was largely successful. This was achieved very soon after the initial introduction of a tuberculosis therapy regime, by the late 1950s there was very little evidence of drug cost, administration or availability issues[469]. This success can be measured in the surplus of hospital beds that was evident by 1955 and the closure of many former tuberculosis treatment centres[470]. Although drug treatment still required hospitalisation the treatment time had been significantly reduced[471].

The only exception to the above success story was salvage therapy which was considerably more expensive than standard therapy. Salvage therapy was necessary for the exceptional cases of standard drug resistance and was over 10 times more expensive than the standard, albeit available if needed[472].

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[465] Ibid
[466] Smith, "The Retreat of Tuberculosis 1850-1950", p. 247
[467] Ibid
[468] Ross, "Modern Drug Treatment in Tuberculosis", p. 20
[469] Ibid
[470] Bryder, "Below the Magic Mountain: A Social History of Tuberculosis in Twentieth Century Britain", p. 262
[471] Ibid
[472] Ross, "Modern Drug Treatment in Tuberculosis", p. 37

Illnesses: Tuberculosis

One of the most far-reaching benefits of these antibiotic inventions is the reduction in the contagiousness of tuberculosis that they created. This meant that the prevalence of tuberculosis declined markedly during the second half of the twentieth century. On an aggregate level this has meant that the threat of tuberculosis was no longer something that triggers anxiety and pandemonium, as it had at the beginning of the twentieth century. In the pre-antibiotic era the contraction of the tuberculosis disease almost certainly meant hardship, suffrage (physically, mentally and often economically) and often death. After the mainstream implementation of chemotherapy tuberculosis no longer had such severe implications. Instead of years of isolation in a treatment centre (usually a chest clinic or sanatoria) and potentially radical resolution, for example, surgery which was rarely successful, a sufferer would now undergo a course of therapy which had a much shorter duration (average treatment time was reduced from 24 to 6 months[473]) and was almost certain to have a successful resolution. Hence, *"before the introduction of chemotherapy in the treatment of tuberculosis, treatment spread over long periods, patients were kept in sanatoria beds, allowed to do very little for themselves and ceased to be productive members of the community, sometimes for years; furthermore, although they were able to return to work and a full life few of them could be said to have been cured from the disease which was to dominate the rest of their lives"*[474].

Hence, these antibiotic developments provided a substantial contribution to the improved quality of life associated with tuberculosis, largely because they substantially reduced the contagiousness and virtually eliminated tuberculosis mortality. An indication of this feat is shown in the table below.

Table 5.12: Percentage reductions in tuberculosis mortality and morbidity, 1950-1970 and 1950-2000[475]

Mortality (Tuberculosis Deaths)		**Morbidity** (Tuberculosis Notifications)	
1950 – 1970	1950 - 2000	1950 – 1970	1950 - 2000
90	98	79	86

[473] Iseman, "Evolution of Drug Resistance Tuberculosis: A Tale of Two Species", p. 2429
[474] The National Archives: MH 154(53): Tuberculosis: Changing Epidemiological Pattern
[475] Mortality: Calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause". Morbidity: 1950: Citron et al, "Tuberculosis Today", p. 6. 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 887

Illnesses: Tuberculosis

The above table illustrates the significant declines in morbidity and particularly mortality associated with tuberculosis, such that during the second half of the twentieth century mortality had been reduced by 98 percent and morbidity by 86 percent.

5.4.2 Vaccination

The benefit of the BCG (Bacille Calmette et Guerin [named after its discoverers]) vaccine was in the preventative effects it had upon tuberculosis, which ultimately contributed to reducing the infectiousness of this disease and therefore provided an (indirect) positive contribution to aggregate (population) health related quality of life[476].

BCG was not administered widely in England until the 1950s, after which the incidence of tuberculosis had fallen sharply with the advent of streptomycin. Despite the vaccination being discovered thirty years earlier plus the necessary proof of its efficacy, the scepticism among British medical practitioners retarded introduction of the BCG. The BCG was first tested on humans in 1921 and had been demonstrated to protect about 80 percent of the population at risk (under early clinical trial conditions), but British doctors dismissed the information and blocked lay attempts to act on it[477]. The mainstream utilisation of the BCG vaccine was also frustrated by problems in testing of the vaccine and errors in accurate dosages. For example, during early testing of the vaccine in 1930 in Lubeck the vaccine was administered to 200 children of which 54 died and many survivors developed tuberculosis[478]. Although it was later proved that this was an error with vaccine administration rather than the vaccine per se, this incident was a great setback in the use of BCG[479].

The culmination of these issues meant that agreement on the use of the BCG vaccine was not achieved in Britain until 1949 in Britain[480]. And initially, the vaccine was only administered to those at especial risk of developing tuberculosis (i.e. through known contact with a case). It was not until 1953 that BCG was recommended for all school children by the Ministry of Health[481].

[476] Citron et al, "Tuberculosis Today", p. 4
[477] Smith, "The Retreat of Tuberculosis 1850-1950", p. 194
[478] Ibid
[479] Ibid
[480] Bryder, "Below the Magic Mountain: A Social History of Tuberculosis in Twentieth Century Britain", p. 244
[481] Citron et al, "Tuberculosis Today", p. 4

Illnesses: Tuberculosis

Despite the above problems regarding the introduction of the BCG, by the 1970s it had provided a great boost in the prevention of tuberculosis in England. Evidence suggested that by the 1970s the BCG was providing a protection level of about 75 percent[482].

Moreover, the vaccine was thought to be so effective as to have eliminated tuberculosis (in conjunction with the above developments), that there were some who thought the school BCG programme should be stopped as a result of a falling incidence of tuberculosis in the UK, during the 1970s[483]. This marks a stark contrast with the situation twenty years earlier when it was claimed, by the government, that *"there is no scientific evidence of its* [BCG vaccination] *true value"*[484].

5.4.3 Radiography

A final area of improved medical technology, which was able to provide a positive contribution to the quality of life associated with tuberculosis, was radiography. Although this invention provided a significantly lower contribution than the other components of this chapter, the aid radiography provided in the detection of tuberculosis ought to be noted.

Mass Miniature Radiography was introduced in 1943, under the recommendations of the Medical Research Council established by the Minister of Health[485]. Since its introduction mass miniature radiography made great strides in the examination of large groups of apparently healthy individuals[486]. As a result, mass miniature radiography, was able to detect cases of tuberculosis by chance, i.e. those cases which had revealed no obvious symptoms. By the mass radiography schemes introduced in the 1940s an active case rate of 1 per 1,000 was discovered among those previously unsuspected of having tuberculosis[487]. It should be noted that the key contribution here would be related to the prevention of further cases being created.

Owing to war time limitations on the production of the requisite apparatus and to the availability of manpower, it was only possible to provide these mass radiography units gradually in selected areas in the country. However, by the end of 1953 there were about 70 units in operation, more than double the number when the NHS was introduced, and

[482] Bannon, "BCG and Tuberculosis", p. 80
[483] Ibid
[484] The National Archives: MH 55(993): Ministry of Health Report: Tuberculosis in England and Wales 1949
[485] Coltart et al, "Social Work in Tuberculosis", p. 86
[486] Medical Research Council, "Mass Miniature Radiography of Civilians for the Detection of Pulmonary Tuberculosis" p. 1
[487] Bryder, "Below the Magic Mountain: A Social History of Tuberculosis in Twentieth Century Britain", p. 5

examined about two million people a year, particularly those in industrial employment[488]. Since the 1950s the use of radiography has been used as a means of effectively diagnosing tuberculosis.

5.4.4 Environmental Factors

There is evidence that environmental factors played some role in the decline in tuberculosis, which began in the mid nineteenth century before the provision of any kind of medical advances and in conjunction with significant improvements in public health reforms which provided cleaner water, more effective waste disposal, safer food handling and improved housing conditions[489]. Hence, there does seem to be strong evidence for environmental factors playing a role in the early declines in the tuberculosis death rate, which fell from about 22.3 per 1,000 in 1840 to 10.6 per 1,000 in 1960, before the mainstream utilisation of antibiotics[490].

When considering environmental factors and their relation to tuberculosis it is possible to dichotomise these features into two broad categories: environmental developments that reduce exposure to tuberculosis and those which increase resistance to tuberculosis. It should be noted that there is strong evidence of interaction between these two broad explanations.

The most popular environmental explanation relates a story about the decline in tuberculosis being related to a reduction in the effective contact between individuals, which is related to many factors associated with improved standards of housing. The most obvious feature here is the reduction in domestic crowding, as the average household size decreased, from at least five individuals per household in 1901 to 2.5 by 1991[491]. I.e. as the population moved away from excessive overcrowding the mortality from tuberculosis diminished[492]. Overcrowding in housing as well as within houses was also important: in districts where all the houses were built in a back-to-back formation the death rate from pulmonary tuberculosis was 5.2 per 1,000 versus 2.8 per 1,000 in districts void of back-to-back housing[493].

[488] Ibid
[489] Smith, “The Retreat of Tuberculosis 1850-1950”, p. 2
[490] Ibid
[491] Ibid
[492] Ibid
[493] Smith, “The Retreat of Tuberculosis 1850-1950”, p. 2

Illnesses: Tuberculosis

Following on from this, the importance of general living conditions in determining the transmission of tuberculosis has also been studied, where the following relationship was identified: the prevalence of tuberculin sensitivity among contacts was inversely proportional to the standards of housing[494]. Improved ventilation in housing and work environments is also thought to have helped reduce the risk of the aerosol transfer of tuberculosis[495].

In all aspects of tuberculosis it is thought that the wealthier faired better, both before and after the introduction of chemotherapy in the 1950s. This provides one of the strongest indications that better nutrition, superior housing with less crowding and better ventilation, the ability to afford warm clothing and the necessary fuel were important in the reduction of tuberculosis.

A final environmental facet which played a role in the early declines in tuberculosis, although to a lesser extent than the features analysed above, is the awareness of tuberculosis and understanding about how to abate tuberculosis. For example *"a potent reason for why so many cases of consumption escape detection at the early stage among working class is the fact that working classes have not yet been educated to the point of grasping the importance of an early diagnosis"*[496].

The table below considers the effectiveness of environmental factors versus medical intervention in the elimination of tuberculosis in the twentieth century. This simple comparison considers the decline in tuberculosis in the pre and post antibiotic era. I.e. any decline in tuberculosis pre 1950 can be generally regarded as a result of environmental factors. Conversely, any reduction in tuberculosis morbidity and mortality post 1950 can be considered as a primary result of medical intervention.

[494] Ibid
[495] Ibid
[496] Latham & Garland, "The Conquest of Consumption: An Economic Study", p. 35

Illnesses: Tuberculosis

Table 5.13: Percentage reductions in tuberculosis mortality and morbidity attributable to environmental and medical developments, 1900-2000[497]

'Environmental'		**'Medical intervention'**	
Mortality	Morbidity	Mortality	Morbidity
1900 - 1950	1900 - 1950	1950 - 2000	1950 - 2000
74	40	98	86

Table 5.13 highlights the significant contribution of improved environmental factors to the decline in tuberculosis and the improvement in the quality of life associated with this disease. The decline in mortality and morbidity in the pre 1950 eras can be considered as a result of environmental factors as this was the only 'therapy' available. Therefore, the table highlights that both environmental and health developments provided valuable contributions to the combat of tuberculosis, even though much of the decline in tuberculosis was well under way before the introduction of medical developments, although tuberculosis would not have been virtually eliminated without medical developments.

5.5 Lack of Progress Considerations

The far-reaching progress in eliminating tuberculosis during the twentieth century began to halt in the 1980s[498]. During the last two decades of the twentieth century tuberculosis recaptured the concerns of public health as a result of increases in the prevalence of this disease. Although it should be noted that this is not strong enough to have caused an increase in the prevalence rate (see Figure 5.13), this reversal still needs to be contemplated.

The most popular reason for the re-emergence of tuberculosis in England is as a consequence of the HIV epidemic. The presence of HIV creates a substantially increased scope for the perpetuation of tuberculosis: an AIDS carrier's immune system is weakened and this helps the tubercle bacilli to survive and spread[499]. This spread is further perpetuated by the living conditions associated with many AIDS victims, i.e. deprived housing, poor nutritional conditions and adverse social circumstances. Additionally, resistance to tuberculosis therapy is a more pertinent issue for AIDS sufferers.

[497] Mortality: Calculations from: Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause". Morbidity: 1950: Citron et al, "Tuberculosis Today", p. 6. 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 888

[498] Vynnycky & Fine, "Interpreting the Decline in Tuberculosis: The Role of Secular Trends in Effective Contact", p. 334

[499] Bannon, "BCG and Tuberculosis", p. 80

Increased migration and globalisation has also contributed to the re-emergence of tuberculosis in populations that have comparatively low levels of tuberculosis. Hence, in developing countries tuberculosis presents a similar scenario to what was evident in Britain at the beginning of the twentieth century. With increased travel and migration the tubercle bacilli are being increasingly introduced back into populations that have achieved a very low prevalence level. In England, at the end of the twentieth century this transmission was most evident within populations from the Indian sub-continent and black Africa. For example, the tuberculosis prevalence rates in these populations, as compared with the indigenous English population were, respectively, 121 and 210 times higher[500]. Along a similar vein to the AIDS carriers, these immigrants tend to live in deprived and overcrowded conditions, which perpetuate the spread of tuberculosis.

The increase in the number of homeless people has also contributed to the re-emergence of tuberculosis. Accurate estimates about the occurrences of tuberculosis in the homeless, and even the number of homeless people, are difficult to obtain because of definition and measurement problems and the mobility of this population[501]. However, all available studies point to tuberculosis being a particular problem in this group[502].

The association between tuberculosis and deprivation is also evident in the unequal social class distribution of tuberculosis. During the first half of the twentieth century, there was a strong relationship between deprivation (social class being the proxy) and tuberculosis mortality, where the poorest social classes experienced higher tuberculosis mortality. This is shown in Table 5.14.

Table 5.14: Standardised mortality ratios for tuberculosis by social class: indexed (all males = 100), males aged 20-64[503]

Period	**Social Class (All males = 100)**				
	I	**II**	**III**	**IV**	**V**
1921-23	49	81	95	97	137
1930-32	61	70	100	104	125
1950	64	62	103	95	149

[500] Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 909
[501] Ibid
[502] Ibid
[503] Logan & Benjamin, "Tuberculosis Statistics for England and Wales 1938-1955: An Analysis of Trends and Geographical Distribution", p. 22

For all the periods in the above table there is a consistent trend between deprivation and mortality, such that the poorer social class experienced greater tuberculosis mortality.

Although this provides further evidence in favour of the intuitive relationship between deprivation and tuberculosis mortality, it does not provide a causal explanation as it is not capable of indicating which deprivation variables are fundamental. I.e. is it a story about unfavourable environmental conditions (e.g. poor nutrition) or is it a story about environmental risks (e.g. crowded housing/working conditions)?

Throughout the twentieth century and especially in the pre-antibiotic era there were persistent debates about the most fundamental environmental facets associated with tuberculosis mortality. The two contrasting opinions were those which prioritised nutrition versus those that emphasised overcrowding in the home and increasingly industrial workplace. Cobbett (1930) claimed that diet was: *"of the utmost importance"*[504], versus Collins (1925) who identified high mortality from respiratory tuberculosis in printers and shoe makers despite low general mortality, both of these industries require men to work under circumstances of poor ventilation and crowding[505].

It is clear that both categories of environmental conditions risks had a strong (positive) association with the tuberculosis infection, and it would seem virtually impossible to disaggregate them further and conclude upon the single most important environmental factor associated with tuberculosis[506].

All of these problems of the re-emergence of tuberculosis have been exacerbated by inadequate medical management and ineffective public health surveillance programmes. In conjunction with adverse patient meddling with dosages by patients, physicians must also accept some of the blame for the re-emergence of tuberculosis through an increase in tubercle bacillus drug resistance[507]. Because of the virtual elimination of tuberculosis in the years preceding the re-emergence many physicians have become less knowledgeable about treatment and consequently prescribe therapy inappropriately[508]. These problems are enhanced by inadequate monitoring of tuberculosis. Despite the introduction of the 1984

[504] Bryder, "Below the Magic Mountain: A Social History of Tuberculosis in Twentieth Century Britain", p. 111
[505] Logan & Benjamin, "Tuberculosis Statistics for England and Wales 1938-1955: An Analysis of Trends and Geographical Distribution", p. 23
[506] Mangtani et al, "Socioeconomic Deprivation and Notification Rates for Tuberculosis in London during 1982-91", p. 964
[507] Ibid
[508] Ibid

Public Health (Control of Diseases) Act, which reiterated the compulsory notification of tuberculosis, there are still shortcomings in the monitoring of tuberculosis[509]. This in turn affects control of the disease and compounds the above problems as it provides scope for the continual re-emergence of tuberculosis.

5.6 Summary

The above analysis has highlighted that the standards of living associated with tuberculosis experienced significant improvements during the twentieth century, particularly between 1950 and 1975. Mortality associated with tuberculosis declined almost consistently throughout the twentieth century, with the exception of war time increases and a marginal re-emergence in the last part of the twentieth century. Although this re-emergence in prevalence detracts from the widespread improvements it was still a very minimal setback, especially compared with the twentieth century in its entirety.

The culmination of the developments documented in this chapter, especially those in medicine, meant that the quality of life associated with tuberculosis had improved considerably during the twentieth century as it advanced from 'poor' levels in 1900, which were associated with significant distress, hardship and usually death to 'good' levels by 2000, when tuberculosis was no longer a significant threat to the population, despite the marginal re-emergence in the 1990s.

[509] Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000", p. 889

6. Cancer

"By the end of the twentieth century cancer had developed into a major public health problem, with over 250,000 people developing cancer each year and over 150,000 dying of the disease"[510]. This scenario had dramatic implications for the health and welfare related quality of life of the population, which became increasingly evident as the twentieth century unfolded.

Cancer is an extremely complex disease, which even by 2000 was only partially understood. This problem is exacerbated by the fact that there is no such thing as cancer per se. I.e. there is no single homogeneous disease and therefore no singular cure or preventative measure to remedy the health burden of cancer. Instead, each cancer (of which there are more than 200 different types) should be considered as a different disease with a different aetiology, pathogenesis and prognosis[511]. Two of these cancers will be considered here: breast cancer and stomach cancer, both featured in the top ten most common cancers during the twentieth century. Breast cancer was one of the most heavily researched cancers during the twentieth century and will therefore be more thoroughly detailed than stomach cancer in many parts of this chapter.

Throughout the twentieth century breast cancer was one of the most common cancers for women. At the end of the twentieth century (1999) one in four female cancers was located in the breast, with around 34,000 new cases diagnosed per annum in England[512]. This cancer develops in the milk-producing glands in the breast, or in the passages or ducts that deliver milk to the nipples. Breast cancer can also occur in men, but this is extremely rare.

[510] Cancer Research UK, (2003). "Breast Cancer – Summary". Retrieved 21 November 2003, from: http://www.cancerresearchuk.org/aboutcancer/specificcancers/breastcancer
[511] Ibid
[512] Ibid

Stomach cancer is another common form of the disease, which is especially prevalent in males (who are two times more likely to develop stomach cancer than their female counterparts)[513]. Stomach cancer (also known as gastric cancer) is caused when cells on the inner lining of the stomach become abnormal and start to divide uncontrollably, which ultimately causes a tumour.

As well as the merits of considering these two diseases independently, they also provide valuable complements in comparison. During the twentieth century there were continual innovations in the identification, treatment and prognosis related to breast cancer, which was not evident for stomach cancer. This scenario provides a contrast about the extent and value of technological developments that have impacted positively upon the health related quality of life of the population with breast cancer, versus those individuals suffering from stomach cancer, who did not enjoy the same improvements.

This chapter will begin with an analysis of the existing data for breast and stomach cancer. This will comprise an investigation into the prevalence and survival rates during different eras of the twentieth century.

The following section will improve the readers understanding of breast and stomach cancer with an outline of the aetiology and pathogenesis of breast and stomach cancer and the fundamental differences between these cancers will also be explained.

After this has been achieved the thesis will provide the twentieth century chronology that details the efforts made by government, charities and other bodies, in the fight to prevent, diagnose and treat cancer and evaluate how these efforts impacted upon quality of life for cancer sufferers. Following on from this, the health history will be considered in order to evaluate the impact of medical improvements upon quality of life for breast and stomach cancer sufferers.

Finally, the thesis will consider why the previously identified improvements have not been more marked. This will be achieved through identifying reasons for the lack of progress in increasing the quality of life for breast and stomach cancer sufferers.

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[513] Health A–Z, (2002). Retrieved 1 October 2002, from: www.healthatoz.com

Illnesses: Cancer

6.1 Cancer Data

Efforts to measure cancer exist in a variety of forms and completeness throughout the twentieth century. Cancer registration began under the aegis of the Radium Commission in the 1920s[514]. Subsequent considerations about cancer and the effect that it was having upon the population appeared in the 1939 Cancer Act. Although a reporting system was not actually called for under this Act, the Ministry of Health specified that *"clinical records relating to each patient should be kept so as to show clearly and accurately the history of the case"*[515]. By 1946 a national system of records for all cancer patients was instituted[516]. The collection and processing of these cancer statistics became the responsibility of the General Register Office (and its successor, the Office of Population Censuses and Surveys) when the National Health Service was introduced in 1948.

During the majority of the twentieth century the most common method for evaluating the incidence of cancer was by referencing mortality statistics. This tendency in conjunction with the voluntary nature of reporting (evident before 1971) contributed to the erroneously low level of cases registered in comparison with the estimated total experiences[517]. For example, in 1946 5,311 individuals were registered with breast cancer and approximately 7,891 died from breast cancer and 1,684 individuals were registered with stomach cancer compared to approximately 14,171 who died from this disease[518].

Progress towards a full national registration of cancer scheme was slow. This is highlighted by the formation of the Cancer Registration Working Party in 1963 (with a membership of officials from the Ministry of Health and General Register Office together with regional representation) to consider the organisation techniques of regional cancer registries and to make recommendation for their improvement[519].

By the end of 1970 the Advisory Committee on Cancer Registration had devised a simplified scheme for the collection of cancer data, with an aim of 100 percent registration of all the patients seen at or admitted to hospital with a diagnosis of cancer, and also to define, as far as possible cases of cancer never apparently seen in hospital, i.e. cases treated

[514] Swerdlow, Silva, Doll, "Cancer Incidence and Mortality in England and Wales: Trends and Risk Factors", p. 2
[515] The National Archives: MH 160 (644) : Cancer Registration: Advisory Committee Reports
[516] The National Archives: MH 160 (71): Conference on Cancer Registration and Subsequent Working Party
[517] The National Archives: MH 160 (644): Cancer Registration: Advisory Committee Reports
[518] Death Rates from cancer: Parliamentary Papers, "Cancer Statistics for England and Wales 1901-1955" : the number is approximated by dividing the 1946-1950 figure by five. Registrations of cancer: Parliamentary Papers, "Cancer Registration in England and Wales".
[519] Ibid

by general practitioners, which came to light through death notification schemes[520]. This initiative was introduced in 1971 and coined the National Cancer Registration Scheme, which provided annual information about the incidence and survival of each cancer and other cancer related statistics. The survival rate is facilitated by the one, five and ten year follow up of patients, which is conducted by the Regional Cancer Registries to provide notification either of either death or survival[521].

The registration of cancer deaths also suffers from various shortcomings, despite complying with the WHO format regulations since 1927[522]. Death data suffer more from bias and therefore criticism as death is not always correctly certified or the underlying cause correctly coded[523]. Many studies have shown wide variability in certification and coding. However the largest problem is not of major consequence here, as the effects are probably only a few percent or less[524]. For example, the percentage of mortality coded to ill defined conditions decreased from 2 percent in 1950 to 0.4 in 1980, but then increased to 0.9 in 1990[525]. Despite the undesirability of these inaccuracies associated with cancer mortality data, they are not of a great enough magnitude to bias the findings of the chapter, which marks a contrast with the morbidity (or incidence) data for the early decades of the twentieth century, which significantly underestimates the number of cancer sufferers.

Neither mortality nor (and especially) incidence rates are ideal measures. Both will be used here as there is no better alternative and these indices are still very indicative, particularly when used in tandem. The advantages and drawbacks of this data are summarised in Table 6.1.

[520] The National Archives: MH 160 (644): "Cancer Registration: Advisory Committee Reports"
[521] Ibid
[522] Rooney & Devis, "Mortality Trends by Cause of Death in England and Wales 1980-94: The Impact of Introducing Automated Cause Coding and Related Changes in 1993", p. 29
[523] Coleman, "Trends in Breast Cancer Incidence, Survival and Mortality in England and Wales", p. 590
[524] Swerdlow, "Cancer Registration in England and Wales: Some Aspects Relevant to the Interpretation of the Data", p. 159
[525] Ibid

Table 6.1: Advantages and disadvantages of incidence and mortality data for considering cancer

Incidence	**Mortality**
Advantages:	**Advantages:**
• High quality coding	• Virtually 100% complete
• Cancer site and histology	• Timely
• Low proportion of site unspecified	• Very long time series
• Incidence date known	
Disadvantages:	**Disadvantages:**
• May not be complete	• Diagnostic accuracy less certain than for incidence
• May not be sufficiently timely	• Site only, no histology
• Evidence of under-ascertainment into the early 1970s	• Around 10% unspecified
	• Deaths in any one year result from cases diagnosed over a long previous period

As a result of these aforementioned weaknesses, the twentieth century analysis of cancer data has to be considered in conjunction with the following caveats.

1. Death rates will be considered in conjunction with incidence rates (although not interchangeably). This is necessary because of the varying formats of cancer data over the twentieth century, whereby the first fifty years consider death rates and the last thirty years of the twentieth century consider the number of registered cancer sufferers. Therefore the terms ‘incidence’ and ‘prevalence’ will be used loosely, so as to describe both forms of measurement.
2. Trends will be considered in favour of explicit numbers, due to the unreliability of twentieth century (especially pre 1970) cancer data.
3. When considering the survival rate it is necessary to be aware of inaccuracies related to ‘loss of follow up’ problems, which arise when cancer registries are unable to trace an individual who will then not be recorded as dead from cancer, when in fact they might be. Although this problem largely subsided by the end of the twentieth century (due to improved registration), it is still necessary to be aware of this distorting factor when considering survival rates.

During the first fifty years of the twentieth century there were significant increases in the number of breast and stomach cancer deaths. During the second half of the century mortality rates for stomach cancer experienced substantial declines. The increase in deaths

from breast cancer was generally constant between 1900 and 2000, with the exception of a decline in the increase around the time of World War Two (which might be more a product of registration problems than a genuine improvement) and a genuine decline in breast cancer mortality during the last decade of the twentieth century.

Figure 6.1: Breast and stomach cancer mortality rate, 1911-1997[526]

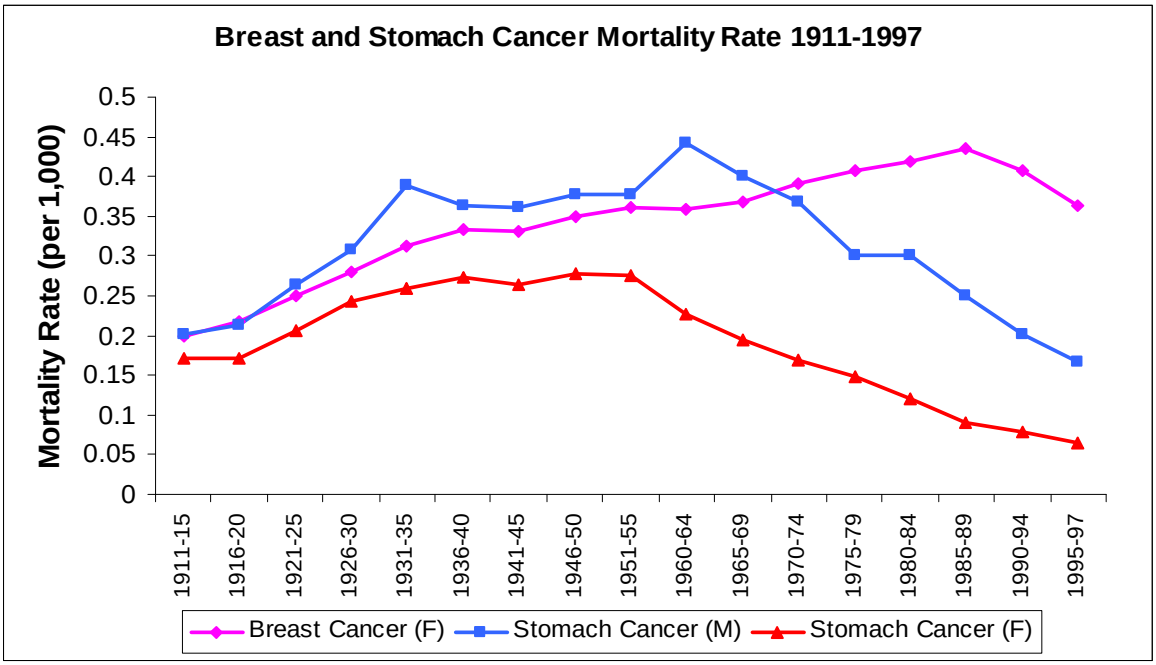


Figure 6.1 shows that, in 1911 the burden of breast and stomach cancer was relatively equal. The increase in male stomach cancer deaths between 1911 and 1955 was more pronounced than for breast cancer and female stomach cancer, respectively. The aggregate number of stomach cancer deaths was greater than breast cancer deaths. This trend was not maintained during the second half of the twentieth century, when mortality from breast and stomach cancer diverged, with an increase in breast cancer mortality, such that by the close of the twentieth century the burden of breast cancer mortality was more prominent than (aggregate) stomach cancer.

These mortality trends are similar to the incidence rates for breast and stomach cancer during the final quarter of the twentieth century. This is shown in the graph below. Between 1972 and 2000 there was a decline in the burden of stomach cancer accompanied by a significant increase in the incidence of breast cancer. Between 1984 and 1992 this increase in breast cancer prevalence was likely to be a result of improved screening, which

[526] 1911-1955: "Cancer Statistics for England and Wales 1901-1955. 1960-1997: Swerdlow, Silva, Doll, "Cancer Incidence and Mortality in England and Wales: Trends and Risk Factors", p. 221, 240

became increasingly evident from 1988 onwards, and also more comprehensive registration, which was increasingly achieved throughout the period in the graph below.

Figure 6.2: Rates of newly registered cases of breast and stomach cancer (per 100,000), 1972-2000[527]

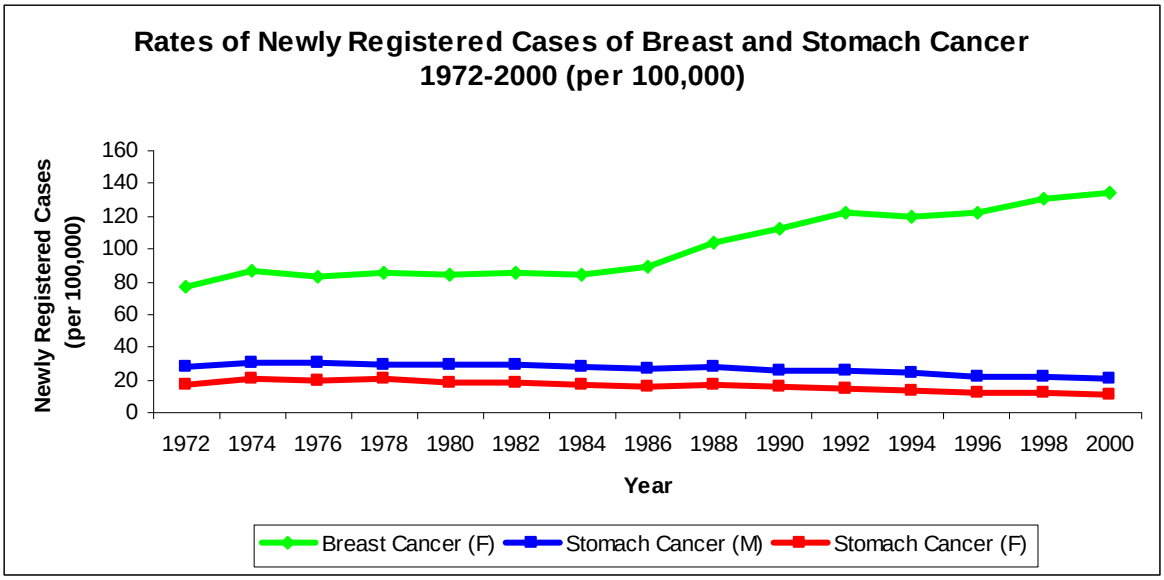


Figure 6.1 and 6.2 show that the incidence and mortality rate of stomach cancer experienced much greater improvements than breast cancer, especially if the relationship is considered for the twentieth century as a whole, when stomach cancer was more prevalent than breast cancer for the first half but as a result of increases in the incidence and mortality rate of breast cancer and a decline in stomach cancer, the opposite was true by 2000.

In order to comprehensively determine the trends in the burden of these two diseases it is necessary to consider two additional factors: the age distribution and the survival rate.

[527] Office of National Statistics, "Cancer Trends in England and Wales"

Illnesses: Cancer

Figure 6.3: Number of breast cancer deaths by age 1911-1955[528]

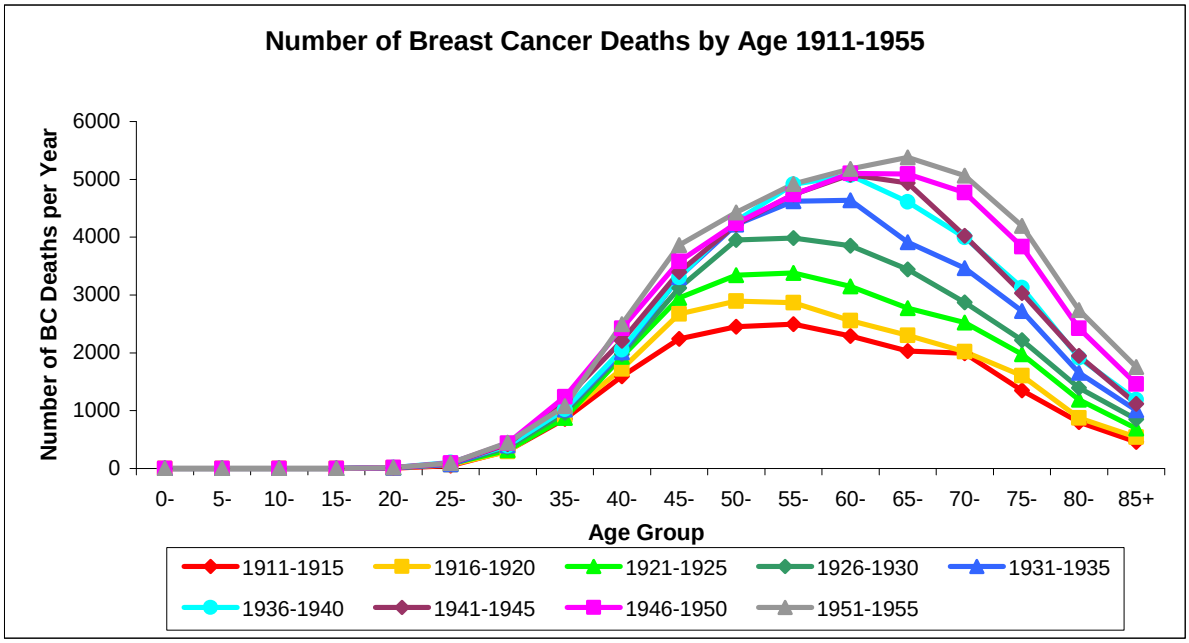
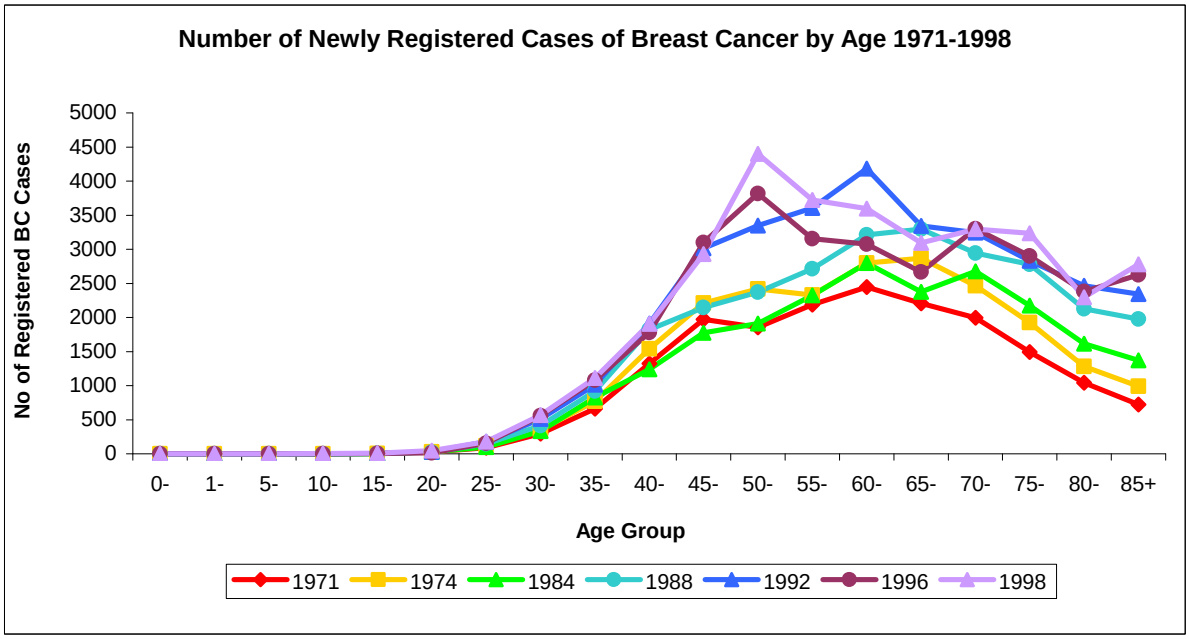


Figure 6.4: Number of newly registered cases of breast cancer by age, 1971-1998[529]



Figures 6.3 and 6.4 highlight that, in conjunction with an increase in the incidence of breast cancer there has also been an increase in the average age of incidence of this disease. The peak age of incidence of breast cancer deaths increased from 55-60 years of age in 1911 to 65-70 in 1955. However, this trend was reversed, so that the age declined between 1992 and 1998, which is likely to be most indicative of more comprehensive screening, such that

[528] Parliamentary Papers, "Cancer Statistics for England and Wales 1901-1955"
[529] Office of National Statistics, "Cancer Trends in England and Wales"

women of younger ages were being tested for breast cancer, and identified as positive for this disease at an earlier stage than would have been the case in earlier time periods. Breast cancer deaths and registrations have experienced an increase in the number of individuals at the oldest ages (85+). This is not the only age group for which there has been an increase but it highlights the only consistent trends over the twentieth century.

The incidence of stomach cancer over the twentieth century shows many similar trends and also a more complementary picture with regards to the burden of this disease upon quality of life.

Figure 6.5: Number of stomach cancer deaths by age, 1911-1955[530]

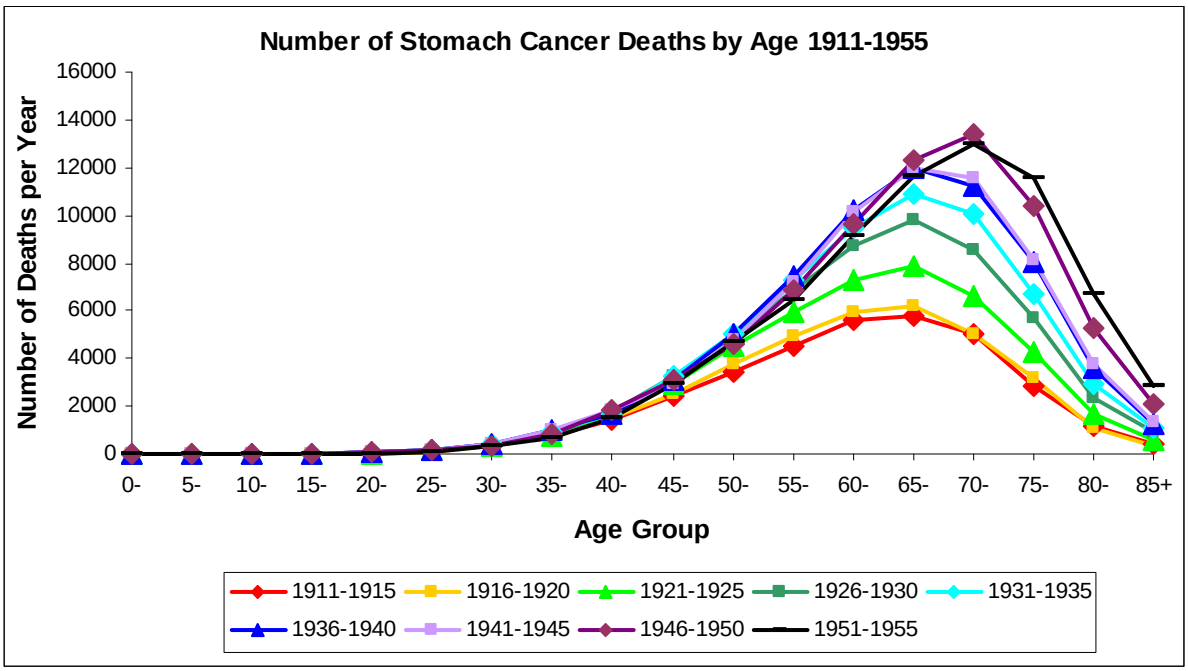


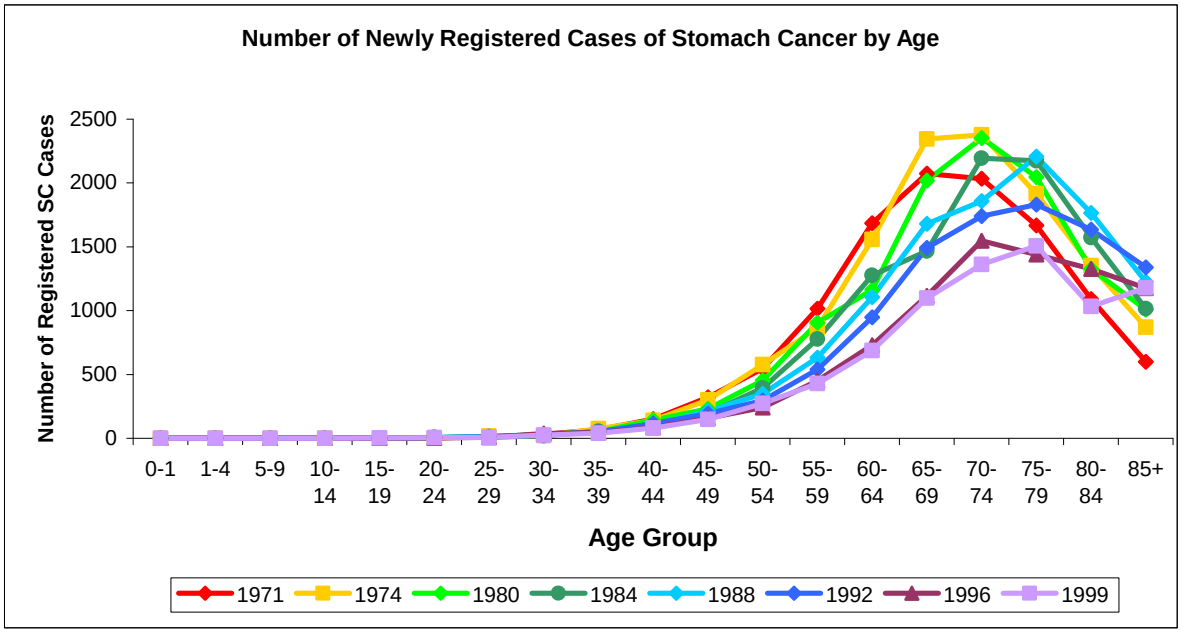
Figure 6.5 highlights the increase in the burden of stomach cancer at the oldest ages in conjunction with a decline at younger and middle ages. If the increase in the incidence of stomach cancer at the oldest ages is seen in relative terms then it becomes particularly pronounced compared to the increase for other ages. For example, between 1911 and 1955 there was more than a twenty fold increase in the number of stomach cancer deaths at ages 85+, compared to the next largest increase, which was about three fold for age 70. This change represents an improvement in the quality of life burden of stomach cancer as there were fewer healthy life years lost to this disease, which was also evident for breast cancer, although to a lesser extent.

[530] Parliamentary Papers, "Cancer Statistics for England and Wales 1901-1955"

Illnesses: Cancer

Figure 6.6 shows that between 1996 and 1999 there was an important decline in the number of registered cases of stomach cancer for all ages, including (although to a lesser extent) the oldest ages. In conjunction with the fall in the burden of stomach cancer there was also a continual increase in the peak age of incidence.

Figure 6.6: Number of newly registered cases of stomach cancer by age, 1971-1998[531]



When considering the age distribution of the burden of breast and stomach cancer, the situation is better than in absolute terms. This is especially true for stomach cancer.

One of the most indicative measures of the burden of these diseases is the survival rate, which identifies the proportion of individuals surviving cancer within a particular time span, usually one, five or ten years. The proportion of cancer patients who survive five years has become the most widely reported figure for cancer survival. Therefore, this provides the best insight into the effects of cancer upon quality of life and life expectancy for sufferers of this disease, when considered in conjunction with incidence and mortality measures[532]. The graphs below provide information about the changes in the one and five year survival rate (1Y SR and 5Y SR) for breast and stomach cancer (BC and SC) during the second half of the twentieth century, the only times at which this data exists.

[531] Office of National Statistics, "Cancer Trends in England and Wales"
[532] Enstrom & Austin "Interpreting Cancer Survival Rates", claim that survival rates cannot be used as a sole or primary measure of progress in cancer control because factors unrelated to the efficacy of treatment play an important role in the determination of those rates and their trends.

Figure 6.7: One and five year survival rate from breast cancer, 1945-2001[533]

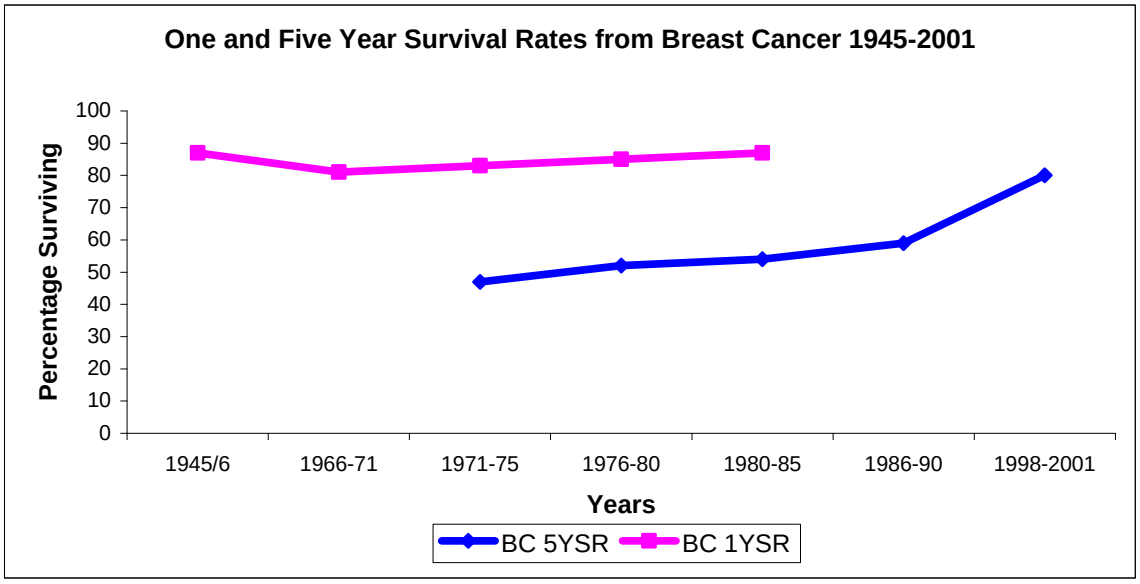
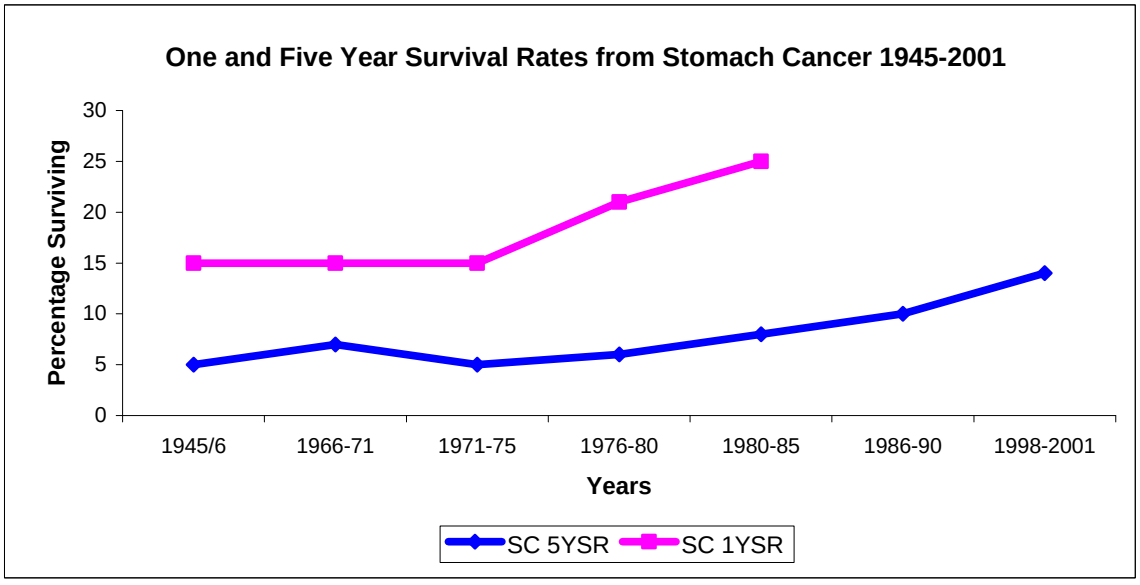


Figure 6.8: One and five year survival rate from stomach cancer, 1945-2001[534]



Figures 6.7 and 6.8 illustrate that the prognosis for breast and stomach cancer improved between 1945 and 1990, particularly during the final years of this period. This is especially pronounced for the five year survival rate for stomach cancer. Between 1945/6 and 1986-1990 there was a 100 percent improvement in the percentage of stomach cancer sufferers surviving longer than five years, albeit at very low levels (from 5 to 10 percent of the total

[533] 1945/6: Parliamentary Papers, "Cancer Registration in England and Wales".. 1966-1971: Office of National Statistics, "Cancer Trends in England and Wales 1971".1971-1990: Coleman, "Cancer Survival Trends in England and Wales, 1971-1995: Deprivation and NHS Region". All of these rates are in crude terms, as this was the only available measure for pre 1971 periods and the need to make considerations about the survival rate comparable between 1945 and 1990. 1998-2000: National Statistics Online (2005), "Cancer Survival: Rates Improved during 1996-2001". Retrieved 24 November 2005, from: http://www.statistics.gov.uk/cci/nugget_print.asp?ID=861

[534] Ibid

number diagnosed with stomach cancer). Breast cancer survival prospects also experienced improvements but of a lesser magnitude.

These survival rates highlight the different burden of these two diseases. For those who were diagnosed in 1998-2001 with stomach cancer there was less than a 15 percent chance of survival beyond five years compared to the more favourable 80 percent chance of survival from breast cancer. The contrast in the breast and stomach cancer one year survival rates is even more pronounced.

The relationship between the one and five year survival rate also has important implications for quality of life. There is a very small margin between the one and five year survival rates for stomach cancer, which implies that many of those who are diagnosed with this disease do not survive beyond one year. This margin is much larger for breast cancer, which provides an additional advantage in the prognosis of this disease. I.e. many people with breast cancer will survive for longer than a year, which is not the case for stomach cancer.

The above analysis has highlighted that the prevalence of breast cancer has increased considerably over the twentieth century, while it has declined for stomach cancer. The age distribution of these diseases shows a general trend of increasing average age and an increase in the burden of these diseases at older ages, which in many cases can be regarded as an improvement in the burden of these diseases with respect to quality of life. Survival probabilities from these two cancers had improved over the second half of the twentieth century, although still remained bleak, particularly for stomach cancer.

6.2 Definition of Breast Cancer and Stomach Cancer

The human body is made up of individual units called cells. Cells make up the tissues and organs, such as the heart or lungs. All cells divide to produce more cells when the body needs them, for example, when an individual is growing. However, sometimes cells can divide when they are not supposed to, as a result of the cells receiving the wrong signals or if there is a mistake in the DNA[535]. If cells engage in this division and the growth of cells gets out of control then they will form a mass of cells, which is a tumour. There are two types of tumours: those that are harmless (benign) and those that are cancerous. Malignant

[535] DNA contains the set of instructions for all cells.

tumours can also spread and damage other tissue, this is known as an infiltrating or invasive carcinoma or metastasis[536].

Since the 1920s, when the staging system was introduced[537], malignant tumours are classified according to the tumour size and extent of metastasis in order to determine the extent of the cancer[538]. Most frequently, cancers are grouped into four stages[539]. Stage I cancers are small, localised cancers that are usually curable, while stage IV usually represents inoperable or metastatic cancer. Stages II and III cancers are usually locally advanced. The precise definition of these stages is different for each cancer. In addition, it is important to realise that the prognosis for a given stage also depends on the type of cancer, such that a stage II stomach cancer has a different prognosis from a stage II breast cancer[540]. For example, if breast cancer is detected when still confined to the breast (stage II) the cure rate is over 95 percent[541]. This is a far better prognosis than stage II stomach cancer. The tables below provide a brief definition of the stages prescribed for breast and stomach cancer.

Table 6.2: Breast cancer diagnostic stages[542]

Stage	**Description**
I	Early stage: tumour is less than 2cm wide and has not spread outside the breast.
II	Tumour is small (2-5cm wide) or has spread to axillary (armpit) lymph nodes, or both.
III	Tumour is larger than 5cm wide and has usually spread to axillary lymph nodes and may have spread to the chest or overlying skin.
IV	Tumour of any size, usually affecting lymph nodes, has spread to other parts of the body such as bones, lungs, or liver (secondary/ metastasic tumours).

[536] Paragraph developed from AstraZeneca (2003). "Course Notes: Patient Education on Breast Cancer". Retrieved 21 November 2003, from: www.AstraZeneca.com
[537] Lee, "Dates in Oncology", p. 87
[538] Metastasis refers to the spread of cancer into other parts of the body.
[539] Cancers are also often defined by the extent of their lymph node involvement (N0–N2) and metastasis (Mo or M1) but this amount of detail is not necessary for the purposes of the thesis.
[540] AstraZeneca (2003). "Course Notes: Patient Education on Breast Cancer". Retrieved 21 November 2003, from: www.AstraZeneca.com
[541] Ibid
[542] Ibid

Table 6.3: Stomach cancer diagnostic stages[543]

Stage	Description
I	Tumour has grown no further than the inner layer of the stomach.
II	Tumour has grown into the muscle layer of the stomach wall.
III	Tumour has broken through the membrane covering the outside of the stomach.
IV	Tumour has grown into other organs or body structures nearby such as the liver or abdominal wall.

6.3 Risk Factors for Breast and Stomach Cancer

Although the precise causes of breast and stomach cancer have not been identified, several potential factors have led to increased numbers of individuals developing the disease and therefore significant risk has been associated.

Gender: Both breast and stomach cancers are associated with this factor. Approximately half of all women who develop breast cancer have no identifiable risk factor other than being female (and ageing)[544]. Although men can also get breast cancer, the incidence is extremely low[545]. Stomach cancer is twice as prevalent in men as it is in women[546].

Age: Besides being female, the greatest aetiological factor for breast cancer is age. Older women are much more likely to develop breast cancer. Figure 6.4 highlights the increase in prevalence (and therefore risk) of breast cancer between the ages of 50 and 70, throughout the twentieth century. The prevalence of stomach cancer is also a direct function of age. During the twentieth century the age incidence of stomach cancer increased, so that during the first half the major risk ages were those between 50 and 70 years old, during the second half of the twentieth century this increased to the region of 60 to 80 years of age, which is shown in Figures 6.5 and 6.6.

Diet: The potential of dietary factors being related to the development of cancer gained much interest towards the end of the twentieth century. Many epidemiological studies have indicated that dietary practices are the most promising area of cancer reduction to

[543] Cancer Research UK, (2003) "The Stages of Stomach Cancer". Retrieved 21 November 2003, from: www.cancerhelp.org.uk
[544] AstraZeneca (2003). "Course Notes: Patient Education on Breast Cancer". Retrieved 21 November 2003, from: www.AstraZeneca.com
[545] Ibid
[546] Health A–Z, (2002). Retrieved 1 October 2002, from**:** www.healthatoz.com

explore[547]. The most notorious study was conducted by Sir Richard Doll (1980), in which he suggested that seventy percent of cancers (other than those caused by tobacco) were related to diet[548]. Breast cancer risk is believed to be increased among women with an unbalanced and unhealthy diet and particularly one high in animal fat and alcohol[549]. For example, women who drink a great deal are more likely to develop invasive breast cancer than women who do not drink[550].

The most popular explanatory variable for stomach cancer is related to diet. An extensive number of studies in many countries were conducted towards the end of the twentieth century, all of which provided a link between dietary factors (which extend to environmental factors) and stomach cancer[551]. The most popular aetiological link is between food preservation (and the availability of fresh fruits and vegetables) and stomach cancer[552]. Coggon et al (1989) have highlighted the link between a low intake of salad vegetables and fruit and a high intake of salt being clearly associated with stomach cancer development[553]. Other studies have highlighted the potential harm of non-refrigeration techniques of food preservation, for example, salting, pickling and smoked foods[554].

Environment: The likelihood of a link between stomach cancer and dietary intake has led many to identify the link between improvements in refrigeration (which occurred during the twentieth century) and the decline in stomach cancer[555]. This ultimately identifies environmental factors as critically important (as this sort of technological development is defined as an 'environmental factor' in the scientific literature). In addition to this type of environmental factor, exposure to radiation, living in close proximity to industrial areas and adverse lifestyle practices in general (i.e. smoking, lack of exercise and an unhealthy diet) are thought to increase the risk of stomach and breast cancer[556].

The importance of environmental factors in the development of cancer is confirmed by cross-country comparisons about the prevalence of cancer[557]. This has been highlighted

[547] Ames, "Dietary Carcinogens and Anticarcinogens"
[548] Doll & Peto, "The Causes of Cancer"
[549] AstraZeneca (2003). "Course Notes: Patient Education on Breast Cancer". Retrieved 21 November 2003, from: www.AstraZeneca.com
[550] Ibid
[551] Coggon et al, Barker et al, Willett & MacMahon, Wei-Chen et al, Cohart, Ames, Risch et al, Hirayama, to name a few of the many studies that have been conducted in the last twenty years of the twentieth century.
[552] Willett & MacMahon, "Diet and Cancer – An Overview". Within this article Doll & Peto claim that dietary factors are second only to cigarette smoking as a determinant for all cancers.
[553] Coggon et al, "Stomach Cancer and Food Storage"
[554] U.S. Department of Health and Human Services, "Cancer Rates and Risks"
[555] Coggon et al. "The Geography of Cancer of the Stomach"
[556] Samet, "Radon and Lung Cancer"
[557] Davis et al, "International Trends in Cancer Mortality in France, West Germany, Italy, Japan, England & Wales, and the USA"

through migrant studies (where individuals develop the cancer patterns of their host country, e.g. Japanese people who have migrated to Hawaii and experienced a decline in stomach cancer rates, which correlates with those in Hawaii)[558]. The importance of environmental factors has also been confirmed by observations that adopted children whose adoptive parents have died of cancer have a five fold increased risk of getting the disease[559].

Genetic and hormonal factors are thought to play a role in the development of breast cancer. Much research has been conducted on the DNA relationships with breast cancer. This has yielded some crucial results, for example, the discovery of two breast cancer genes, namely, BRCA1 (in 1994) and BRCA2 (in 1996)[560]. Approximately five to ten percent of breast cancers occur as a result of highly penetrant germline mutations in cancer predisposing genes and half of these are due to mutations in BRCA1 or BRCA2[561].

Breast cancer is also related to the sex hormones[562]. Oestrogen imbalances can cause cells to grow and divide rapidly. These imbalances can occur for numerous reasons, e.g. prolonged periods of oestrogen production (due to the early age of menarche and late menopause), the long term use of oral contraceptives and late age of the first child birth or not having children[563]. Alternatively, women who have their first baby before the age of 30 and who breast feed are less at risk from breast cancer[564].

Previous exposure: several studies have identified a bacterium (Helicobacter pylori) that may cause stomach ulcers and chronic infections in the stomach may lead to stomach cancer[565]. Furthermore, individuals who have suffered from ulcers and other forms of stomach disorder could potentially be at an elevated risk of developing stomach cancer[566].

6.4 Government and Charity Initiatives towards Cancer

Shimkin (1977) noted that man recognised cancer and named it over two thousand years ago, but for all except the last 100 years he could do little against it[567]. Despite the long

[558] Nomura, "Stomach"
[559] Davis et al, "International Trends in Cancer Mortality in France, West Germany, Italy, Japan, England & Wales, and the USA"
[560] Lee, "Dates in Oncology", p. 122
[561] Eeles, "Future Possibilities in the Prevention of Breast Cancer: Intervention Strategies in BRCA1 and BRCA2 Mutation Cancers"
[562] AstraZeneca (2003). "Course Notes: Patient Education on Breast Cancer". Retrieved 21 November 2003, from: www.AstraZeneca.com
[563] Schrijvers et al, "Deprivation and Survival from Breast Cancer", p. 738
[564] AstraZeneca (2003). "Course Notes: Patient Education on Breast Cancer". Retrieved 21 November 2003, from: www.AstraZeneca.com
[565] Chronic infection of the stomach with these bacteria may lead to a particular type of cancer (lymphomas or mucosa-associated lymphoid tissue [MALT]) in the stomach. Dunn, S. (2003) "Cancer Guide". Retrieved 21 November 2003, from: www.cancerguide.org
[566] Ibid
[567] Breslow & Breslow, "Historical Perspectives on Cancer"

history of cancer, it only became a relatively (and increasingly) important public health problem only during the twentieth century.

6.4.1 Government

The first significant action towards the English cancer problem was provided by the 1939 Cancer Act. The main purpose of this bill was to establish a cancer service under which, in every part of the country, modern facilities for diagnosis and treatment of cancer would be available[568]. This was to be achieved through improvements in facilities, better liaison between local authorities, the Radium Commission, and local medical professionals and an increase in funding from the Exchequer to facilitate the desired improvements in cancer services[569]. However, this Act was never actually implemented because of the war[570].

In 1948 this Cancer Act was repealed when the National Health Service Act came into force. From this time the provision of cancer service was to be achieved under the National Health Service, which would provide more accessible service for the population[571]. Prior to the introduction of the NHS, healthcare was provided under a two-tiered (voluntary [only in name, by the beginning of the twentieth century] and municipal) system, which was facing increasing financial difficulties, which were largely unaided by the government[572]. For example, immediately prior to the introduction of the NHS, the government (local and central) had provided less than one-tenth of hospital funding[573].

For cancer sufferers, the principal innovation of the NHS was twofold. The first change was improved access to existing cancer services for large groups of the population who had previously been excluded[574]. The second change arose from the introduction of state-owned and funded hospitals and the government plans to modernise and equalise hospital services in England during the 1960s[575]. Before these initiatives, cancer services had been very unequally distributed geographically. Therefore, the NHS provided more accessible (and affordable) treatment for cancer sufferers. Although, even at the end of the twentieth century, services were by no means universally accessible or ideal.

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[568] The National Archives: MH 80(14): Cancer Bill
[569] Ibid
[570] Ibid
[571] The National Archives: MH 160(644): Cancer Registration: Advisory Committee Reports
[572] Powell, "Hospital Provision Before the National Health Service", p. 485
[573] Cherry, "Before the National Health Service: Financing and Voluntary Hospitals, 1900-1939", p. 315
[574] Webster, "The National Health Service: A Political History", p. 29
[575] The 1962 Hospital Plan envisaged a national network of District General Hospitals, which was to involve the building of 90 new hospitals and the rebuilding of 134 more, over the following 10 years. Hardy, "Health and Medicine in Britain Since 1860"

Illnesses: Cancer

As well as data collection policies, a major part of the government's contribution towards increased awareness, understanding and help for improving the quality of life of cancer patients was born from the numerous working committees that the government established. The Radium Commission were initially in charge of cancer data collection and processing, and also provided valuable advice for the government in the form of policy suggestions and approaches to improve cancer facilities and practices[576]. The Working Party on Cancer Registration (1962) and the Advisory Committee on Cancer Registration were also prominent as they were responsible for the introduction of the first comprehensive (nearly 100 percent coverage) cancer registration system in 1971[577]. The Joint Working Party on Computers in Radiotherapy (1967) provided initial initiatives for the electrical processing of cancer data. This working party also provided the stimulus necessary to achieve a more comprehensive understanding of cancer (and the many years of historical statistics about the diagnosis, treatment and survival of cancer patients) through electronic data processing with the use of digital computers.

The Standing Advisory Committee on Cancer and Radiotherapy (1949) and the Central Health Services Council Standing Medical Committee on Cancer Care Organisation (1969) provided valuable considerations and findings about the most productive organisation of cancer services. The former stimulated advances in radiotherapy practices and improvements in the provision of radiology services. The latter considered the organisation of cancer care under the NHS and provided numerous recommendations for improvements, many of which were still being contemplated by equivalent committees at the end of the twentieth century.

The government also supported working groups that could consider the nature and aetiology of cancers and subsequently recommend the optimal methods for combating these diseases. The Standing Sub-Committee on Cancer (1967), which contained members as distinguished as Sir Richard Doll, was sponsored by the state to make considerations about the effects of early diagnosis of cancer, the value of public awareness and various other issues related to the identification, diagnosis and eventual treatment of cancer[578].

[576] The National Archives: MH 160 (644): Cancer Registration: Advisory Committee Reports
[577] The National Archives: MH 160 (71):Conference on Cancer Registration and Subsequent Working Party and The National Archives: MH 160 (644): Cancer Registration: Advisory Committee Reports
[578] The National Archives: 160 (680): Early Diagnosis: Effect and Survival Rates

Illnesses: Cancer

Despite the continual efforts of the government to improve registration and understanding about cancer through various initiatives and working parties, the 'Smithers Report' (1971) highlighted that there were numerous shortcomings in the government's efforts to combat cancer[579]. The 'Smithers Report' painted a depressing picture of the limitations in data gathering, the lack of coordination, and the absence of standardisation in procedures in the cancer services, all of which jeopardised the early detection and effective treatment[580]. A later indictment on the NHS was their failure to adhere to any of the suggestions made in the 'Smithers Report'. By the time of the NHS reorganisation in 1974, only four regions had produced provisional regional cancer schemes. Continued reflection of slow progress of the organisation of cancer services came in 1995, when the government conceded that the quality of cancer care was patchy and variable in the skills and technology available in different hospitals and also in clinical outcomes.

During the last decade of the twentieth century the government embarked on a wide ranging initiative to improve the health of the nation, which was incorporated in the 1992 'Health of the Nation' strategy, which was designed to identify the key areas for health improvements, set targets for such improvements and also improve knowledge and understanding of these illnesses[581]. Within this Act, the government identified cancer as one of the major burdens to the nation's health[582]. However, only certain cancers were included in the legislation: cancers of the lung, breast, cervix and skin were singled out for special attention and numerical targets were set for the reduction in the incidence or mortality within a period of 10-15 years. The target for breast cancer was to reduce the death rate by at least 25 percent by the year 2000 (i.e. from 95.1 per 100,000 in 1990 to no more than 71.3 per 100,000)[583]. This was to be achieved through "*everyone with suspected breast cancer* [being] *able to see a specialist within two weeks of their general practitioner deciding they need to be seen urgently and requesting an appointment*"[584].

In 1995 the Calmann-Hine Report was published. This report recommended major organisational changes at the local level and an improvement in specialisation in cancer care, which, in 1997, became policy objectives. Hence, it was not until the

[579] Webster, "The National Health Service: A Political History", p. 116
[580] Ibid
[581] Department of Health, "The Health of the Nation: One Year On"
[582] Cancer was identified as a major cause of mortality.
[583] Department of Health, "The Health of the Nation: One Year On". This target was not achieved.
[584] Coleman, "Cancer Survival Trends in England and Wales, 1971-1995: Deprivation and NHS Region"

recommendations of the Calman-Hine Report were implemented in 1997 that cancer became a top priority and additional resources were committed to its treatment and care[585].

During the last year of the twentieth century the Health Secretary announced a £96 million plan for reducing the number of deaths from five major causes, one of which was cancer. Furthermore, this white paper, entitled 'Saving Lives', also considered the health divide and the social inequality gradient in cancer prognosis, and aimed to improve the quality of life of the population as a whole, and not just those in the highest social classes[586].

Finally, in the year 2000 the government introduced 'The NHS Cancer Plan'. This established the first comprehensive national cancer programme for England. This plan had four aims: saving more lives, ensuring that patients get the right professional support and best treatments, tackling inequalities in health (that mean unskilled workers are twice as likely to die from cancer as professionals) and investment in the cancer workforce and treatment infrastructure. These objectives are valuable in their own right and are also useful for indicating where the NHS was continuing to fail cancer sufferers most.

The effects of the persistent failure of the NHS are vividly illustrated through international comparisons of the age standardised death rate during the second half of the twentieth century, which is shown in the following tables and figures[587].

[585] Commission for Health Improvement/Audit Commission, "NHS Cancer Care in England and Wales"
[586] Hardy, "Health and Medicine in Britain since 1860", p. 172
[587] The age standardised rate (world) is a summary measure of a rate that a population would have if it had a standard age structure (the most frequently used standard population is the world standard population, which will be used here). This is necessary when comparing several populations with different age structures, due to the powerful influence of age upon the risk of cancer. The calculated mortality rate is then called the world standardised mortality rate and it is expressed per 100,000.

Illnesses: Cancer

Table 6.4: Stomach cancer age standardised death rate (world) for males and females, 1950-2000 (19 country comparison)[588]

Stomach Cancer – Male			**Stomach Cancer - Female**		
Rank 1950	**Rank 1970**	**Rank 2000**	**Rank 1950**	**Rank 1970**	**Rank 2000**
USA (17.7)	USA (8.4)	USA (3.9)	USA (9.3)	USA (4)	USA (1.9)
Australia (25.4)	Australia (14.7)	Australia (5.6)	Canada (13.3)	Australia (7.1)	Australia (2.4)
Canada (25.9)	Canada (15.2)	Canada (5.7)	Australia (13.9)	NewZealand (7.3)	Canada (2.5)
Spain (26.1)	NewZealand (16.5)	Sweden (6.3)	NewZealand (15)	Canada (7.5)	France (2.5)
NewZealand (28)	Sweden (17.6)	Switzerland (6.6)	Spain (15.6)	France (8.2)	Denmark (3)
Ireland (28.5)	France (17.6)	France (6.8)	France (15.6)	Sweden (9.6)	Switzerland (3.1)
France (29.1)	Denmark (17.7)	Denmark (6.9)	**England** (17)	Denmark (10)	**England** (3.2)
Portugal (29.1)	Scotland (20.2)	NewZealand (7.1)	Portugal (17.7)	**England** (10.3)	Sweden (3.4)
England (29.8)	Switzerland (21.1)	**England** (8)	Japan (20.4)	Switzerland (12)	Belgium (3.5)
Scotland (30.8)	**England** (21.7)	Belgium (8.1)	Sweden (20.7)	Scotland (12.1)	NewZealand (3.5)
Sweden (32.6)	Ireland (21.7)	Finland (8.2)	Ireland (21.6)	Belgium (12.2)	Netherlands (3.6)
Belgium (33.6)	Norway (22.5)	Netherlands (8.5)	Italy (21.7)	Netherlands (12.2)	Norway (3.7)
Italy (36.9)	Belgium (23.0)	Scotland (8.6)	Scotland (22.3)	Norway (12.6)	Finland (4.1)
Netherlands (41)	Netherlands (23.9)	Norway (8.8)	Netherlands (26.3)	Ireland (14.2)	Scotland (4.1)
Norway (43.4)	Spain (27.2)	Ireland (9.7)	Switzerland (29.2)	Spain (14.5)	Ireland (4.5)
Switzerland (43.5)	Finland (30.6)	Spain (11.2)	Norway (29.2)	Italy (15.2)	Spain (4.7)
Denmark (46)	Italy (30.9)	Italy (12.5)	Denmark (32.9)	Finland (16)	Italy (6.1)
Japan (66.3)	Portugal (34.3)	Portugal (20.2)	Japan (35.8)	Portugal (18.9)	Portugal (9.4)
Finland (69.1)	Japan (62.9)	Japan (29.2)	Finland (40.2)	Japan (33)	Japan (11.7)
Rank	8 / 19	9 / 19	9 / 19	6 / 19	8 / 19	6 / 19
Vs Leader	+ 41 %	+ 61 %	+ 51 %	+ 45 %	+ 61 %	+ 41 %

[588] The number in brackets after each country name is the (world) standardised death rate. The last two (detached) rows represent: 1) England's rank relative to the 19 countries considered in the table. 2) The extend to which the standardised death rate is lower in the leading country compared to England, represented in percentage terms. I.e. in 1950 male stomach cancer mortality was 41% lower in the USA (lowest death rates related to stomach cancer) than in England.

Table 6.4 shows that during the second half of the twentieth century English stomach cancer mortality maintained a relatively stable relationship with the 19 countries considered in the above table, with the exception of a worsening (compared to the [USA] leader country) during the 1970s, which was evident and equally extreme for both males and females. By 2000 the situation had improved such that females regained their 1950 rank and reduced their percentile difference with the leader. Males had moved closer towards their comparatively more favourable 1950 rank and had also partially abridged their percentile lag behind the leader country.

The international relationship for female breast cancer highlights a slightly more favourable trend. However, this could potentially be related to the very poor starting point. In 1950 breast cancer was 83 percent lower in the leading country (Japan) than in Britain. Out of the 19 country comparison, England only achieved seventeenth place. By 2000 the situation had improved as England moved into twelfth place and reduced their relative excess breast cancer mortality (in relation to the leader, which was still Japan) by 19 percent (from 83 to 64 percent).

Illnesses: Cancer

Table 6.5: Breast cancer age standardised death rate (world) for females, 1950-2000 (19 country comparison)[589]

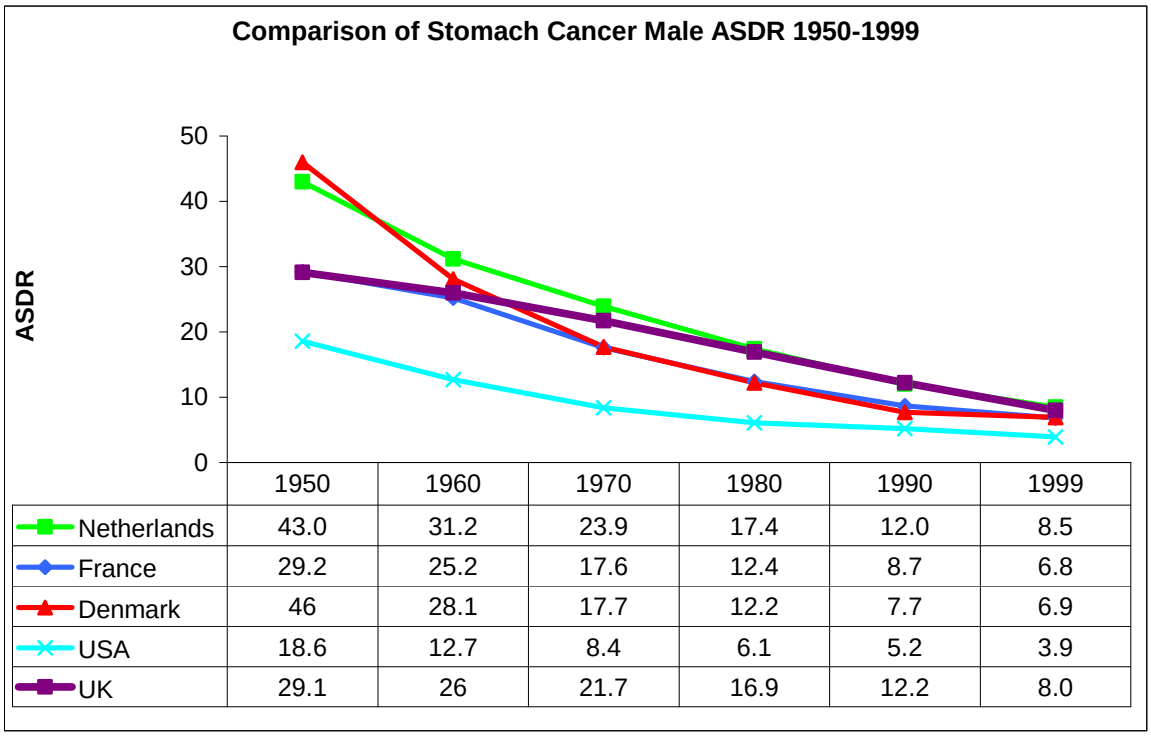
Breast Cancer – Female		
Rank 1950	**Rank 1970**	**Rank 2000**
Japan (3.9)	Japan (4.4)	Japan (7.9)
Spain (5.4)	Spain (11.2)	Spain (15.6)
Portugal (10.4)	Portugal (12.6)	Finland (16.1)
Finland (10.7)	Finland (14.1)	Portugal (16.2)
Italy (12.6)	Norway (17)	Sweden (16.4)
France (12.8)	France (17)	Norway (17.5)
Sweden (15.5)	Italy (17.6)	Australia (17.5)
Ireland (17.7)	Sweden (18.5)	USA (18.4)
Norway (19.2)	Australia (19.9)	Italy (18.8)
Australia (19.3)	Ireland (22)	France (19.2)
Belgium (20.1)	USA (22.5)	Canada (20)
USA (21.2)	Belgium (23.1)	Switzerland (20.1)
Scotland (21.8)	Switzerland (23.2)	**England (22)**
Switzerland (22)	**England (23.8)**	Scotland (22.2)
Canada (22.4)	New Zealand (25.6)	New Zealand (22.5)
New Zealand (23.1)	Canada (26.4)	Ireland (24.8)
England (23.5)	Scotland (26.4)	Netherlands (25.3)
Netherlands (24.2)	Netherlands (26.5)	Belgium (26.3)
Denmark (29)	Denmark (26.6)	Denmark (27.2)

Rank	17 / 19	13 / 19	12 / 19
Vs Leader	+ 83 %	+ 82 %	+ 64 %

Tables 6.4 and 6.5 highlight that, despite the operation of the NHS and the aforementioned efforts of charities and medical technology, England was still faring relatively badly in the war against cancer when compared to similar economies. These ominous findings are generally maintained when England is considered in a smaller, more homogenous country comparison, which is illustrated in the figures below.

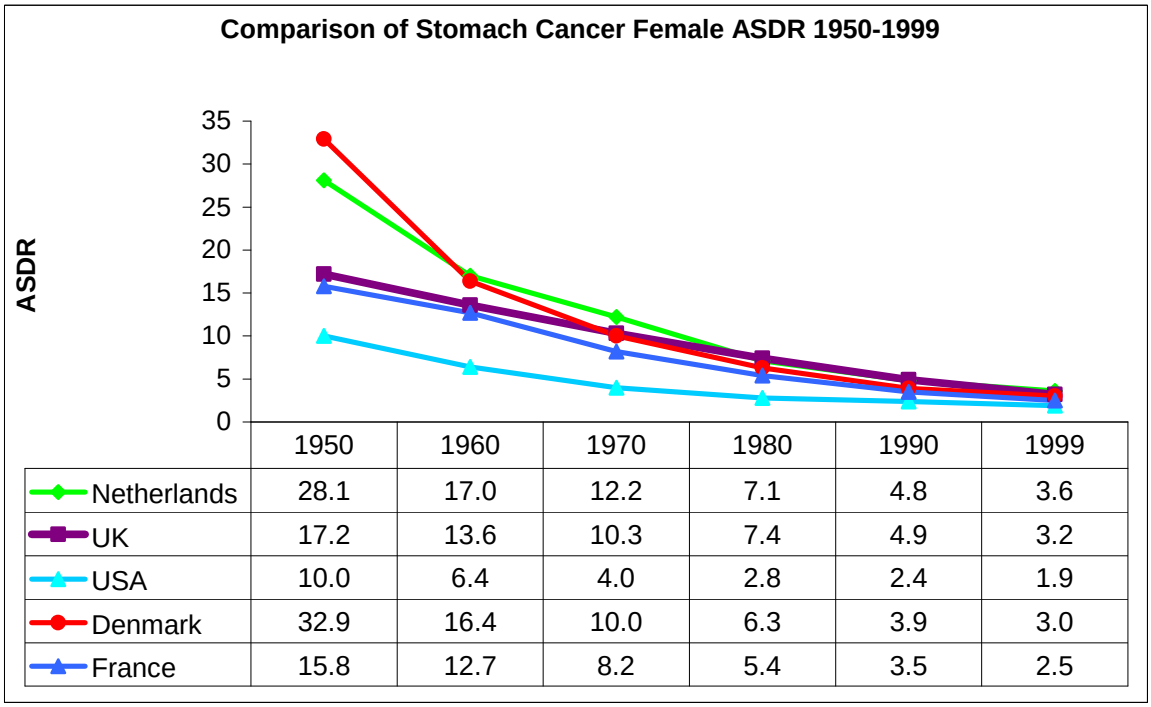
[589] The number in brackets after each country name is the (world) standardised death rate. The last two (detached) rows represent: 1) England's rank relative to the 19 countries considered in the table. 2) The extend to which the standardised death rate is lower in the leading country compared to England, represented in percentage terms. I.e. in 1950 female breast cancer mortality was 83% lower in Japan (lowest death rates related to breast cancer) than in England. World Health Organisation (2002), "WHO Mortality Data Base". Retrieved 1 October 2002, from: http://www.who.org

Figure 6.9: Stomach cancer age standardised death rate (ASDR) for world, males, 1950-1999 (5 country comparison)[590]



	1950	1960	1970	1980	1990	1999
Netherlands	43.0	31.2	23.9	17.4	12.0	8.5
France	29.2	25.2	17.6	12.4	8.7	6.8
Denmark	46	28.1	17.7	12.2	7.7	6.9
USA	18.6	12.7	8.4	6.1	5.2	3.9
UK	29.1	26	21.7	16.9	12.2	8.0

Figure 6.10: Stomach cancer age standardised death rate (ASDR) for world, females, 1950-1999 (5 country comparison)[591]



	1950	1960	1970	1980	1990	1999
Netherlands	28.1	17.0	12.2	7.1	4.8	3.6
UK	17.2	13.6	10.3	7.4	4.9	3.2
USA	10.0	6.4	4.0	2.8	2.4	1.9
Denmark	32.9	16.4	10.0	6.3	3.9	3.0
France	15.8	12.7	8.2	5.4	3.5	2.5

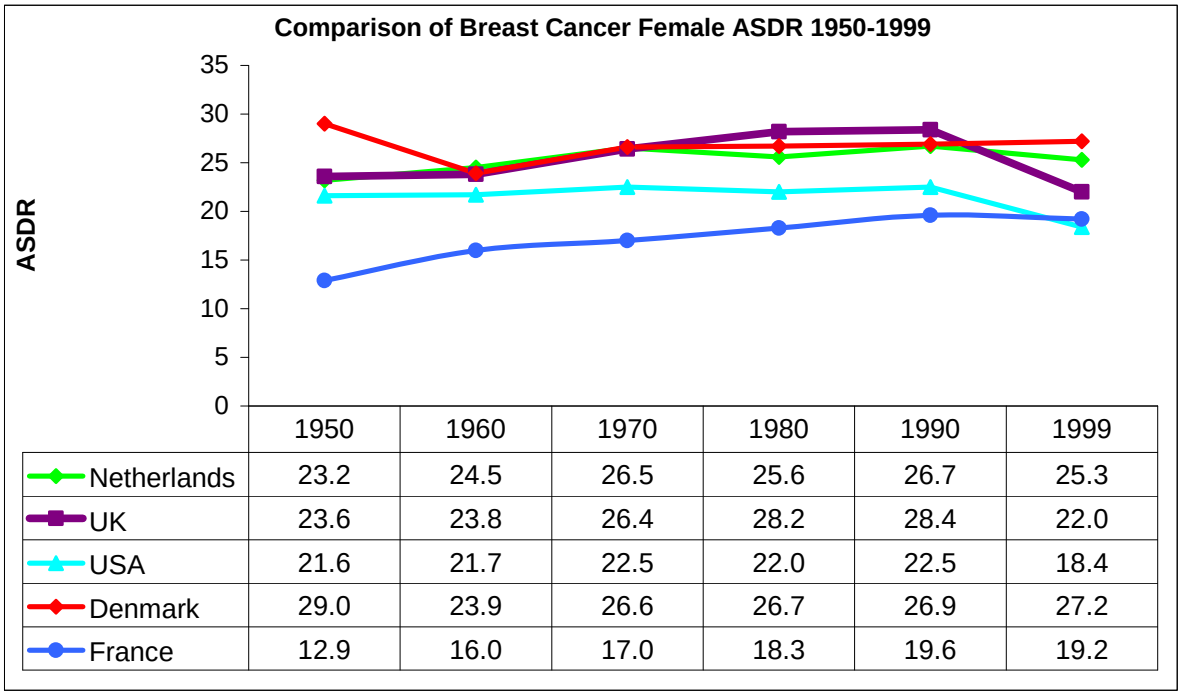
[590] World Health Organisation (2002), "WHO Mortality Data Base". Retrieved 1 October 2002, from: http://www.who.org
[591] World Health Organisation (2002), "WHO Mortality Data Base". Retrieved 1 October 2002, from: http://www.who.org

Illnesses: Cancer

The death rates in comparable economies (namely, the Netherlands, Denmark, France and America) show greater improvements than in the UK. As a result of Britain's smaller relative decline in the stomach cancer death rate, her comparative position has worsened. This scenario is also evident for British females, although there has been greater convergence between the five countries and therefore less of a difference in the stomach cancer death rate by the end of the twentieth century.

England's international performance for breast cancer has been more favourable. During the 1990s the UK experienced important declines in the death rate, which enabled Britain to improve their rank among the five countries.

Figure 6.11: Breast cancer age standardised death rate (ASDR) for world, females, 1950-1999[592]



	1950	1960	1970	1980	1990	1999
Netherlands	23.2	24.5	26.5	25.6	26.7	25.3
UK	23.6	23.8	26.4	28.2	28.4	22.0
USA	21.6	21.7	22.5	22.0	22.5	18.4
Denmark	29.0	23.9	26.6	26.7	26.9	27.2
France	12.9	16.0	17.0	18.3	19.6	19.2

Figures 6.9, 6.10 and 6.11 highlight that, despite the introduction of the NHS, Britain has not experienced any relative improvements for stomach cancer. And although the performance is more favourable for breast cancer sufferers, this was not achieved until the very end of the twentieth century and had still not reached levels associated with the claims (about a leading healthcare economy) of the British government about the superiority of the NHS.

[592] World Health Organisation (2002), "WHO Mortality Data Base". Retrieved 1 October 2002, from: http://www.who.org

There is speculation about the precise causes of these lags. The most common propositions all indicate shortcomings in the operations of the NHS: problems such as a variation in the provision of services across the country, under investment in specialists and equipment and a failure to modernise are blamed for Britain's relatively poor prognosis[593]. Hence, there is considerable evidence, summarised most recently in the UK Treasury's 'Walness Report', that health outcomes in the UK lag behind those in other advanced industrialised countries[594], and this evidence had facilitated the Treasury's decision to increase NHS expenditure[595].

Concurrently, the NHS could be complimented for its policies on mammography. The English screening framework falls within the consensus of efficient screening practices: the age range (50 to 69 years of age), the frequency of screening (every three years), and mammography methods are considered acceptable to women[596]. However it is important to recognise the debate about the efficacy of breast cancer screening. Some have claimed that, *"the effect of the screening programme is small, if any, and the balance between beneficial* (saved lives) *and harmful* (over diagnosis and false positive diagnosis due to carcinoma in situ) *effects is delicate"*[597]. Others have highlighted that, given the uncertainty about the effects of screening funds may be better investing in developing Calman-Hine proposals for specialist cancer care[598]. Both of these findings are especially applicable to pre-menopausal women, but towards the end of the twentieth century some started to consider whether these accusations are applicable to all women. The bottom line for the thesis is that it is not possible to conclude on the precise value of breast cancer screening here, but government provision of mammography provided women with an option for screening.

The government's biggest contribution to the alleviation of the standard of living burden of cancer came through their commitment to a better understanding of the disease (both through their sponsorship of a wide variety of working parties and also through their public awareness and target initiatives). The provisions provided by the National Health Service Act 1948, facilitated increasingly improved cancer treatment for the population. Finally, the strategic approach provided in the 'Health of the Nation' 1992 legislation indicates that the government had recognised that, although life expectancy had improved, individuals

[593] Government Papers, "The NHS Cancer Plan, A Plan for Investment, A Plan for Reform"
[594] Leon et al, "Understanding the Health of Scotland's Population in an International Context", p. 70
[595] Ibid
[596] Fletcher et al, "Report of the International Workshop on Screening for Breast Cancer", p. 1645
[597] Gotzsche & Olsen, "Is Screening for Breast Cancer with Mammography Justified?", p. 131
[598] Baum, "Breast Cancer Screening for Younger Women Is Not An Efficient Use of Resources", p. 1834

were still dying prematurely and having their quality of life impaired by potentially avoidable ill-health[599]. 'The NHS Cancer Plan 2000' marked an increase in the government's commitment to cancer care, which had previously been lacking. However, the policies and initiatives introduced by the government in the last years of the twentieth century indicate that the government was still failing to reach its potential, especially given the various retardations in the introduction of genuinely effective and helpful legislation for cancer sufferers.

6.4.2 Charities

Another important medium for generating improvements in the population's quality of life related to cancer was provided by the initiatives of cancer charities, which were especially pertinent as a result of the NHS shortcomings. Increasingly throughout the twentieth century there has been a growth in the number and diversity of such support mechanisms.

The largest in England (and Europe) was the Imperial Cancer Research Fund, which was established in 1902 and, throughout the twentieth century was prominent and active in a variety of arenas, all aimed at improving the quality of life for cancer sufferers[600]. The objectives of the Imperial Cancer Research Fund, which have been maintained throughout the twentieth century, highlight this[601]:

1. *To provide, extend and equip and maintain laboratories to be devoted to cancer research.*
2. *To encourage research on the subject of cancer in the UK or in the British Dominions beyond the seas.*
3. *To assist in the development of cancer research in various hospitals and institutions approved by the executive committee.*
4. *And, generally to provide means for systematic investigations into the causes, prevention, and treatment of cancer.*

Therefore, the key contributions of the Cancer Research Fund and numerous other cancer charities have been the following.

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[599] Department of Health, "The Health of Nations"

[600] This was then renamed the Imperial Cancer Research Fund in 1904. This organisation split into the British Empire Cancer Campaign for Research (BECC) and The Imperial Cancer Research Fund in 1923. In 1970 the BECC was renamed The Cancer Research Campaign. During the next 30 years these two fundamental charities worked in close collaboration and their cooperation culminated in 2002 when they merged and became Cancer Research UK. Cancer Research UK (2003) "History of Cancer Research". Retrieved 21 November 2003, from: www.cancerresearchuk.org

[601] Austoker, "A History of the Imperial Cancer Research Fund 1902-1986", p. 323

Illnesses: Cancer

To conduct research into the biology and causes of cancer: it is through efforts of this nature that many important breakthroughs have occurred. For example, the discovery of the BRCA1 gene was partially dependent upon charity funding from the UK's 'Cancer Research Campaign'[602].

To provide authoritative information about findings from their research: many cancer charities provide the public, government, commercial organisations and those responsible for cancer care with their research results. This is best highlighted by campaigns aimed at eliminating smoking in an effort to reduce lung cancer. These findings are also valuable for their potential to improve treatment and prevention strategies which can be implemented by governments and physicians.

Although, it could be argued that cancer charities were too conformist with the government rather than campaigning more heavily for early cancer legislation and perhaps a stronger emphasis on preventative initiatives. Hence, another criticism of cancer charities is that they should have concentrated more on preventative initiatives.

Through developing effective treatments that can improve the quality of life for cancer patients: research that is conducted by cancer charities is aimed at improving cancer cure rates and ultimately translating knowledge about this disease into effective treatments. Research is also directed towards improving the identification and diagnosis of cancer. This is best illustrated through the continual discoveries and implementation of screening for breast cancer. Hence, the demonstration in the 1980s of the efficacy of mammography in reducing mortality from breast cancer by twenty five to thirty percent led to the adoption of guidelines in a number of countries, including England, to introduce routine screening on a population basis[603]. Furthermore, many charities – initiated by the introduction of the UK National Society for Cancer Relief in 1911 – provide research into life prolonging medication and emotional support in an effort to ensure that for those patients whose disease cannot be cured, their quality of life is somewhat improved[604].

Cancer charities have continually developed throughout the twentieth century to remain on the leading edge of cancer research and provide a key support mechanism for the

[602] Science Blog (2003), *"Second Breast Cancer Gene Located"*. Retrieved 20 December 2003, from: http://www.scienceblog.com/community/older/1996/A/199600114.html
[603] Shapiro et al, "Breast Cancer Screening Programmes in 22 Countries: Current Policies, Administration and Guidelines", p. 735
[604] Lee, "Dates in Oncology", p. 81

government, doctors and patients. This dynamism has meant that charities are often the change agents and improvement implementers, which provides an example for the government and the most valuable support for cancer sufferers. However, it would seem that the approach of cancer charities was generally complimentary to government policy rather than substitutive and therefore it can be argued that cancer charities did not manage to initiate change or policy responses.

Cancer charities have played a crucial role in helping to improve the quality of life of the English population during the twentieth century, through providing diagnosis and treatment innovations, identifying lifestyle risks, providing guidelines and encouraging the government and doctors about the ideal codes of conduct. Additionally, the provision of information and condolence for individuals and families that have been affected by cancer has also contributed to improving the quality of life of cancer sufferers. However, a drawback is likely to have been that there were inequalities in these provisions. Gerard among others claims that for all charities, the rich receive as much as the poor and perhaps more convincingly, that the educated population enjoyed considerably higher levels of charity help[605]. This implies that to some extent there were inequalities in charity which indicates a setback in charity provision. An example of how this is manifest would be in the understanding and embracing of cancer prevention campaigns, e.g. anti smoking campaigns, which seem to have been more effective on richer social classes.

6.5 Medical Developments

The twentieth century provided some of the most far reaching developments in medical technology, many of which provided a substantial improvement in the understanding and treatment of cancer. When analysing medical developments and the impact they had upon the quality of life of breast and stomach cancer sufferers there are four key areas to consider: screening and scanning, radiotherapy, surgery, chemotherapy and hormone treatment. It will also be necessary to consider environmental factors[606] as changes in this broad category have provided many boosts to the welfare of cancer patients, both independently and synergistically with medical developments.

[605] Gerard, "Charities in Britain: Conservatism or Change?", p. 21
[606] 'Environmental' is used here in the widest sense, encompassing all those external influences impinging on an individual organism.

6.5.1 Screening and Scanning

"The surgical revolution that occurred during the twentieth century, which has enabled this 100 years of medical innovation to be coined 'The Golden Age of Surgery', would have been impossible without all manner of technological innovations that aid surgery"[607]. The breakthrough of greatest significance for the treatment of cancer (beyond surgical removal of tumours) was the discovery of X-rays[608]. Even in their most primitive form X-rays provided an indication of the presence of cancer, which enabled surgical removal of the cancer.

A further contribution of x-rays was the foundation they provided for more sophisticated visual diagnostics. In 1972 Hounsfield pioneered a system whereby x-ray beams could be resolved with computer assistance to produce a cross-sectional picture of the human body. The result – computer-assisted axial tomography (CAT) was a major breakthrough in the non-invasive diagnosis of disease, which provided an important contribution to the diagnosis and prognosis of cancer[609]. The development of magnetic resonance imagery (MRI), which is specifically useful for the detection of abnormalities in the soft tissue of the body, has also provided a boon to the identification, treatment and outcome of many cancers[610]. As a result of increasingly precise mechanisms for identifying tumours, the potential of successfully removing cancerous tumours before they became large and metastasised was improved with modern x-ray techniques, facilitated by the development and introduction of the CAT and MRI scans.

Modern x-ray techniques have also helped improve cancer prognosis through improved and frequent screening. In 1967 mammography for detecting breast cancer was introduced, and provided a much more efficient approach to the identification of cancerous tumours[611]. By the end of the twentieth century nearly all females between the ages of 50 and 69 were being screened for breast cancer on a triennial basis. For example, between March 1991 and April 1992, 71.3 percent of women aged 50 to 64 accepted screening[612]. This exceeds the target of a seventy percent acceptance rate and is considered to be extremely satisfactory[613].

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[607] Porter, "The Cambridge Illustrated History of Medicine", p. 233
[608] In 1895 Roentgen discovered that by passing a high voltage through a vacuum tube (perfected by Crookes), he could generate electromagnetic vibrations capable of penetrating human flesh and leaving an imprint on a photographic plate on the other side. By 1896 the X-ray was implemented for diagnostic purposes.
[609] Porter, "The Cambridge Illustrated History of Medicine", p. 243
[610] Ibid
[611] Porter, "The Cambridge Illustrated History of Medicine", p. 377
[612] Chamberlain et al, "National Health Service Breast Screening Programme Results for 1991-2", p. 353
[613] Ibid

A final contribution of the introduction of X-rays was the discovery of the effects of radioactivity upon cell destruction and ultimately radiotherapy.

6.5.2 Radiotherapy

By 1900, through the work of radium institutes (the London Radium Institute was founded in 1911, and numerous regional branches were developed soon after[614]), radiology journals and societies, there were more than a hundred diseases for which the new miracle cures had been used, although it was for cancer that the new therapies seemed to promise the most[615]. By the 1920s many surgeons were adopting radium therapy as a substitute or an adjunct to surgery[616]. In 1929 radium therapy was officially introduced in the UK as a cancer therapy and the National Radium Trust and Radium Commission were established[617]. By 1937 the Ministry of Health had drafted implementation policies for radiotherapeutic departments in general hospitals nationwide[618]. These developments were further fostered by the 1948 NHS Act, as this provided the initiatives and funds necessary to install hospitals with the necessary equipment and increase the number of centres providing treatment[619].

Despite the improvements in quality of life that these preliminary developments in radium therapy were yielding, the initial enthusiasm for radium therapy was largely uncritical and exposed patients, doctors and technicians to heavy doses of radiation, with results that were disastrous to many, most notoriously, Marie Curie[620]. This situation detracted from the overall contribution to improved standards of living provided by the introduction of radium therapy, as did the shortcomings in the NHS radiology provision objectives, which meant that, at no point in the twentieth century was radiotherapy care universally and equally accessible to the English population.

A more comprehensive improvement in quality of life was facilitated by radiation in the post World War Two era. Technological advances (driven by a better understanding about the process and dangers of radiation) during this time resulted in the replacement of radium

[614] Austoker, "A History of the Imperial Cancer Research Fund 1902-1986", p. xv
[615] Porter, "The Cambridge Illustrated History of Medicine", p. 243
[616] Hardy, "Health and Medicine in Britain since 1860", p. 104
[617] Lee, "Dates in Oncology", p. 94 and Austoker, "A History of the Imperial Cancer Research Fund 1902-1986", p. xv
[618] Austoker, "A History of the Imperial Cancer Research Fund 1902-1986", p. xv
[619] The National Archives: MH 80(14):Cancer Bill
[620] Hardy, "Health and Medicine in Britain since 1860", p. 104. Marie Curie died of leukaemia at the age of 67, which was undoubtedly a result of prolonged exposure to high energy radiation.

therapy by more precise and effective radiation therapy[621]. Since this time there have been continual innovations in the methods of utilising radiation for the treatment of cancer, such that by the close of the twentieth century this provided an important complement to surgery.

The ability to use a combination of complementary approaches improved the survival prospect of many breast cancer patients. The benefits of radiotherapy are more pronounced in many stomach cancer cases. Because of the nature of stomach cancer, the mild symptoms and long gestation period until the disease has progressed into an advanced stage means that many are faced with harrowing surgery needs, for example, a total gastrectomy or worse still, the inability of their cancer to be cured by surgery. Radiation therapy has provided a viable option for those sufferers who, during the twentieth century were faced with the aforementioned predicaments. Hence, in many instances of stomach cancer radiation therapy has prolonged the life of sufferers. Although this therapy does not provide a cure for stomach cancer it does provide a slight retardation to the cancer process, which albeit small is an improvement in the quality of life for sufferers of this disease.

6.5.3 Surgery

Cancer surgery was introduced in the 1880s. By the turn of the century there had been many extensions and improvements in technique, but it rarely yielded a cure for cancer[622]. This is because by the time the tumour had become identifiable (through primitive forms of diagnosis) the cancer had already advanced (metastasised)[623].

For the first half of the twentieth century breast cancer surgery was drastically destructive. Radical mastectomies were commonplace treatment for breast cancer from the 1890s until they began to be questioned in the 1960s. This procedure involved the surgical removal of the breast, all the lymph glands in the nearest armpit and the chest wall muscles, and ultimately the complete disfiguration of female breast cancer sufferers[624]. A further blow to quality of life was the non existence of any type of counselling for this disfiguration and the lack of life saving success yielded by this technique.

[621] Ibid
[622] Hardy, “Health and Medicine in Britain since 1860”, p. 103
[623] Ibid
[624] Porter, “A Medical History of Humanity From Antiquity to the Present”, p. 604

In 1964 these draconian methods started to be questioned. The first scientific study of breast-sparing surgery was begun at Guy's Hospital in London. This study compared tumour excision plus low dose radiotherapy with radical mastectomy[625]. This particular study did not achieve sufficiently powerful results to overturn the then current medical practices and mainstream beliefs about the superiority of radical mastectomies. However, the dispute continued and escalated, such that by the 1970s the treatment of breast cancer was one of the most argued subjects in medicine[626]. For example, a 1972 editorial in the British Medical Journal stated that *"there is more controversy about the management of breast cancer than almost any other topic in tumour therapy, and more so now than ever before"*[627].

By the 1980s it had been affirmed that breast conserving surgery worked and that it was just as effective as radical surgery in the treatment of breast cancer[628]. By the 1990s only 39 percent (approximately) of consultant surgeons would perform a radical mastectomy[629]. This development provided the largest single improvement in quality of life for twentieth century breast cancer sufferers. Additionally the increased utilisation of adjuvant therapy (i.e. conservative breast surgery complemented by chemotherapy, hormone therapy or radiotherapy) and the improvement in survival it facilitated consolidated this leap forward in standards of living.

The twentieth century history of stomach cancer provides a comparatively mundane and unsuccessful contrast. By the beginning of the century operations for the removal of stomach tumours had been introduced. These surgical interventions became increasingly sophisticated by the continual developments in the field of medicine[630]. For example, the ability to be more precise reduced the amount of stomach that had to be removed due to cancer. Furthermore, developments in radiotherapy and chemotherapy meant that it was sometimes possible to reduce the size of a tumour before surgery and consequently diminish the loss[631]. These advances in medical techniques would have provided small improvements in the quality of life of stomach cancer sufferers throughout the twentieth

[625] Texas Cancer Centre (2005), "The Cancer Breakthrough You've Never Heard Of: Breast Sparing Treatment Gains Momentum". Retrieved 25 November 2005, from: http://www.texascancercenter.com/cancerhistory.html
[626] Ibid
[627] British Medical Journal: Editorial, " Treatment of Early Carcinoma of Breast"
[628] Texas Cancer Centre (2005), "The Cancer Breakthrough You've Never Heard Of: Breast Sparing Treatment Gains Momentum". Retrieved 25 November 2005, from: http://www.texascancercenter.com/cancerhistory.html
[629] Morris et al, "Changes in the Surgical Management of Early Breast Cancer in England"
[630] Porter, "The Cambridge Illustrated History of Medicine", p. 245
[631] Health A–Z, (2002). Retrieved 1 October 2002, from: www.healthatoz.com

century, although the magnitude of improvement was still rather minimal by the close of the twentieth century.

6.5.4 Chemotherapy and Hormone Treatment

In the explosion of biochemical and pharmaceutical research which followed the Second World War, drugs were discovered that relieved a multitude of ailments[632]. The biggest contribution to cancer chemotherapy was the discovery that substances called nitrogen mustards selectively kill a particular type of cancerous cell[633]. This provided the foundation for a continued improvement in the sophistication of cancer chemotherapy, although cancer chemotherapy drugs were largely palliative and consequently, the contribution of chemotherapy to improved quality of life has experienced diminishing marginal improvements since the initial revolutionary introduction of this therapy in 1960[634].

Around the middle of the twentieth century the potential for hormone therapy to treat cancer was discovered[635]. Hormone therapies became increasingly used as adjuvant therapies for early stage breast cancer and provided a valuable alternative to chemotherapy (in older women [usually aged 50+] who have the type of breast cancer that is responsive to oestrogen blocking drugs – oestrogen receptor positive tumours)[636].

The scope of hormone therapy was consolidated with improvements in hormone drugs, particularly the breakthrough provided by Tamoxifen. This drug was introduced in the early 1970s and over the proceeding thirty years increasingly became the endocrine treatment of choice in breast cancer (especially during the 1990s when the risk-reducing benefits of Tamoxifen were unequivocally demonstrated)[637]. Tamoxifen has undoubtedly been one of the greatest success stories in the pharmacological management of breast cancer and has consequently provided many valuable improvements in quality of life for post menopausal, early stage, oestrogen receptor positive breast cancer[638].

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[632] Porter, "The Cambridge Illustrated History of Medicine", p. 152
[633] Porter, "The Cambridge Illustrated History of Medicine", p. 272
[634] Lee, "Dates in Oncology", p. 109
[635] Huggins discovered hormone therapy for the treatment of bladder cancer in his research into the physiology and biochemistry of prostate cancer in 1941. He also developed the use of hormones in treating female breast cancer. Porter, "The Cambridge Illustrated History of Medicine", p. 234
[636] Jonat, "Luteinizing Hormone –Releasing Hormone Analogues – The Rationale for Adjuvant use in Pre-Menopausal Women with Early Breast Cancer"
[637] Baum, "Tamoxifen – The Treatment of Choice. Why Look for Alternatives?"
[638] Ibid

Furthermore, during the last years of the twentieth century initial discoveries were being made, such as Goserelin, which signified the birth of a new generation of hormone drugs that will eventually add new vitality and diversity to the search for an improved endocrine therapy for early breast cancer[639].

The reduction in breast cancer mortality, which has been evident during the last fifteen years of the twentieth century in England, is partly attributable to the widespread adoption of adjuvant, systematic treatment, which includes hormone therapies, such as Tamoxifen[640]. Certain studies have attributed these treatments to a reduction in mortality of between 12.2 and 14.9 percent by the end of the twentieth century[641]. Hence, hormone therapies have provided a significant improvement towards quality of life for late twentieth century breast cancer patients, both through saving life years and through improving the general health of many females who have been living with breast cancer.

6.5.5 Environmental Factors

By the year 2000 it was thought that up to a third of all cancer deaths were related to dietary factors[642]. Knowledge about the potential influence of environmental factors became increasingly prominent as the century unfolded and the cure for cancer persisted to elude modern medicine. Environmental factors resemble potential preventions for cancer, which, at the close of the twentieth century, remained the most promising mechanism for superseding cancer.

Many authors have emphasised the importance of dietary factors in the prevention of cancer, especially stomach cancer. For example, dietary factors which are related to improvements in food storage and handling are thought to be the leading catalysts for the worldwide reductions in stomach cancer mortality[643]. Conversely, the increase in breast cancer has often been partially attributed to increased fat consumption[644]. The culmination of these discoveries meant that, by the end of the twentieth century, environmental factors featured as one of the key strategies in the British government's battle against cancer. For example, 'The NHS Cancer Plan 2000' five-a-day programme and continued initiatives to

[639] Ibid
[640] Baum, "Tamoxifen – The Treatment of Choice. Why Look for Alternatives?"
[641] Blanks et al, "Effects of the NHS Breast Screening Programme on Mortality from Breast Cancer in England and Wales 1990-8: Comparison of Observed with Predicted Mortality", p. 668
[642] Government Papers, "The NHS Cancer Plan, A Plan for Investment, A Plan for Reform", p. 26
[643] Hoel et al, "Trends in Cancer Mortality in 15 Industrialized Countries, 1969-1986", p. 315
[644] Hoel et al, "Trends in Cancer Mortality in 15 Industrialized Countries, 1969-1986", p. 316

reduce smoking[645]. As a result of twentieth century economic and technological advances, this type of diet (high in fruit, vegetables and fibre, and low in fat, sugar and salt) has become increasingly achievable.

An additional environmental harm that has received increased awareness during the twentieth century is industrial/ work related carcinogens. Some of these cancers are now so well established that they are designated as 'prescribed diseases' entitling the victim to possible compensation[646], e.g. skin cancer from employment in soot, tar and mineral oil industries[647].

Despite the developments in recognising these potentially harmful environmental factors, progress to reduce these adverse factors had been slow in twentieth century England. There were very few organisations that represented the broader public interest and lobbied the government accordingly. This marks a contrast with other developed countries, for example, America, where environmental and consumer lobbies are larger, more broadly based and have formed better alliances with trade unions, which have been able to use their power to achieve more protection from these environmental externalities[648].

Finally, during the twentieth century it has become increasingly recognised that the general environment is a potential source of carcinogens. I.e. that people are not only potentially at risk from their occupations and the commodities they consume but also from the air they breathe and the water they drink. This is best illustrated by the marked difference between higher urban and lower rural rates of cancer in England during the twentieth century[649].

Improvement in environmental factors provided an ebb in cancer, but not a cure. Because of the perceived trade-offs (in the short term in order to reduce long term risk) and the inability of many to change their circumstances (largely the most deprived who are faced with an excess cancer risk as a result of the aforementioned environmental factors) a better understanding of environmental factors was not able to provide any kind of cure for the twentieth century English population.

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[645] Ibid
[646] Doyal et al, "Cancer in Britain: The Politics of Prevention", p. 17
[647] Ibid
[648] Doyal et al, "Cancer in Britain: The Politics of Prevention", p. 50
[649] Ibid

6.6 Lack of Progress Considerations

Despite the research and funding that has been dedicated to cancer (cures, screening, diagnosis, treatment) the returns have been scanty. For example, the prognosis and quality of life faced by stomach cancer sufferers was still poor at the end of the twentieth century. The outlook was more favourable for breast cancer sufferers. However, breast cancer is the most heavily funded and researched cancer and therefore, in this respect the outcome is still relatively meagre compared to the investments towards curing this disease. Nevertheless, the quality of life for breast cancer sufferers has improved favourably during the twentieth century and the breakthroughs in 'prevention' and treatment for breast cancer are commendable. This marks a stark contrast with stomach cancer. The key frustration for stomach cancer improvements is that there is no mechanism for early identification of the disease. Furthermore, because stomach cancers engulf a major bodily organ there are few options for remedy at a late stage. These are the reasons why quality of life improvements have not been more pronounced for stomach cancer during the twentieth century.

Health providers, charities and governments ought to be commended for their efforts towards trying to improve the standards of living of cancer sufferers. Their efforts during the twentieth century, increasingly as the century unfolded provided many valuable improvements in the quality of life associated with stomach and particularly breast cancer. The main drawback was that despite substantial investments, the benefits that they yielded were very low. This was largely a result of the complicated and poorly understood nature of cancer throughout the twentieth century and numerous weaknesses in the functioning of the NHS, which have been detailed above. A final factor that could have been improved, and would subsequently have improved the overall prognosis from breast and stomach cancer, were the socioeconomic inequalities in health that persisted and increased during the twentieth century.

Sigerist (1956) stated that *"in any given society the incidence of illness is largely determined by economic factors"* and that *"the problem of public health is ultimately political"*[650]. Despite the introduction of the NHS in 1948, socioeconomic mortality [and morbidity] differences have persisted and often increased. E.g. for the ten most common cancers (including breast and stomach), Schrijvers (1995) identified much better five-year survival rates for affluent patients[651]. Tomatis (1995) went as far as claiming that *"the total*

[650] Tomatis, "Socioeconomic Factor and Human Cancer"
[651] Schijvers et al, "Deprivation, Stage at Diagnosis and Cancer Survival"

incidence of cancer at all sites is greater in lower socioeconomic groups"[652]. Kogevinas et al (1991) found that both male and female council tenants had significantly worse survival than owner-occupiers[653]. Moreover, other studies have found that this inequality is exacerbated for cancers with poor prognosis (e.g. stomach cancer), as these are more prevalent in the lower socioeconomic groups[654].

These social class inequalities are not an English phenomenon and are evident in other, comparable countries. However the extent of the social class inequality seems to be more severe in the UK[655]. The twentieth century evolution in social class inequality adds a further blow to NHS progress.

The breast cancer social class gradient was consistently in favour of the professional classes, whereby their five years survival rate was higher than their social class V counterparts. Stomach cancer illustrates a different trend: the survival rate was equal across all social classes, but was marginally more favourable for lower classes, i.e. social class V, in the 1970s and probably earlier. This margin was eliminated by the later years of the twentieth century (the 1980s), when the higher social classes, e.g. social class I, were enjoying equal or marginally greater years of survival. A noteworthy point to recognise for stomach cancer, when evaluating the social class mortality gradient, is the prevalence of stomach cancer in social class V, which was significantly higher than for social class I. The social class survival rates for breast and stomach cancer are shown in the table below.

[652] Tomatis, "Socioeconomic Factor and Human Cancer"
[653] Kogevinas et al, "Socioeconomic Differences in Cancer Survival"
[654] Ibid and Lipworth et al, "Socioeconomic Factors in the Prognosis of Cancer Patients"
[655] Logan, "Cancer Mortality by Occupation and Social Class 1851-1971", p. 109 and 122

Illnesses: Cancer

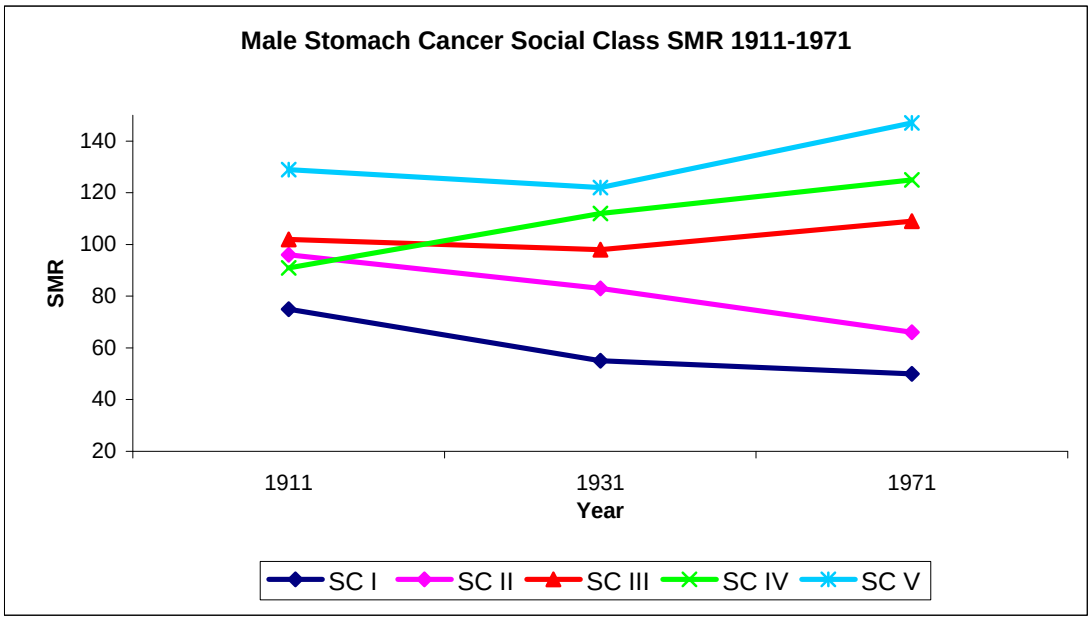
Table 6.6: Five year survival rate for breast and stomach cancer by social class (I to V), 1971-1990 (%)[656]

	1971-1975					**1976-1980**					**1981-1985**					**1986-1990**				
Social Class	I	II	III	IV	V	I	II	III	IV	V	I	II	III	IV	V	I	II	III	IV	V
Breast Cancer	52	49	47	45	42	57	54	51	50	47	60	56	54	52	49	63	60	59	56	53
Stomach Cancer	4	4	4	4	5	5	5	5	5	5	8	7	6	6	7	8	8	7	7	8

Table 6.6 highlights a clear social class gradient for survival from breast cancer, which was consistently about 10 percent more favourable for the top social class versus the lowest. This relationship was not evident for stomach cancer, where there are marginal differences between the survival rates across the social classes. Table 6.6 also highlights that very few sufferers survive stomach cancer. This point is particularly important when the prevalence of stomach cancer by social class is considered, whereby stomach cancer is more prevalent in the lower social classes. Therefore, there is some form of a social class gradient for stomach cancer, which is not highlighted above but will be illustrated in the Figures below, which represent the standardised mortality rate for stomach cancer (and indicates the greater prevalence of this disease in lower social classes). The standardised mortality rate considers the level of mortality relative to the population. For males and females there was generally more stomach cancer deaths in social class V than I and hence there is a vivid social class gradient for stomach cancer mortality.

[656] Coleman, "Cancer Survival Trends in England and Wales, 1971-1995: Deprivation and NHS Region"

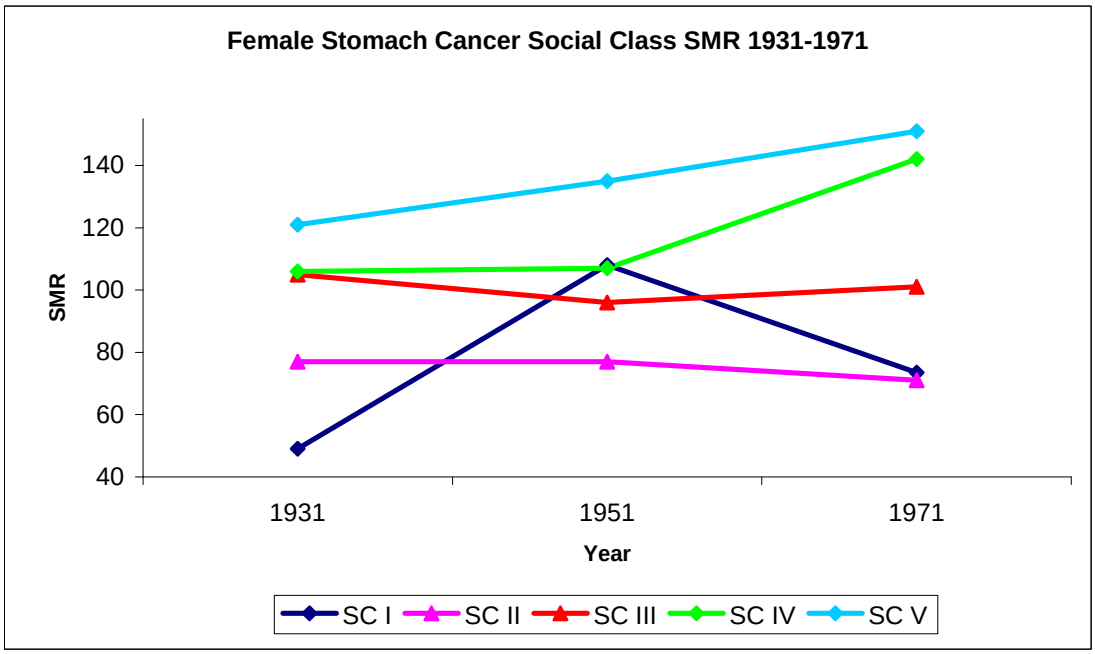
Figure 6.12: Stomach cancer standardised mortality rate (SMR) by social class, males, 1911-1971[657].



With the exception of 1911 (where stomach cancer mortality was higher in social classes II and III than IV), there is an ordinal and increasingly substantial social class gradient associated with stomach cancer in English males. The social class gradient increased throughout the period largely as a result of the decline in stomach cancer between 1931 and 1971 in social classes I and II in conjunction with the increase in social classes III to V.

[657] Logan, "Cancer Mortality by Occupation and Social Class 1851-1971", p. 30

Figure 6.13: Stomach cancer standardised mortality rate (SMR) by social class, females, 1931-1971[658]



The relationship for female stomach cancer mortality is not as clear as that for males. This is largely due to the anomaly of 1951 mortality in social class I (for which there is no obvious explanation) and also because mortality was greater in social classes I than II in 1971. The data used for the above table is somewhat hybrid (1931 considers only married women, 1951 considers only single women and 1971 contains an average of both) and this may be the cause of the anomalies. However there is still an evident general trend of an increasing social class female stomach cancer mortality gradient.

The causes of this socioeconomic cancer gradient are not as pronounced as the social inequalities themselves. This is because of the difficulty in measuring different social classes and also because of the incomplete knowledge of cancer causes, which was still limited at the end of the twentieth century. However, there is a general consensus in the literature about the most likely causes of the gradient, which are summarised in Table 6.7.

[658] Ibid

Table 6.7: Summary of major studies and their findings about the cause of social inequalities in cancer survival[659]

Study	**Key findings**
Gillis & Hole (1996)	• There is a substantial survival benefit for patients cared for by a specialist. • Specialist care is more commonly received by higher social classes.
Kogevinas (1991)	• Differences in treatment. • Time of diagnosis. • Stage at presentation: delay in seeking treatment in lower social classes is a major contributing cause of the gradient. • Poor host resistance among the deprived.
Linden (1996)	• Stage at diagnosis.
Richards et al (1997)	• Variations in the management of breast cancer in clinics and hospitals and in surgical standards, whereby the affluent receive better quality treatment.
Schrijvers et al (1995)	• Poor host resistance among the deprived (co-morbidity, adverse nutritional status, poor social support, likelihood of anxiety and depression due to cancer, are all more severe in deprived classes).
Wells & Horm (1992)	• Stage at diagnosis strongly influences cancer survival. Women of lower socioeconomic status are more likely to be diagnosed with breast cancer at a later stage.

Later stage at diagnosis, poorer host resistance (related to nutritional and health status) and a variation in cancer treatment were largely responsible for the socioeconomic cancer gradient at the end of the twentieth century, when social inequalities in cancer became a major public health issue. The variation in quality and provision of cancer care (as a result of NHS weaknesses) is a key factor when considering England's lag behind other comparable, developed countries[660].

It should be noted that the lack of improvement in the quality of life associated with cancer during the twentieth century could partially be a result of non-health and welfare related factors. Firstly, the ageing of the population as a result of the nature of degenerative diseases, which has meant that people are living longer and are therefore, increasing the scope for developing cancer, as a result of the nature of degenerative diseases. Or, as Davis

[659] Gillis & Hole, "Survival Outcome of Care by Specialist Surgeons in Breast Cancer: A Study of 3,786 Patients in the West of Scotland" and Kogevinas et al, "Socioeconomic Differences in Cancer Survival" and Richards et al, "Inequalities in Breast Cancer Care and Outcome" and Schrijvers et al, "Deprivation and Survival from Breast Cancer" and Wells & Horm, "Stage at Diagnosis in Breast Cancer" and Linden, "The Influence of Social Class in the Survival of Cancer Patients"

[660] Iliffe, "The NHS: A Picture of Health?", p. 88

et al (1990) have claimed *"because the elderly have had more opportunity for exposure to carcinogens in their lifetime"*[661].

Therefore, although the problems associated with cancer could have been more comprehensively remedied by the government, there are aspects of this disease that continue to frustrate all of those involved in the fight against it. The key problem is the failure in finding a cure and understanding this disease more comprehensively. And the nature of the ageing of the population has also exacerbated the twentieth century burden of cancer.

6.7 Summary

The above analysis has highlighted that the standards of living associated with breast and stomach cancer improved over the twentieth century, although to a lesser extent for stomach cancer. These advances are important as quality of life improved from a 'poor' level in 1900 to a 'good' level for breast cancer and a 'fair' level for stomach cancer by the year 2000.

The prevalence of breast cancer increased considerably during the twentieth century, while it declined for stomach cancer. This worsening situation was aided by the improvement in the age distribution and also survival prospects of these diseases (although less so for stomach cancer). Furthermore, enhancements in the quality of life experienced by sufferers of stomach and breast cancer have also helped to relieve the aggregate burden of these diseases upon overall standards of living.

Medical developments, increased medical practitioner skills, improvements in the interaction and understanding of environmental factors, government initiatives and the work of charities all played key roles in initiating these quality of life improvements.

[661] Grulich et al, "Is the Apparent Rise in Cancer Mortality in the Elderly Real? Analysis of Changes in Certification and Coding of Cause of Death in England and Wales, 1970-1990"

PART III

This part of the thesis reports the results, which are derived from the previous analysis. Chapter 7 will also provide the conversion of the previous qualitative analysis to quantitative summary results.

The first stage of these results will concentrate on the construction of a 'quality adjusted life year' (QALY) measure from the previous qualitative analysis. The QALY that is derived below is the result of an original process, which was necessary because of the central questions of the thesis (i.e. the historical health and welfare quality of life of sufferers had to be approximated). The best solution was to consider the historical health and welfare quality of life in detail, for a sample of illnesses. The next stage was to apply a mechanism that transformed this detailed qualitative analysis into a series of quantitative indices. This was achieved through applying the EuroQol standardised spectrum of development to all the key quality of life features (see Chapter 3: Tables 3.5, 3.6 and 3.7 for an outline of the key EuroQol variables) in the qualitative analysis. This yielded a series of results (or EuroQol ranks which can be converted into QALY indices) about the quality of life for different illnesses, eras and aspects of health and welfare standards of living, which are comparable across all illnesses and eras. The justification for utilising this approach and a more detailed explanation about the construction of this methodology (i.e. the conversion of the qualitative analysis into a quantitative QALY index through applying EuroQol) is provided in Chapter 3: 3.5 *QALY Estimating Process* and 3.6 *QALY Value Accreditation.*

Hence, the QALY in this thesis (which represents the quantitative index) will be based upon the previous qualitative analysis. This information will be transformed from qualitative into quantitative through the thesis consistently (for all illnesses and eras in the thesis) gauging the qualitative results on the EuroQol spectrum. The EuroQol results (or ranks) will then be evolved into QALY weights. The range of EuroQol ranks (and how they translate into associated QALY weights) is shown below.

Results

Table 7.1: EuroQol standardised spectrum and ranks and corresponding QALY values

EuroQol standardised spectrum	**EuroQol Rank**	**Corresponding QALY value**
Complete quality of life	1	1
Good quality of life	2	0.8333
Fair quality of life	3	0.6667
Some quality of life	4	0.5000
Poor quality of life	5	0.3333
No quality of life	6	0.1667

The QALY results in Table 7.1 are achieved through converting EuroQol ranks into QALY ranks. On EuroQol there are 6 possible values between 1 and 6: 1, 2, 3, 4, 5, or 6. For the QALY there are 6 possible values between 0 and 1: 1, 0.8333, 0.6667, 0.5, 0.3333, 0.1667. These QALY rank are equivalent to their respective EuroQol rank, but expressed on a different scale.

This exercise provides one of the thesis' key contributions to knowledge. Hence, the QALY is crucial for making quantitative considerations about health improvements which is facilitated by applying the EuroQol standardised spectrum of development to the qualitative analysis of all illnesses in all eras in the thesis.

Hence, the key value of developing and utilising a standardised spectrum, namely EuroQol, is so that the twentieth century developments in quality of life (documented in Part II of the thesis) can be converted into QALYs, so that morbidity can be included in an extended (quantitative) willingness to pay (or QALE) model, which includes mortality as well as morbidity (which is comparable across all illnesses and era). This process will be utilised (in Chapter 7 and 8) in order provide quantitative answers to the major questions of the thesis: to what extent have improvements in mortality and morbidity improved quality of life and contributed to better standards of living and economic growth in twentieth century England? And, how valuable (in monetary terms) are the improvements in quality adjusted life expectancy (QALE gains)?

Results

The development and implementation of this new approach to measuring health (mortality and morbidity), both the qualitative and quantitative aspects of historical health and welfare related quality of life, will enable the thesis to provide robust estimates about the contribution of improved health to standards of living, which will provide an original contribution to the existing literature. Furthermore, these considerations and the results that the QALE methodology will yield represent a more precise health measurement methodology in adherence to the criteria mentioned in earlier sections of the thesis (see *'Representational Measurement'* in Chapter 2.2.7) and a more comprehensive 'Fisherian' measure of twentieth century English economic development (explained under the rationale of *'Willingness to Pay'* in Chapter 3).

7. Qualitative Findings: EuroQol Summary and Quantitative (QALY) Transition

The following chapter will consolidate the findings of Part II through providing the EuroQol summary of these results, which can then provide overall health and welfare associated quality of life (QALY) approximations. This chapter will summarise the evolution of the quality of life associated with blindness, tuberculosis and cancer during the twentieth century, both qualitatively and quantitatively by presenting the range of EuroQol scores and the associated QALY. This will provide a verbal and numerical answer to the key underlying question of the chapter: what was the overall quality of life for sufferers (in the years 1900, 1925, 1950, 1975 and 2000)?

This will provide the EuroQol standardised comparative analysis – between the government, charity, medical developments, pain and discomfort, and ability to lead a normal life variables – of the developments that were made for blindness, tuberculosis and cancer quality of life, which are used to construct a corresponding QALY index.

The EuroQol and subsequent QALY values that are presented here represent what the author deems to be the most appropriate value, from a conservative standpoint (this is best highlighted by the QALY result comparison in Chapter 3: Table 3.9). The QALY values outlined here pertain to the ‘Mid’ values utilised in later stages of the quantitative analysis.

7.1 Blindness

7.1.1 Twentieth Century Qualitative Results

The table below provides a summary of the EuroQol rank results for the quality of life variables (see Chapter 3: Table 3.5 and 3.7 for a detailed explanation of these variables), for blindness in the key eras of the twentieth century considered in the thesis.

Table 7.1.1: EuroQol results for blindness and EuroQol aggregate average conversion into QALY

Blind: EuroQol Variable	**Blind: EuroQol Score and Equivalent QALY**				
	1900	**1925**	**1950**	**1975**	**2000**
Government initiatives and help	6	3	3	3	3
Recognition/ awareness	5	3	3	3	2
Health developments	6	5	3	2	2
Status	5	4	3	3	3
Ability to lead a normal life	5	4	3	3	3
Aggregate Average EuroQol Score	5	4	3	3	3
Equivalent QALY	0.3333	0.5000	0.6667	0.6667	0.6667

Note: In Table 7.1.1 the aggregate average is calculated as the average score (to the nearest whole number) of the key variables: government initiatives and help, recognition/ awareness, health developments, status, ability to lead a normal life. Ability to lead a normal life variable score is calculated as the average of the sub-variables in this category, which are presented below in Table 7.1.2. For the definitional interpretation of the EuroQol ranks shown in Table 7.1.1 see Table 7.1, above.

Table 7.1.1 illustrates that, in 1900 there was 'no' quality of life (EuroQol rank = 6) facilitated for the blind by *'government initiatives and help'*. When the Blind Persons Act was introduced in 1920 government aid improved considerably and the blind population experienced 'fair' quality of life (EuroQol rank = 3) related to government help. However this state of legal recognition and help did not improve enough between 1925 and 2000 to move the blind to a higher indifference curve (i.e. an extra mark on the spectrum). There were numerous Acts designed to help the blind, which were seemingly very comprehensive, but none of these were effective enough to become more far reaching than the 1920 Blind Persons Act. This meant that the standards of living for the blind were only partially improved by government legislation: from 'no' to 'fair' quality of life during the twentieth century.

Results: Qualitative: Blindness

After the introduction of the 1920 Blind Persons Act the blind experienced a considerable improvement in their levels of *'recognition'* (from 'poor' quality of life to 'fair' quality of life). During the last decade of the twentieth century a stream of legislation pertaining to equal rights helped change the perception of the disabled and improved recognition of the blind, such that by the year 2000 the blind were experiencing 'good' quality of life regarding their levels of recognition (shown in Table 7.1.1 as a EuroQol rank of 2 in 2000, for 'recognition /awareness').

The introduction of the 1920 Blind Persons Act meant that by 1925 the blind were experiencing 'some' quality of life in their *'status'* compared to 'poor' quality of life which was previously the case. By 1950 the blind had achieved 'fair' quality of life regarding their standing, largely as a result of the improvement in employment opportunities (discussed below). Finally, although there were numerous government Acts which extended the achievements of the 1920 Blind Persons Act (especially during the 1990s) these attempts at improving the status of the blind population were largely unsuccessful, as the blind status did not improve beyond the 1950 level of 'fair' quality of life for 1950-2000.

The state of *'health developments'* at the start of the twentieth century contained no prevention or provisions for treatment of blindness ('no' quality of life). Increased awareness about infectious diseases causing blindness, the introduction of safe and effective treatment, and improvements in the prevention of blindness facilitated 'fair' quality of life for blind health by 1950. These breakthroughs and developments were enhanced during the last quarter of the twentieth century through further technological developments, e.g. cataract surgery, so that by 2000 the blind were enjoying 'good' quality of life levels of prevention and treatment of blindness (shown in Table 7.1.1 as a EuroQol rank of 2 in 2000, for 'health developments').

The value of the improvements in these (prevention and treatment of blindness) *'health developments'* are best illustrated by (approximations about) the increasing average age of onset of blindness: in 1900 16 percent of the blind were over 70, in 1950, 24 percent and by 2000, 69 percent of the blind population were over the age of 75. Consequently there would seem to be much fewer years spent in blindness: in 1900 the average number of years of blindness was about 16 years versus 11 years in 2000 (this is especially impressive as life expectancy increased by 27 years between 1900 and 2000). Even though these numbers are approximate, due to the

assumptions that had to be made in order to consider the prevalence by age of blindness (see Chapter 4), this trends is very important for the welfare of the blind.

The *'ability to lead a normal life'* also improved for the blind population. This variable comprises: employment, wages and education. This breakdown and the associated EuroQol scores are shown in the table below.

Table 7.1.2: EuroQol results for sub-variables in 'Ability to lead a normal life' for disability

Blind: EuroQol Sub-Variables	**Blind: EuroQol Score**				
	1900	**1925**	**1950**	**1975**	**2000**
Ability to lead a normal life	5	4	3	3	3
Employment	6	5	4	4	4
Wages	5	5	3	3	3
Education	4	3	3	3	2

The development of help and equality for the blind in the labour market was one of the slowest aspects of socio-economic quality of life to improve. Table 7.1.2 highlights that the blind experienced 'no' quality of life in employment in 1900, and this did not improve to 'some' quality of life levels until 1950. The developments that occurred between 1950 and 2000 were not far reaching enough to improve the blind's quality of life associated with employment, beyond the 1950 level of 'some' quality of life.

The increase in the blind's wages provided an important development in equality, such that by 1950 the blind were experiencing 'fair' quality of life in wages. During the last quarter of the twentieth century there were no far reaching improvements in equality of wages upon the 1950 level of 'fair' quality of life and this remained the scenario for blind wages for the second half of the twentieth century.

Education was one of the most developed and equal areas for blind welfare during the twentieth century. Table 7.1.2 highlights that as early as 1900 the blind were enjoying 'some' quality of life in education. Between 1925 and 1975 blind children in education experienced continual improvements, so that during this time they enjoyed 'fair' quality of life in the provision and standards of blind education. The 1981 Education Act illustrates an initiative to provide improved

equality, opportunity and standards for educating the blind, and although this was not overly successful, this was still a development that marked an improvement in blind quality of life, which meant that by the end of the twentieth century the blind were enjoying 'good' quality of life in education, even if the result of those in higher education were unsatisfactory.

Therefore, the culmination of these developments (or lack thereof) meant that the '*aggregate average quality of life'* faced by the blind evolved from; 'poor' in 1900 to 'some' by 1925 and peaked at 'fair' in 1950 to 2000 (shown in Table 7.1.1).

This final aggregate EuroQol rank for the quality of life associated with blindness can now be converted into a QALY value. Table 7.1 provides the translation from EuroQol rank to QALY value. For a detailed explanation about the nature and appeal of this process, see Chapter 3: 3.5 *QALY Estimating Process* and 3.6 *QALY Value Accreditation.*

Therefore, Table 7.1.1 highlights that the blind QALY improved from 0.3333 of a healthy life year (which is represented as 1) to 0.6667 of a healthy life year. Hence, in 1900 the collection of health and welfare standards of living meant that the blind only enjoyed about one third of a healthy life year. By the year 2000, improvements in health and welfare enabled the standards of living for the blind to improve to about two thirds of a healthy life year. This numerical index will be utilised below, in the wider QALE methodology in order to estimate the value of the above improvements in the quality of life associated with blindness (shown here, as the improvements in the QALY from 0.3333 in 1900 to 0.6667 in 2000).

Finally, although the developments made in the quality of life for the disabled are not as far reaching as those experienced by disease sufferers, these improvements are still commendable. This will be highlighted in the following chapters of the thesis.

7.2 Tuberculosis

The table below provides a summary of the EuroQol rank results for the quality of life variables (see Chapter 3: Table 3.5 and 3.7 for a detailed explanation of these variables), for tuberculosis for the key eras of the twentieth century considered in the thesis.

Table 7.2.1: EuroQol results for tuberculosis and EuroQol aggregate average conversion into QALY

Tuberculosis: EuroQol Variable	**Tuberculosis: EuroQol Score and Equivalent QALY**				
	1900	**1925**	**1950**	**1975**	**2000**
Government initiatives and help	5	4	3	3	3
Recognition/ awareness	4	3	2	3	3
Health developments	5	5	4	2	2
Understanding	5	3	2	2	2
Treatment	5	5	5	2	2
Cure	6	6	4	1	1
Pain and discomfort	5	4	3	2	2
Ability to lead a normal life	5	5	4	1	1
Financial burden	5	4	3	1	1
Social difficulties	5	5	4	1	1
Anxiety/ depression	5	5	4	2	2
Aggregate Average EuroQol Score	5	4	3	2	2
Equivalent QALY	0.3333	0.5000	0.6667	0.8333	0.8333

Note: In Table 7.2.1 the aggregate average is calculated as the average score (to the nearest whole number) of the key variables: government initiatives and help, recognition/ awareness, health developments, pain and discomfort, ability to lead a normal life. Health developments and ability to lead a normal life variable scores are calculated as the average of the sub-variables in each category. For the definitional interpretation of the EuroQol ranks shown in Table 7.1.1 see Table 7.1, above.

Table 7.1.2 highlights that during the twentieth century the implications of having tuberculosis improved substantially: in 1900 tuberculosis sufferers experienced ‘poor’ quality of life versus 2000 when the quality of life associated with tuberculosis was ‘good’. The variable EuroQol rank results in Table 7.2.1 are indicative of tuberculosis being a rampant, incurable, poorly

understood and life threatening illness, which inspired widespread fear and anxiety and a significant threat to the public health of the nation at the beginning of the twentieth century. And, by the year 2000 tuberculosis was an innocuous disease, which, in most cases was relatively easily cured and short-lived.

Unlike the other illnesses considered in the thesis, tuberculosis had standard of living ramifications beyond the individual sufferer, because in addition to debilitating the health of the sufferer, tuberculosis was a destructive social force. Therefore, there are numerous developments which are far reaching but do not appear in Table 7.2.1 (and in the quantitative results below), for example, the contribution to the aggregate population of the reduced infectiousness of tuberculosis. These are not direct features of the thesis and cannot be included in the methodology, however it should be noted that there were additional values to the documented tuberculosis reduction. This also represents another instance where the thesis Extended Results are lowest bound estimates.

7.2.1 Nineteen Hundred

Table 7.2.1 highlights that in 1900 the quality of life burden generated by tuberculosis was substantial. There was relatively little *'government help'* or understanding about the specific problems and distinctive needs of tuberculosis sufferers (embodied by 'poor' quality of life associated with *'government recognition and help'* in Table 7.2.1: EuroQol rank = 5). Although tuberculosis had been prevalent for nearly a century, and charities and committees were already active by 1900 (embodied by 'some' quality of life associated with *'recognition/ awareness'* in Table 7.2.1: EuroQol rank = 4), understanding of the aetiology of tuberculosis was still very limited (embodied by 'poor' quality of life associated with *'health developments'* in Table 7.2.1: EuroQol rank = 5).

The *'pain and discomfort'* associated with this tuberculosis was significant and in 1900 there was relatively little that could be done to abate the symptoms. Because of the method of treatment this physical pain was also likely to have been matched by emotional and mental distress as a result of the segregation that many experienced as a course of their treatment, and the financial burden that was placed upon a family, particularly if the sufferer was the breadwinner, and the *'anxiety'* associated with the contraction of a disease that was so strongly

associated with no cure. This burden is represented by the 'poor' quality of life associated with tuberculosis sufferers' *'ability to lead a normal life'*, shown as EuroQol = 5 in Table 7.2.1.

This meant that the overall quality of life experienced by tuberculosis sufferers was 'poor' in 1900. Standards of living had been slightly improved by the work of charities, which helped promote recognition and aid for the condition, although this could have been more effective and far reaching in some instances. Most detrimental to tuberculosis sufferers' quality of life was the complete lack of any kind of cure, which exacerbated all of the other quality of life variables.

7.2.2 Nineteen Twenty-Five

The most important quality of life variable, the cure for tuberculosis, continued to elude the population, but there were evident developments made in its pursuit. By 1925 understanding of tuberculosis had fundamentally advanced, although this had not managed to yield any positive results for the treatment of tuberculosis at this time. Therefore, by 1925 quality of life associated with *'health developments'* had still not managed to improve from 'poor' levels, although the foundations for a later improvement were evident.

This phenomenon was also evident with tuberculosis sufferers' quality of life associated with their *'ability to lead a normal life'*. The key reason for this is that the social difficulties, anxiety and depression invoked by tuberculosis had not declined and therefore, quality of life related to the ability to lead a normal life remained 'poor' in 1925.

By 1925 *'government initiatives and help'* had begun to develop as legislation began to display that the state had adopted a more comprehensive stance towards tuberculosis. E.g. the 1921 Public Health (Tuberculosis) Act included after-care legislation for tuberculosis sufferers. The increase in government involvement in welfare issues associated with tuberculosis welfare provided an improvement in quality of life for tuberculosis sufferers, from 'poor' in 1900 to 'some' by 1925 (shown in Table 7.2.1 as an improvements in the EuroQol rank from 5 to 4).

The state also ought to be partially credited with the improvement in *'pain and discomfort'* associated with tuberculosis (which, like government help above, improved from 'poor' to

'some' quality of life between 1900 and 1925). The most important mechanism for this improvement is related to the expansion and improvement in treatment facilities for tuberculosis, generated by the state subsidising the provision of treatment for tuberculosis. E.g. under the 1911 Finance Act £1,500,000 was provided for the building of sanatoria and, under the 1912 National Health Insurance Act funds were provided for the treatment of tuberculosis and, the 1916 Tuberculosis (Domiciliary Treatment in England) Order enabled treatment in the home in an attempt to alleviate the shortages in sanatoriums.

During this period the efforts and campaigns of charities continued to provide a contribution to the plight of tuberculosis sufferers and as a result continued to yield a 'fair' quality of life for tuberculosis patients, regarding *'recognition and awareness'* (this is shown as EuroQol = 3 for recognition/ awareness in 1925 in Table 7.2.1).

The culmination of these developments and persistent frustrations was an improvement in the *'aggregate average quality of life'* associated with tuberculosis: from 'poor' in 1900 to 'some' in 1925 (this is shown in the final row of Table 7.2.1, where the EuroQol rank improves from 5 to 4).

7.2.3 Nineteen Fifty

By the middle of the twentieth century improvement in the health and welfare related quality of life for tuberculosis sufferers had made significant progress: Table 7.2.1 highlights that nearly every variable enjoyed an improved EuroQol rank between 1925 and 1950.

The most impressive improvements were related to *'medical developments'*. By 1950 streptomycin had been established and was widely available. However, this antibiotic did not provide a universal cure for tuberculosis (due to resistance problems), and it was not until 1975 that this aggregate *'health developments'* variable achieved 'complete' quality of life for tuberculosis sufferers. Nonetheless, by 1950 this *'cure'* sub-variable of *'health developments'* had improved from a level of 'no' quality of life to a level of 'some' and was rapidly on the way to 'complete' quality of life. This development drove the improvement in overall *'health developments'* from 'poor' in 1900 and 1925 to 'some' by 1950.

Results: Qualitative: Tuberculosis

As a result of the improved medical profile associated with tuberculosis, the pain, discomfort, anxiety, depression and adverse social circumstances triggered by tuberculosis had all improved by 1950 (shown in Table 7.2.1 as the improving EuroQol rank for the *'ability to lead'* a normal life variable and associated sub-variables). However, because streptomycin did not actually provide a universal cure and as a result of the newness of this treatment, the general stress and burden associated with tuberculosis welfare was still only at a level of 'some' quality for the *'ability to lead a normal life'*. The *'pain and discomfort'* associated with tuberculosis had improved to a level of 'fair' quality of life for tuberculosis sufferers (shown in Table 7.2.1 as EuroQol rank = 3).

By 1958 the work of charities and committees and various organisations had helped to highlight the conditions and additional needs associated with tuberculosis and to an extent provide these accordingly, and therefore by 1950 charities were providing 'good' quality of life. The government also improved their contributions to tuberculosis welfare during this period. The main contributions from the state were an improvement in medical access for tuberculosis sufferers and an improvement in welfare payments, which also improved the standards of living associated with tuberculosis. However, there still were numerous flaws in legislation and the state's approach to the problems of tuberculosis sufferers, such that *'government initiatives and help'* had only reached 'fair' levels of quality of life for tuberculosis sufferers.

As a direct result of government and charity aid and also a reduced severity of tuberculosis (as a result of medical advances), the *'financial burden'* of tuberculosis had declined and this variable was now associated with 'fair' levels of quality of life.

Therefore, by 1950 the most widespread range of twentieth century improvements had occurred for the health and welfare quality of life for tuberculosis sufferers. This can be largely attributed to the early developments in antibiotics that had begun to provide a cure and valuable improvements in the profile of tuberculosis. As a result of these far-reaching developments the overall or *'aggregate average quality of life'* associated with tuberculosis had reached 'fair' levels by 1950 (shown in the final row of Table 7.2.1 as EuroQol rank = 3).

7.2.4 Nineteen Seventy-Five

The most remarkable and important improvement in the tuberculosis story had been achieved by 1975. In the 1960s tuberculosis therapy became common place and changed the entire aetiology and pathogenesis of tuberculosis, such that this disease was no longer associated with death. Hence, by 1975 the *'cure'* for tuberculosis had achieved 'complete' quality of life levels.

This was not the case for the *'understanding'* of the disease and *'treatment'*, largely because there was still evidence of problems of notification and therapy and although the new antibiotics were able to prevent mortality they still had adverse side effects which detract from overall quality of life. However, these variables had improved in general and particularly in comparison to the levels they were at during earlier eras of the twentieth century. By 1975 *'understanding'* and *'treatment'* of tuberculosis had reached 'good' levels. Therefore, by 1975 overall levels of *'health development'* had reached 'good' levels of quality of life for tuberculosis sufferers.

These improvements in *'medical developments'* had a positive impact on the *'pain'* and *'anxiety'* associated with tuberculosis, which both had also improved to 'good' levels by 1975. Further improvements were evident in the *'financial burden'* and *'social difficulties'* related to tuberculosis, which had improved to 'complete' levels of quality of life. This meant that the overall *'ability to lead a normal life'*, despite having the tuberculosis disease had improved to 'complete' levels.

By 1975 there had been no genuine advances in *'government initiatives'* beyond 1950s levels. The state was providing the curative medical therapy which provided a significant contribution to improved quality of life, but this was evident in 1950 under the 1948 National Health Service Act. Therefore, the level of government initiatives remained at 'fair' quality of life for tuberculosis sufferers.

Finally, the work of charities and committees in raising *'recognition and awareness'*, which had been so fundamental in early quality of life improvements for tuberculosis sufferers, declined significantly in importance during this new era, when tuberculosis was curable. Charities and committees were still evident but their importance and popularity had declined

substantially and they were therefore unable to play as much of a role as they had in previous times. Consequently, the contribution for *'recognition and awareness'* worsened from 'good' levels in 1950 to 'fair' levels in 1975. However, it should be noted that the major reason for this is positive. I.e. recognition and awareness diminished because tuberculosis was no longer a subject of concern to the population. Although it could be argued that this decline was too exaggerated when considering the re-emergence of tuberculosis in the later years of the twentieth century.

Therefore, the combination of these developments meant that the *'aggregate average quality of life'* facing 1975 tuberculosis sufferers had reached 'good' levels.

7.2.5 Two Thousand

By the close of the twentieth century there had been no further improvements (upon the 1975 level) in health and welfare related quality of life for tuberculosis sufferers. This is not to say that the conditions did not improve slightly, as they did, but it was more a micro consolidation of earlier developments rather than any macro achievements.

Despite the void of any further EuroQol rank improvements in the last era of the twentieth century, those which had been evident by 1975 ought to receive credit for their substantial contribution to the quality of life associated with tuberculosis, this is most true for *'medical developments'* and least for *'government initiatives'*. The culmination of this progress meant that during the twentieth century the *'aggregate average quality of life'* associated with tuberculosis improved from 'poor' to 'good' levels and in doing so provided one of the most significant contributions to standards of living for the twentieth century English population.

These improvements in the aggregate average EuroQol have facilitated equally far reaching improvements in the QALY. Table 7.2.1 highlights that, the tuberculosis QALY improved from 0.3333 of a healthy life year (which is represented as 1) to 0.8333 of a healthy life year. Hence, in 1900 the collection of health and welfare standards of living meant that tuberculosis sufferers only enjoyed about 30 percent of a healthy life year. By the year 2000, improvements largely in health but also welfare enabled the standards of living associated with tuberculosis to improve to a level that represents about 80 percent of a full healthy life year. The numerical indices will be utilised below in the wider QALE methodology in order to estimate the value

of the above improvements in the quality of life associated with tuberculosis, which was extensive.

7.3 Cancer

At the beginning of the century cancer was an incurable, largely unidentifiable, poorly treated, crippling and completely life threatening illness. By the year 2000 cancer was more feared (as it was seen as the fundamental killer, for example, several population surveys have shown that cancer is perceived with more alarm and considered more seriously than any other disease) but was less disabling, unmanageable, painful and fatal[662]. This outcome, which is very valuable for the population was created by the developments in medical technology, government initiatives and the work and campaigning of charities. The twentieth century transition of these developments, documented by the EuroQol standardised spectrum, are shown in the table below.

[662] Fallowfield, "Breast Cancer", p. 4

Results: Qualitative: Cancer

Table 7.3.1: EuroQol results for breast and stomach cancer and EuroQol aggregate average conversion into QALY

Breast Cancer: EuroQol Variable	**Breast Cancer: EuroQol Score and Equivalent QALY**				
	1900	**1925**	**1950**	**1975**	**2000**
Government initiatives and help	6	6	4	3	3
Recognition/ awareness	6	4	4	3	2
Health developments	5	5	4	3	2
Pain and discomfort	5	5	4	3	2
Ability to lead a normal life	5	4	4	4	3
Aggregate Average EuroQol Score	5	5	4	3	2
Equivalent QALY	0.3333	0.3333	0.5000	0.6667	0.8333
Stomach Cancer: EuroQol Variable	**Stomach Cancer: EuroQol Score and Equivalent QALY**				
	1900	**1925**	**1950**	**1975**	**2000**
Government initiatives and help	6	6	4	3	3
Recognition/ awareness	6	4	4	3	2
Health developments	5	5	4	3	3
Pain and discomfort	5	5	4	3	3
Ability to lead a normal life	5	4	4	4	3
Aggregate Average EuroQol Score	5	5	4	3	3
Equivalent QALY	0.3333	0.3333	0.5000	0.6667	0.6667

Note: In Table 7.3.1 the aggregate average is calculated as the average score (to the nearest whole number) of the key variables: government initiatives and help, recognition/ awareness, health developments, pain and discomfort, ability to lead a normal life. Health developments and ability to lead a normal life variable scores are calculated as the average of the sub-variables in each category. The sub-variables for cancer are the same as those presented in Table 7.2.1 for tuberculosis, but are not presented here and the following text provides no crucial reference to these sub-variables.

7.3.1 Nineteen Hundred

In 1900 cancer sufferers were not a recognised or prominent group. There were no *'government initiatives'* to help prevent, treat or cure cancer (embodied by 'no' quality of life associated with *'government initiatives and help'* in Table 7.3.1: EuroQol rank = 6) and cancer charities had not been established (embodied by 'no' quality of life associated with *'recognition/ awareness'* in Table 7.3.1: EuroQol rank = 6). The state of medical technology was largely primitive as concepts of prevention, screening, comprehensive treatment (e.g. adjuvant therapy) and pain relief had not yet been coined in relation to cancer (embodied by

'poor' quality of life associated with *'health developments'* and *'pain and discomfort'* in Table 7.3.1: EuroQol rank = 5).

This meant that the *'aggregate average quality of life'* experienced by cancer sufferers in1900 was 'poor' (embodied by EuroQol = 5 for the *'aggregate average quality of life'* in the last row of Table 7.3.1). Standards of living had been slightly improved by limited *'health developments'* namely, surgery and x-rays. And, comparatively speaking (to the end of the century) notions of healthy life years and the hysteria associated with cancer had not become commonplace, which alleviated some of the perceived adversities of cancer. During the early 1900s, the English population was only just departing from an age of epidemics which entailed widespread mortality at relatively young ages and therefore the depression and anxiety which had been so closely linked to cancer by the 1970s was not yet evident.

7.3.2 Nineteen Twenty-Five

This overall quality of life situation for cancer sufferers had not really improved by 1925. Early developments were being made by cancer charities: the Cancer Research Fund had been established and was operational and the British Empire Cancer Campaign for Research (BECC) began to invest in detailed research to try and identify the causes of cancer[663]. Early developments were also being made in the treatment of cancer: the introduction of radium was laying the initial foundations for radiotherapy. These two developments provided small improvements in the quality of life associated with *'recognition/ awareness'* and the *'ability to lead a normal life'*. However the overall or *'aggregate average quality of life'* associated with cancer was still 'poor' in 1975.

7.3.3 Nineteen Twenty-Five to Nineteen Fifty

By the middle of the twentieth century there had been many valuable developments in the treatment of cancer and also provisions by the government to aid the battle against this disease. Many of these developments were the fruition of earlier inventions and also the initial benefit of new post war inventions and interventions. The 1939 Cancer Act provided an increase in *'government initiatives and help'* through its aim to ensure that every cancer patient received the medical attention they required, even though the provision of this Act was

[663] Porter, "The Cambridge Illustrated History of Medicine", p. 335

not implemented until the introduction of the NHS in 1948. The policies of the NHS provided an important boost to the quality of life faced by cancer sufferers as it meant that treatment for their illness became more accessible, which in turn improved their prognosis and the negative effects that cancer would have had upon quality of life. However, services and provisions were by no means ideal and treatment options were severely limited. Therefore government changed their position from 'no' help towards improving the cancer related quality of life to 'some' help (shown in Table 7.3.1 as an improved EuroQol rank, from 6 to 4).

The most important development for cancer in 1950 were the initial breakthroughs associated with medical technology, which improved the *'health developments'* and *'pain and discomfort'* related standards of living for cancer from 'poor' to 'some' quality of life.

Improvements in *'government initiatives and help'*, *'pain and discomfort'* and *'medical developments'* (and the stability of all the other variables in Table 7.3.1) fostered an improved *'aggregate average quality of life'* score for stomach and breast cancer: the quality of life improved from 'poor' to 'some' between 1925 and 1950.

7.3.4 Nineteen Fifty to Nineteen Seventy-Five

The period between 1950 and 1975 included some of the most important inventions and innovations of the twentieth century, from the perspective of care for cancer that could improve the quality of life of sufferers. During this time the *'government initiatives'* provided an important contribution to knowledge through their sponsorship of numerous working parties that catered for many aspects of cancer, from the causes to diagnosis, statistical recording and analysis of cancer prevalence to the potential benefits of screening. This awareness, about the potential benefits of screening was shared by cancer charities. The fusion meant that by 1963 a screening programme had been introduced for breast cancer, which was especially beneficial for quality of life, as it meant that mammography could identify cancer much earlier and consequently reduce many of the potential adversities of later stage cancer.

The invention of the CAT scan in 1972 provided one of the most valuable weapons in the therapeutic armamentarium against cancer. This equipment allowed a substantially heightened precision in scanning for cancer, thus improving the prognosis and conditions faced by cancer sufferers and ultimately the quality of life associated with cancer. However the availability of

CAT scans was severely limited, largely due to costs *"which were beyond the capacities of many district health funds and hospitals"*[664]. Nonetheless, this benefit facilitated developments in screening and improved this aspect of quality of life.

During this time some of the largest gains were experienced in adjuvant therapies. Chemotherapy experienced many improvements that were largely facilitated by the post World War Two pharmaceutical revolution. Radium therapy became much better understood and as a result much safer and more effective. It was during this time that hormone therapy was introduced and significantly enhanced the management of breast cancer. This meant that by 1975 adjuvant therapy for breast cancer had become increasingly systematic and was contributing to a reduction in recurrence rates and mortality from breast cancer[665]. This created a 'good' quality of life for *'medical developments'* in breast cancer treatment and 'fair' quality of life for stomach cancer.

The combination of these factors meant that by 1975 the overall or *'aggregate average quality of life'* associated with cancer had reached levels that were considered 'fair'.

7.3.5 Nineteen Seventy-Five to Two Thousand

During the final quarter of the twentieth century the foundations of the aforementioned developments were built upon. Surgical techniques became more advanced, which yielded more precision, accuracy and less disruption to an individual's *'ability to lead a normal life'*. Another development in surgery was provided by reconstructive surgery. This provided the biggest boost to breast cancer sufferers, who underwent mastectomies. Hence, it was possible to remove the cancer, even a large stage II type tumour, and not loose a breast as it was possible to recreate one artificially. Chemo, hormone and radio therapies also became more sophisticated and effective. The combination of these developments and screening meant that many cases of breast cancer were survivable by the end of the century. This was not the case for stomach cancer, which, despite these improvements, was still associated with a harrowing prognosis, even by the year 2000.

[664] Webster, "The National Health Service: A Political History", p. 114
[665] Richards et al, "Variations in the Management and Survival of Women Under Fifty Years with Breast Cancer in the South East Thames Region"

Another development which provided a glimmer of hope for eventual reduction in cancer rates and in an overly idealistic form, a potential cure was the increasing understanding of environmental factors and their ability to prevent the onset of cancer.

The combination of these developments in prevention, screening and treatment created an improvement in *'medical developments'* and *'pain and discomfort'* which had reached 'good' (breast cancer) and 'fair' (stomach cancer) quality of life levels by the year 2000. Table 7.3.1 highlights how this marks an improvement over the 1975 health related quality of life for breast cancer and that stomach cancer did not improve beyond its 1975 level. This emphasises the value of improvements in surgery and therapy for breast cancer.

During the last decade of the twentieth century the government provided valuable initiatives, which were aimed at reducing the number of cancer deaths through: improved screening (ensuring that all women between the ages of 50 and 64 received mammograms every three years, which continually achieved its targets since its introduction in 1988-1991[666]), trying to educate the public about the risks of cancer, and their efforts to eliminate social inequalities in cancer incidence. The government should be complimented with at least part of the improvements in the quality of life associated with cancer mortality and morbidity.

However, the improvements in government efforts and NHS cancer treatment during the last quarter of the twentieth century were not far reaching enough to improve *'government initiatives and help'* related quality of life above levels that were 'fair' for cancer sufferers. Hence, by the close of the twentieth century the government had failed to achieve ideal or even 'good' levels of quality of life for cancer patients. This is largely a result of decades of under-investment in the people and equipment involved in the fight against cancer. The NHS has too few cancer specialists of every type for example, at the close of the twentieth century, England had approximately eight oncologists per million population, which is less than half the number in other comparable European countries[667]. All of these factors had contributed to England lagging behind comparable economies (namely, America and European Union members) in cancer survival rates at the end of the twentieth century and, consequently, not

[666] The NHS breast cancer screening programme has continued to meet its target rates for uptake, recall, biopsy, benign biopsy, and detection of cancer. Quinn et al, "Changes in the Incidence of and Mortality from Breast Cancer in England and Wales since Introduction of Screening", p. 1394
[667] Government Papers, "The NHS Cancer Plan, A Plan for Investment, A Plan for Reform", p. 19

achieving any fundamental or genuine improvements in government related quality of life during the last quarter of the twentieth century.

During this time cancer charities enhanced their efforts and there was increased collaboration between the two largest cancer charities: the Imperial Cancer Research Fund and the Cancer Research Campaign, which helped the plight of cancer sufferers. This meant that charity support had achieved 'good' quality of life levels by the year 2000 for *'recognition and awareness'* associated with stomach and breast cancer.

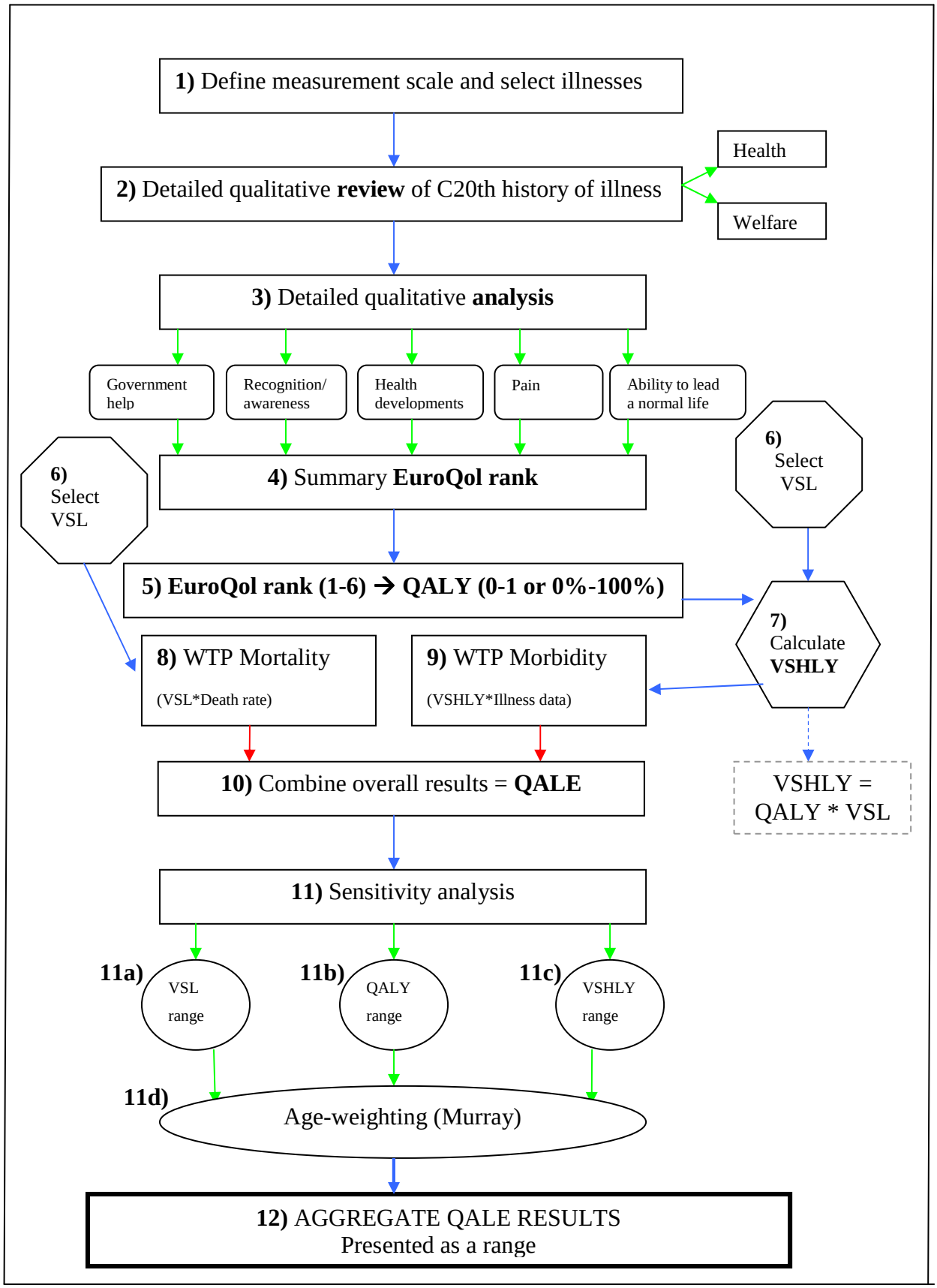
7.3.6 Two Thousand

By the close of the twentieth century the *'aggregate average quality of life'* associated with breast cancer had improved further to reach a level that could be considered to be 'good', and stomach cancer had improved to a lesser extent, to quality of life levels that were 'fair'. This should be considered as an important development from the 1900 level of 'poor', although less so for stomach cancer. The final row for the breast and stomach cancer sections in Table 7.3.1 shows the QALY equivalent of the aggregate EuroQol score, which evolved from 0.3333 in 1900 to 0.6777 in 2000 for stomach cancer and 0.8333 for breast cancer. Both of these QALY transitions, although especially breast cancer, represent important developments in the quality of life for cancer sufferers. The precise value of this gain will be estimated below.

8. Quantitative Findings

This Chapter will contain the quantitative analysis that is evolved from the previous qualitative results. The twentieth century improvements that have been identified for morbidity (defined by the QALY weights which were transformed from EuroQol, above) and the improvements in life expectancy (defined by the death rate) will be considered here, in conjunction with other crucial facets of the thesis' quantitative, extended willingness to pay or QALE methodology. The theoretical details of the thesis' QALE methodology are provided in Chapter 3. Before this methodology is applied it is desirable to reiterate the key features and mechanics of this methodology, which will be achieved here, with the aid of the flow chart summary of the thesis' methodological process. This will also identify the location of all the components of the QALE methodology (in earlier chapters of the thesis) and highlight the interaction of these features in this quantitative section.

Figure 8.1: Flow chart summary of the thesis' methodological process



Results: Quantitative

Figure 8.1 represents the summary of the thesis methodological processes that were necessary to facilitate the quantitative results contained below. These processes are summarised here (in accordance with the numbering in Figure 8.1).

1) Define measurement scale and select illnesses: A detailed outline and justification of the thesis methodology and illnesses applied to this QALE calculation is provided in Chapter 3. To summarise: the methodology used here is an original adaptation of willingness to pay, where quality of life improvements in mortality and morbidity are valued, which forms an extended willingness to pay or quality adjusted life expectancy, QALE. The morbid states that are utilised in this QALE methodology are: blindness, tuberculosis, and breast and stomach cancer. A key contribution of this measurement is the evaluation (and later quantification) of the quality of life associated with morbidity over the twentieth century.

2) Detailed qualitative review of C20th history of illness: This provides an introduction to the thesis (and is subsequently presented in Part I of the thesis). This provides a valuable foundation for the more detailed and specific considerations and claims of the thesis. This exercise is also valuable because it provides a practical justification for the selection of the thesis illnesses.

3) Detailed qualitative analysis: The literature is currently void of any historical (pre 1970) QALY estimates for illnesses and therefore the thesis had to make an informed best estimate from reviewing major literature sources for the thesis morbid states (namely, breast and stomach cancer, tuberculosis and blindness). This warranted a comprehensive account of the conditions associated with the thesis morbid states for different eras of the twentieth century, from a health and welfare perspective for quality of life. This was formulated around the thesis' spectrum of key standardised variables: government help, recognition and awareness, health developments, pain and discomfort, ability to lead a normal life (for an elaboration see Chapter 3: Table 3.5 to 3.7). These considerations represent the main body of the thesis and are presented in Part II.

4) Summary EuroQol rank: In addition to a detailed and standardised qualitative analysis about the health and welfare related quality of life of the thesis' morbid states, the thesis has also gauged these on a standardised spectrum of development, namely EuroQol. This was necessary so that the qualitative analysis (summarised by EuroQol) could be evolved

into quantitative indices (summarised by the QALY). EuroQol is explained in more detail in Chapter 3 and applied in Chapter 7.

5) EuroQol rank (1-6) → QALY (0-1 or 0%-100%): This stage is crucial for the quantitative thesis methodology because it provides the conversion from qualitative to quantitative. In doing this, it also provides one of the thesis' key contributions to knowledge. Hence, the QALY is crucial for making quantitative considerations about health improvements. The QALY is derived by applying the EuroQol standardised spectrum of development to all illnesses in all eras considered in the thesis. This has facilitated a quantitative index (QALY) from the detailed qualitative analysis in the thesis (summarised by EuroQol). The summary EuroQol and subsequent QALY results are presented in Chapter 7: Table 7.1.1, 7.2.1, 7.3.1 and the QALY results are summarised in the table below.

Table 8.1: QALY estimates (derived from EuroQol analysis) for thesis illnesses and eras[668]

Year	**QALY (as a proportion of one healthy life year)**			
	Blind	**Breast cancer**	**Stomach cancer**	**Tuberculosis**
1900	0.3333	0.3333	0.3333	0.3333
1925	0.5000	0.3333	0.3333	0.5000
1950	0.6667	0.5000	0.5000	0.6667
1975	0.6667	0.6667	0.6667	0.8333
2000	0.6667	0.8333	0.6667	0.8333
1900-1925	0.4167	0.3333	0.3333	0.4167
1925-1950	0.5834	0.4167	0.4167	0.5834
1950-1975	0.6667	0.5834	0.5834	0.7485
1975-2000	0.6667	0.7485	0.6667	0.8333
1900-2000	0.5000	0.5833	0.5000	0.5833

6) Select VSL: The VSL represents the valuation for the decline in mortality. It was therefore necessary to identify the most plausible VSL so that the twentieth century declined in mortality could be most accurately valued. This was achieved through adopting the VSL multiples that were presented by Miller's study. The table below provides the VSL values utilised in the thesis, which uses the VSL multiple approach (VSL multiple *

[668] See Appendix 12.6.1 and 12.6.2 for alternative QALY values and calculations.

GDP per capita at mid point) in order to yield the VSL. The rationale and selection of Miller's best VSL estimate are outline in Chapter 3.

Table 8.2: Calculation and results of VSL values utilised in the thesis, derived from Miller's VSL multiple estimates (1990 international $)[669]

Period	**VSL Multiple**	**GDP per capita (GDP pc) at period mid-point**		**VSL (Millions)**
		Mid-point	**GDP pc**	
1900-1925	128	1913	5032	0.64
1925-1950	128	1938	5983	0.77
1950-1975	128	1963	9070	1.16
1975-2000	128	1988	15988	2.05
1900-2000	128	1950	6907	0.88

7) Calculate VSHLY (QALY * VSL = VSHLY): The VSL was also necessary in the thesis methodology as it forms part of the VSHLY measure. Hence, the VSHLY represents the valuation for the decline in morbidity and is a function of the VSL and the QALY (for the selected illnesses and eras). The table below provides the VSHLY values utilised in the thesis, which uses the VSL identified in Table 8.2 combined with the QALY values identified in Table 8.1 (average QALY of the era under consideration, e.g. for breast cancer 1900-2000, the 1900 QALY = 0.3333 and 2000 QALY = 0.8333 and therefore, the average QALY for 1900-2000 = [0.3333 + 0.8333)] / 2 = 0.5833) for the associated illness and era (VSL * QALY = VSHLY) in order to yield the VSHLY. The explanation and justification for the VSHLY is outlined in Chapter 3.

[669] Miller, "Variations between Countries in Values of Statistical Life", p. 180. See Appendix 12.1.1 for alternative 'High' and 'Low' VSL estimates used in the sensitivity analysis.

Table 8.3: Calculation (VSL * QALY) and results of VSHLY values utilised in the thesis: for thesis morbid states (1990 international $)[670]

Period	**VSL (Millions)**	**QALY**				**VSHLY (Millions)**			
		Blind	**Breast cancer**	**Stomach cancer**	**Tuberculosis**	**Blind**	**Breast cancer**	**Stomach cancer**	**Tuberculosis**
1900-1925	0.64	0.4167	0.3333	0.3333	0.4167	0.27	0.21	0.21	0.27
1925-1950	0.77	0.5834	0.4167	0.4167	0.5834	0.45	0.32	0.32	0.45
1950-1975	1.16	0.6667	0.5834	0.5834	0.7485	0.77	0.68	0.68	0.87
1975-2000	2.05	0.6667	0.7485	0.6667	0.8333	1.36	1.53	1.36	1.71
1900-2000	0.88	0.5000	0.5833	0.5000	0.5833	0.44	0.51	0.44	0.51

8) WTP Mortality and **9)** WTP Morbidity: Once all of the QALE methodology variables (QALY, VSL, VSHLY, death rates and illness data [represented by the QALY and the prevalence data]) have been identified and valued it is possible to combine these in the thesis methodology. This QALE methodology is outlined in Equation 8.1 below.

[670] See Appendix 12.1.1 (Diseases) and 12.1.2 (Disability) for alternative 'High' and 'Low' VSHLY estimates used in the sensitivity analysis

Results: Quantitative

Equation 8.1: Summary of the thesis methodology: quality adjusted life expectancy (QALE)

Willingness to Pay Morbidity (MB):

WTP_{MB} Considers increased quality of life with an illness or disability →

WTP_{MB} = VSHLY * population weighted fall in the burden of disease/disability (or the change in the morbidity burden) = morbidity gain

For WTP_{MB} this would have to be calculated for each type of illness and disability and their associated QALY (λ)

This will then be combined with the equivalent information for mortality improvements

Willingness to Pay Mortality (MT):

WTP_{MT} Considers increased life expectancy →

WTP_{MT} = VSL * population weighted fall in the death rate (or the change in the mortality burden) = mortality gain

Quality Adjusted Life Expectancy (QALE):

Such that WTP_{MB} + WTP_{MT} OR morbidity gain + mortality gain = $QALE$ improvement:

$$\frac{dc^*}{d\mu+\lambda} = \frac{-u(c^*)}{(\rho+[\mu+\lambda])}$$

$u(c^*)$ = the goods value of life,

c^* = consumption,

ρ = the pure rate of individual time preference,

μ = set of mortality rates,

$\lambda = (\sum \Pr[\text{ Condition D at t + k}] * [\text{QALY for D at t + k}])$

10) Combined overall results = QALE: Hence, the QALE is a function of the following: i) Morbidity Gain (QALY * prevalence yields the change in the morbidity burden. The change in the morbidity burden * VSHLY = morbidity gain). This is simultaneously valued with ii) Mortality Gain (change in the mortality burden [embodied in the death rate]*VSL = mortality gain).

Results: Quantitative

This will provide results about the (monetary) value of improvements in 'quality adjusted life expectancy' (QALE), which is the combined measure of the mortality and morbidity gains and forms the basis of the thesis' extended willingness to pay methodology. These results will be presented in absolute (monetary form) and also as an additional proportion (percentage) to GDP growth for the era under consideration.

11) Sensitivity analysis: This QALE result will be applied to a series of sensitivity analyses in order to provide a range of results and escape from accusations of bias. This analysis has been designed to consider the most contentious variables in the thesis' QALE methodology, namely:

> **11a)** VSL range, **11b)** QALY range and **11c)** VSHLY range: The sources of contention for these variables are outlined in Chapter 3. The thesis will overcome these as far as possible through considering a 'High' and 'Low' value estimates as well as the existing 'Mid' estimate for these three variables (VSL, QALY and VSHLY).

These calculations will require the QALE methodology to be reformulated (with alternative QALY, VSL and VSHLY values) and this will entail recalculating the QALE in the same process as was explained above (from stage 5 to stage 10).

> **11d)** Considerations about age weighting: The appeal and approach of this sensitivity analysis are outlined in Chapter 3. Essentially, Murray's weights for different ages will be applied to the QALE results in order to provide estimates that adhere to claims of different QALE gain values for different ages.

12) Aggregate QALE results (presented as a range): Due to the subjective and often controversial nature of illness and welfare measurement, the estimates of the burden of disease will be provided as a range of possible values and will be presented in a tentative fashion, whereby the reader will be invited to consider the results as a function of what they are willing to believe, rather than stating the results as a definitive estimate. I.e. this approach will provide results that are dependent upon whether or not the reader will accept a given scenario. Chapter 8.3 will provide a range of estimates, based on the sensitivity analysis for the results in Chapters 8.1 and 8.2, for the entire twentieth century (1900-2000).

The quantitative analysis (which has been explained above: through the flow chart worked example) will be applied to 'Disability' (i.e. blindness in Chapter 8.2) and 'Disease' (i.e.

cancer and tuberculosis in Chapter 8.1) separately (i.e. in separate chapters) due to the subtle but profound differences in the aetiology, prognosis, burden and ultimate measurement of illness linked with disease and disability.

The most noteworthy difference between disease and disability is their relationship with death, whereby disease is more closely linked with death. Even though cases of disease are not necessarily resolved in death, as increasingly in the twentieth century they increasingly resulted in a cure, disability is not usually a direct cause of death. Following on from this observation is the generally more confined nature of disease relative to the more ambiguous prognosis for disabilities. For example, tuberculosis and cancer are associated with a survival rate throughout the twentieth century, usually five years from the date of diagnosis in the context of the thesis, which essentially provides a time period for the burden of illness, which is either resolved in survival/cure or death. There is no equivalent notion for disability, where the average duration of disability, in the context of the thesis, varied from 26 to 4 years[671].

Because of the different nature of disease and disability, the key quality of life considerations are also different. Hence, EuroQol (the thesis' foundational measuring tool) is considering slightly different facets. For example, disability is much more concerned with rights and status experienced by sufferers whereas quality of life associated with infectious disease is more affected by antibiotic therapy. These distinctions and the key variables for measurement associated with disease and disability are explained in Table 3.5, 3.6 and 3.7 in Chapter 3: Methodology. The result of this is that there is a slight variation in the QALY for disability and disease because the base measure (EuroQol) is measuring slightly different features.

Although the burden of illness for disability and disease are very similar and both equally crucial for the thesis' consideration of twentieth century quality of life it is necessary to consider them separately at a first approximation. Once this has been achieved it will be possible to combine these results, in Chapter 8.3 and Chapter 9. This is possible because Chapter 8.3 is a summary and Chapter 9 makes more broad and generalised considerations, which are based on conservative estimates from the major sources of illness burden (analysed in detail in the thesis) in order to extrapolate forward from the thesis illness

[671] See Appendix 12.13.1 for an elaboration

results and provide a lower bound estimate about the value of improved quality of life associated with illness for all morbidity and mortality in twentieth century England.

Chapter 8.1 will contain the more detailed quantitative disease (tuberculosis and cancer) analysis. Chapter 8.2 will make the same quantitative considerations for the thesis' sample disability, blindness. Chapter 8.3 will provide an aggregate (disease and disability) quantitative summary and range or results about the twentieth century value of QALE gains for the diseases and disability considered in the thesis. This will enable the reader to identify their most preferred QALE gain result from the extensive range (i.e. this represents stage 12 in the flow chart example). These results from Chapter 8 will then be used as the foundation for the Extended Results calculations in Chapter 9.

8.1 Disease (Cancer and Tuberculosis)

The previous chapters of the thesis have highlighted the considerable and extensive improvement in health experienced by the population in twentieth century England. These developments have provided important contributions to the quality of life of the population and to wider, more Fisherian notions of economic development[672]. This chapter will provide the quantitative estimates of the value of these health improvements, through applying the QALE methodology to the thesis diseases: breast and stomach cancer and tuberculosis.

Through applying the QALE methodology to the mortality and morbidity data and providing a value for the mortality and morbidity gain this chapter will provide a first approximation about the (monetary) value of increased life expectancy and improved quality of life associated with health. After the methodology has been applied to the data and the QALE gain identified, it will be possible to consider a broader range of QALE gain results through recalculating the QALE with a range of different weights for the most tenuous variables within the QALE methodology: the VSL, VSHLY and QALY. This sensitivity analysis represents an important part of the thesis' quantitative results as this exercise will generate 27 estimates (instead of one) for each disease and era and in aggregate, 405 estimates (instead of 15)[673].

These sensitivity analyses will be further complemented through applying all of the above ranges of results to an age-weighting function. This will yield results about the range of possible (monetary) values of improved health, from an un-weighted and age-weighted perspective. The appeal of such considerations has been outlined in Chapter 3.

[672] Fisherian growth is a notion that was coined by Nordhaus (1999) and is defined as the maximum amount that a nation can consume while ensuring that members of all future generations can have life time utility that is at least as high as that of current generations. When this yardstick is utilised, life expectancy and in the context of the thesis, quality adjusted life expectancy, are included in the production function and the value of improvements in mortality can be accounted, in terms of consumption."

[673] Calculation: 27 results for three illnesses and five eras = (27*3)*5 = 405 versus one result for three illnesses for five years = (1*3)*5 = 15.

It will then be possible to add further significance to these results through considering them in conjunction with the growth of national income (represented as Gross Domestic Product [GDP hereafter] or most frequently, GDP per capita [GDP pc] in the thesis) for different eras during the twentieth century. This will add significance to the previously identified QALE gain for disease through standardising the value of improved health. This exercise will authenticate Fisherian type claims about the desirability of including health in some form of extended national income measure, because the contribution of improved health to economic growth, identified here for twentieth century England, has been considerable compared to GDP per capita only (as shown by Table 8.1.15).

8.1.1 Primary Valuation of Improvements in Morbidity

The first stage in valuing morbidity improvements is the calculations of the QALY. Chapter 7 of the thesis provides the qualitative summary (of Part II) and also the first stage in evolving these qualitative interpretations into quantitative indices, through using EuroQol to summarise these developments. Once the EuroQol rank had been identified, it was converted into a QALY (this represents stage 5 in the previous flow chart example). The results of this conversion for the thesis diseases are shown above, in Table 8.1, where the QALY is reported for the thesis diseases for different eras of the twentieth century.

The bounds of the QALY are 0.1 and 1, whereby 1 is a full healthy life year and zero approximates death. Therefore the closer the QALY is to 1, the better the quality of life illness sufferers were experiencing. In Table 8.1 tuberculosis reached a level very close to 1 or 100 percent of a healthy life year (0.8333 or 83 percent by 1975) and stomach cancer remained at a much less favourable standard, closer to 50 percent of a healthy life year (0.6667 or 67 percent in 2000). Breast cancer, like tuberculosis, also experienced important improvements in quality of life (0.8333 or 83 percent by 2000). These improvements have occurred for numerous reasons, which have been analysed in previous qualitative chapters of the thesis[674].

When this QALY is utilised in the wider thesis QALE methodology (i.e. stages 8, 9 and 10 in the flow chart example above) it will be computed inversely. For example, breast cancer in 1900 achieved a QALY of 0.3333 (which has been evolved from a EuroQol rank of 5 = ‘poor quality of life’ (see Tables 7.3.1 and 8.1 above) in the QALE methodology the QALY will be computed as 1 - QALY = 1 - 0.3333 = 0.6667. This is because of the

[674] See Part II and Chapter 7 of the thesis

difference in approach of measurement between the EuroQol QALY and the subsequent QALY in the QALE methodology. Although these are measuring the same features, they are considering these features from different angles: in the thesis EuroQol (and the subsequent thesis QALY) is measuring the fraction of a healthy life year that is achieved and the QALE methodology is considering the QALY as an adjustment for the fraction of a healthy life year that is lost. Therefore, to standardise the thesis QALY in the QALE calculation it is necessary to calculate 1-(EuroQol) QALY.

In the next stage of the thesis QALE methodology these QALY results can be combined with prevalence for the associated disease and era, in order to calculate the morbidity burden, which comprises the number of disease sufferers (prevalence) multiplied by the burden (i.e. 1-QALY in the context of the QALE methodology considering the amount of life year lost [rather than the initial EuroQol QALY considering how much of a life year gained]) of this disease (QALY).

In an attempt to generate the most precise estimates of the morbidity burden (= prevalence*QALY) there are two additional specification for the morbidity burden calculation: i) the burden and prevalence are calculated by age group in order to provide more detailed estimates[675], and ii) these age specific morbidity burden estimates are standardised for the age distribution across the population structure at the start and end point of the era under consideration. This process yields two sets of results for any era (the change in the morbidity rate [or morbidity burden by age relative to the population by age] fixed to either the start [T1] point or end [T2] point population), which are then averaged in order to present the most indicative estimate about the change in the burden of disease, while accounting for the changing age structure of the population between the start and end point in any era. This represents a standard weighing/index number process and using the mid-point or average represents an orthodox approach.

The morbidity burden results differ for the start and end point populations because of the distribution of morbidity across ages. E.g. in the thesis breast and stomach cancer represent diseases that are most prevalent at the oldest ages: if there is a significant increase in the old age population (as there was over the twentieth century), then the rate of this morbidity burden will be less at the end point (2000) than at the start (1900) because the rate represents the number of morbidity incidents per population size. Therefore, to try and

[675] The data is divided into the following age groups: 0-4, 5-9, 10-14, 15-19, 20-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75+

standardise these external influences the thesis will consider the rate for the both the start and end point population structures and use the average of these values as the final morbidity burden estimate. This process is desirable as it provides additional necessary detail about the morbidity environment, standardised to the era being considered.

This process of calculation will be applied to the thesis diseases, but not the disability as there is not (age-specific prevalence) data of enough detail to perform such calculations.

After the morbidity burden has been identified for different eras it will be possible to identify the change in the morbidity burden between these eras. This change in the morbidity burden will then be valued (using the VSHLY) in order to estimate the morbidity gain, which is essentially the value of a decrease in the morbidity burden over time. With reference to the flow chart example at the start of Chapter 8, identifying the value of the morbidity gain (change in the morbidity burden * VSHLY) represents stage 9.

Hence, in order to estimate the morbidity gain, the change in the morbidity burden needs to be valued. This is achieved through establishing the VSHLY and applying it to the change in the morbidity burden. The VSHLY is equated through combining the VSL and the QALY for the illness and period being considered (VSL * QALY = VSHLY). Calculating the VSHLY represents stage 7 in the above flow chart (Figure 8.1) explanation and Table 8.3 provides the calculations and result of the VSHLY.

Because of the level of detail in the base morbidity burden calculations (as a result of the age standardised population considerations explained above) only the final results (about the change in the morbidity burden) will be presented here. However, this will be supplemented by a detailed worked example of this meticulous methodological process utilised in the thesis in order to estimate the most accurate morbidity gain (see Table 8.1.2.i and 8.1.2.ii). The detailed calculations are shown in Appendix 12.3. (In reference to the flow chart analysis above, this worked example provides a more detailed explanation of stage 9). Once the morbidity burden changes have been identified they will be applied with the VSHLY to determine the morbidity gain. This stage of the calculation will be shown below (in Tables 8.1.3, 8.1.4 and 8.1.5), after the worked example indicating the calculation of the morbidity burden change. This worked example will consider breast cancer for 1900-2000.

Results: Quantitative: Disease

The morbidity burden change for breast cancer between 1900 and 2000 is established through considering the prevalence of breast cancer multiplied by the QALY for breast cancer at the start and end point of the period: 1900 and 2000. In an effort to yield the most accurate results, this calculation will be done on an age standardised population basis. Table 8.1.1 shows the non-age standardised, or aggregate prevalence and QALY information. For example, the aggregate morbidity burden for breast cancer in 1900 and 2000: 15,980 cases of breast cancer in 1900 with a (1-QALY) of 0.6667 (in simplistic aggregate terms, this translates into a morbidity burden of 10,654 cases {15,980 * [1 - 0.3333=] 0.6667} – the inverse of the QALY for calculation purposes[676] = 10,654) versus the 2000 burden: 87,915 cases of breast cancer with a (1-QALY) of 0.1667 which translates into a morbidity burden of 14,655 cases (87,915 * [1-0.8333 =] 0.1667 = 14,655). These aggregate key components for the thesis disease morbidity burdens (aggregate prevalence and QALY) are shown in the table below.

Table 8.1.1: Calculation of morbidity burden of thesis diseases: breast cancer, stomach cancer and tuberculosis[677]

Year	**Breast Cancer**		**Stomach Cancer**		**Tuberculosis**	
	Prevalence	**(1-QALY)**	**Prevalence**	**(1-QALY)**	**Prevalence**	**(1-QALY)**
1900	15980	0.6667	7989	0.6667	71959	0.6667
1925	27055	0.6667	10512	0.6667	57605	0.5000
1950	61477	0.5000	17300	0.5000	42024	0.3333
1975	68935	0.3333	14097	0.3333	7119	0.1667
2000	87915	0.1667	7704	0.3333	6031	0.1667

Table 8.1.1 highlights a subtle and important trend. For modern type illnesses, i.e. breast and stomach cancer, a major improvement in the morbidity burden (shown below in Tables 8.1.3 and 8.1.4) is facilitated by an improved QALY as the prevalence generally increases. This trend is most pronounced for breast cancer (and not consistently evident for stomach cancer), due to both a significant increase in prevalence and an improvement in the QALY (especially after 1950). This trend is crucial because it illustrates one of the key claims of the thesis: even though the prevalence of disease and disability increased as the twentieth

[676] Considering the inverse QALY is necessary when calculating prevalence, because EuroQol in the thesis (and the subsequent thesis QALY) is measuring the fraction of a healthy life year that is achieved and the QALE methodology is considering the QALY as an adjustment for the fraction of a healthy life year that is lost. Therefore, to standardise the thesis QALY in the QALE calculation it is necessary to calculate 1-(EuroQol) QALY. I.e. EuroQol and the subsequent thesis QALY consider how much of a life year, the QALY in the QALE methodology considers the burden or how much of an unhealthy life year → 1-(EuroQol) QALY = QALY in QALE methodology.

[677] Table 8.1.1 considers the burden of morbidity for different eras of the twentieth century, which is calculated by considering the prevalence multiplied by the inverse QALY. The more detailed and age-specific calculations of the morbidity burden see Appendix 12.2.

century unfolded the quality of life associated with these diseases and disabilities improved. Although many have speculated whether this phenomenon is genuine, the results contained in this thesis provide a more substantiated indication about the accuracy of such speculation.

Table 8.1.1 also shows a series of trends which all indicate an improvement in the burden of disease as the twentieth century unfolded. Tuberculosis displays the most straightforward example of this claim, where the prevalence fell consistently and markedly during the twentieth century and the QALY experienced equally impressive improvements, such that the morbidity burden of tuberculosis was minor by 2000 (see Table 8.1.5). Breast and stomach cancer show more mixed and seemingly contradictory (stomach cancer) results. For stomach cancer there was an improvement in the morbidity burden, which was largely a result of a fall in prevalence as the QALY associated with stomach cancer did not improve substantially over time. Conversely, breast cancer shows a large increase in prevalence which was compensated by far reaching improvements in the QALY, to the extent that the morbidity burden began to decline from 1950 onwards, to such a degree that the year 2000 morbidity burden (prevalence*QALY) had returned to a level similar to the year 1900 morbidity burden.

The next stage of the thesis methodology is to consider this burden as a rate of the population for which it relates. Table 8.1.2.i considers the morbidity rate or age specific morbidity index (shown as MBR/1000) which is calculated through dividing the morbidity burden number by the population number for the corresponding age group. Also shown in Table 8.1.2.i is the percentage of population by age group for 1900 (T1) and 2000 (T2).

Results: Quantitative: Disease

8.1.2.i: Morbidity burden rate (morbidity/population) by age: breast cancer: 1900 and 2000[678]

1900: Breast Cancer			**2000: Breast Cancer**		
Age	**% Population**	**MBR/1000**	**Age**	**% Population**	**MBR/1000**
0-4	0.114068	0	0-4	0.0606449	0
5-9	0.107086	0	5-9	0.0647742	0
10-14	0.102585	0	10-14	0.0654486	0
15-19	0.099601	0.0009236	15-19	0.061065	0
20-24	0.095741	0.0032028	20-24	0.0587045	0.001969409
25-34	0.161673	0.036605	25-34	0.1456456	0.024475432
35-44	0.123151	0.3884269	35-44	0.1471602	0.105797936
45-54	0.089448	0.9862535	45-54	0.1322942	0.290137932
55-64	0.059846	1.4674386	55-64	0.1048284	0.470378472
65-74	0.033199	2.2693267	65-74	0.0559221	0.682198245
75+	0.013602	1.5554554	75+	0.0753201	1.57410013

Once the calculations in Table 8.1.2.i have been made, the morbidity burden – standardised to the age structure of the population – has been identified for the start (TI) and end (T2) point of the period. Also the distribution of the population has been identified for T1 and T2.

The next stage in the QALE calculation (for morbidity) is to calculate the difference (or change) in the morbidity burden (MBR/1000) between T1 and T2. This is derived by identifying the weighted morbidity rates (WTD MBR) for T1 and T2, which is achieved by multiplying the relevant MBR/1000 and % population. The process and result of this calculation is shown below in Table 8.1.2.ii, where the difference in MBR/1000 (fixed to the population at the start and end point) is calculated to estimate the change in the morbidity burden (standardised for the population). The final stage of this calculation is to convert the result from a rate into a number, shown in Table 8.1.2.ii as 'Morbidity Burden'. This conversion was achieved through multiplying the 'Decrease/1000' (of the WTD MBR) by the relevant population number. The T1 and T2 'Morbidity Burden' numbers are averaged in the next stage of the thesis methodology (see Table 8.1.3, 8.1.4, 8.1.5). The Table below makes the 1900-2000 breast cancer morbidity burden change calculations fixed to the start point (T1) population and then the end point (T2) population.

[678] Extract from Appendix 12.3 and 13.1

Table 8.1.2.ii: Morbidity burden rate (morbidity/population) and morbidity burden change calculation by age: breast cancer: 1900-2000[679]

1900-2000: Fixed to [T1]								
Age	**% Pop**	**MBR/1000**	**WTD MBR**	**% Pop**	**MBR/1000**	**WTD MBR**	**Decrease/1000**	**Morbidity Burden**
	1900	**1900**	**1900&1900**	**1900**	**2000**	**1900&2000**		
0-4	0.114068	0	0	0.114068	0	0	0	0
5-9	0.107086	0	0	0.107086	0	0	0	0
10-14	0.102585	0	0	0.102585	0	0	0	0
15-19	0.099601	0.0009236	9.199E-05	0.099601	0	0	9.19904E-05	3.887021995
20-24	0.095741	0.0032028	0.00030663	0.095741	0.00196941	0.0001886	0.000118082	4.989531439
25-34	0.161673	0.036605	0.00591805	0.161673	0.02447543	0.003957	0.001961029	82.86260848
35-44	0.123151	0.3884269	0.04783501	0.123151	0.10579794	0.0130291	0.034805926	1470.712206
45-54	0.089448	0.9862535	0.08821879	0.089448	0.29013793	0.0259524	0.062266418	2631.045684
55-64	0.059846	1.4674386	0.08782016	0.059846	0.47037847	0.0281502	0.059669949	2521.332801
65-74	0.033199	2.2693267	0.07534013	0.033199	0.68219825	0.0226485	0.052691607	2226.465416
75+	0.013602	1.5554554	0.02115779	0.013602	1.57410013	0.0214114	-0.000253612	-10.71628367
							0.211351389	**8931**
1900-2000: Fixed to [T2]								
Age	**% Pop**	**MBR/1000**	**WTD MBR**	**% Pop**	**MBR/1000**	**WTD MBR**	**Decrease/1000**	**Morbidity Burden**
	2000	**1900**	**2000&1900**	**2000**	**2000**	**2000&2000**		
0-4	0.060645	0	0	0.060645	0	0	0	0
5-9	0.064774	0	0	0.064774	0	0	0	0
10-14	0.065449	0	0	0.065449	0	0	0	0
15-19	0.061065	0.0009236	5.6399E-05	0.061065	0	0	5.63989E-05	2.383115096
20-24	0.058705	0.0032028	0.00018802	0.058705	0.00196941	0.0001156	7.24037E-05	3.059393999
25-34	0.145646	0.036605	0.00533136	0.145646	0.02447543	0.0035647	0.001766622	74.64800639
35-44	0.14716	0.3884269	0.05716096	0.14716	0.10579794	0.0155692	0.041591718	1757.443481
45-54	0.132294	0.9862535	0.13047564	0.132294	0.29013793	0.0383836	0.092092068	3891.318088
55-64	0.104828	1.4674386	0.15382923	0.104828	0.47037847	0.049309	0.104520214	4416.465061
65-74	0.055922	2.2693267	0.1269055	0.055922	0.68219825	0.03815	0.088755543	3750.334394
75+	0.07532	1.5554554	0.11715698	0.07532	1.57410013	0.1185613	-0.001404325	-59.33925127
							0.327450642	**13836**

[679] Extract from Appendix 12.3 and 13.1

Hence, between 1900 and 2000 the morbidity burden of breast cancer decreased by 8931 (fixed to start point [T1]) – 13,836 (fixed to end point [T2]). This represents the change in the morbidity burden. Hence, it is this change in the morbidity burden (prevalence*QALY, for the age standardised population) of breast cancer shown in Tables 8.1.2.i and 8.1.2.ii (and all other morbid states in the thesis) that is then valued between different eras (this can be done between any two eras) to yield the morbidity gain.

As has been explained above, the next stage in the thesis QALE methodology is to value the change in the morbidity burden in order to estimate the morbidity gain. In order to identify the morbidity gain (which is the value of the change in the morbidity burden), it is necessary to identify the value of a statistically healthy life year (VSHLY) and apply this to the morbidity burden change. The calculation of the VSHLY has been outlined above, as stage 7 in the flow chart example, and in Table 8.3.

Continuing with the 1900-2000 breast cancer example, the VSHLY is identified by determining the VSL multiplied by the average QALY for the period. The VSL is determined through identifying the most reasonable approximate of a VSL multiple value and the GDP per capita at the mid-point of the period being considered[680]. These two numbers are then multiplied together to provide the VSL. Therefore (as in Table 8.2), the VSL multiple = 128 and GDP per capita in 1950 (mid-point between 1900 and 2000) = 6,907 (1990 international $) and multiplied together to yield the VSL = 0.88 (millions of 1990 international $). The VSHLY is the sum of the VSL (0.88 million) multiplied by the QALY for 1900-2000, which is 0.5833 (shown in Table 8.3). Hence, the VSHLY for 1900-2000 breast cancer is 0.88*0.5833 = 0.51 (millions of 1990 international $).

The morbidity gain can now be calculated through combining the change in morbidity burdens (shown above in bold in Table 8.1.2.ii) with the VSHLY ([8931 * 0.51 + 13,836 * 0.51]/2), which yields an average morbidity gain of 5,806 (millions of 1990 international $). This represents the value of an improved breast cancer burden between 1900 and 2000. This process and result is shown in the final row of Table 8.1.4, for breast cancer. Calculation of the morbidity gain is shown for all illnesses and eras in the thesis in Tables 8.1.3, 8.1.4, and 8.1.5, below.

[680] See Chapter 3 for a more detailed explanation about the VSL utilised in the thesis.

Results: Quantitative: Disease

Once the morbidity gain has been identified it can be combined with the mortality gain in order to calculate the QALE gain. This process and the evaluation of the mortality gain will be explained below.

First, the above example (for breast cancer 1900-2000) will be animated through providing the results about the morbidity gain for the thesis illnesses. As has been outlined above, this is shown in a summarised format (from the base results in appendices 12.3, 12.4, and 13.1) because of the intricacy of the base calculations: the thesis diseases have been considered by age, i.e. the morbidity burden rate has been considered for each age group (0-4, 5-9, 10-14, 15-19, 20-24, 25-34, 35-44, 45-54, 65-74, 75-84, 85+) and the subsequent percentage of the population that this age group represented. It was necessary to weight these calculations to age profiles at the start and end point (T1 and T2, respectively) of the populations being considered. This process yields two sets of results (T1 and T2) for the change in the morbidity burden and the subsequent morbidity gain. The reason for the difference in these results is the different distribution of the population across age groups coupled with the different decline in the morbidity burden for different age groups over time. E.g. in 1900 there was a greater proportion of the population in the youngest age groups compared to 2000: if these youngest age groups experienced significant declines in morbidity then results that are fixed to the population distribution in 1900 will yield a greater value. In order to avoid any bias caused by this feature the thesis will utilise the mid point between these two sets of results. The detailed calculations for this process are contained in appendices 12.3 and 13.1. The summarised results of this analysis are shown in the series of tables below (and Appendix 12.4).

Table 8.1.3, 8.1.4 and 8.1.5 consider the value of declines in the burden of morbidity through: first, applying the above exercise of identifying the change in the morbidity burden (prevalence * QALY) weighted to the start (T1) and end (T2) point populations, which is then applied to the VSHLY estimate to generate the monetary value of improvements in morbidity, the morbidity gain. These are then averaged through identifying the mid point between the T1 and T2 morbidity gain, in order to provide a single morbidity gain estimate (morbidity burden change 1 * VSHLY = morbidity gain 1 and morbidity change 2 * VSHLY= morbidity gain 2 → [morbidity gain 1 + morbidity gain 2] / 2 = morbidity gain).

It is noteworthy that in all of the tables in Chapter 8 and 9, that the result for 1900-2000 does not equal the sum of the previous five entries in these tables. This is because all of these entries are considering averages that relate to the specific numbers of each period. For example, the VSL and VSHLY and QALY and prevalence all differ for each era (i.e. 1900-2000 is not simply summing the results of all the other eras) and the subsequent interaction in the methodology yields different average estimates. Furthermore, when considering growth (which is achieved in Table 8.1.14, 8.1.15, 8.2.8, 8.3.4, and 9.3) the methodology is considering a compound average rate of growth, which again considers an average (rate) that is different for each computation of the twentieth century[681].

Table 8.1.3: Morbidity gain (morbidity burden change*VSHLY): monetary value of improvements in the burden of morbidity: stomach cancer (millions of 1990 international $)[682]

Period	**Morbidity Burden Change 1**	**Morbidity Burden Change 2**	**VSHLY**	**Morbidity Gain T1**	**Morbidity Gain T2**	**Morbidity Gain Average**
1900-1925	1116	1510	0.21	234	317	278
1925-1950	121	-164	0.32	39	-52	-7
1950-1975	5598	6611	0.68	3807	4495	4151
1975-2000	2678	2714	1.36	3642	3691	3677
1900-2000	6235	12779	0.44	2743	5623	4183

Table 8.1.3 calculates the morbidity gain. Morbidity burden 1 and 2 are calculated in appendices 12.3 and 13.1 (which consider the morbidity burden rate change for the age distribution of the population, in the same format as Table 8.1.2.i and ii). The morbidity burden changes 1 and 2 are calculated as the difference in the morbidity burden in T1 versus T2, fixed to the corresponding age distribution of the population in T1 (morbidity change 1) and T2 (morbidity change 2). The morbidity gain T1 and T2 are the result of: morbidity burden change 1 and 2 multiplied by the VSHLY. The morbidity gain average is the median of morbidity gain T1 and morbidity gain T2.

The most far reaching morbidity gains related to stomach cancer (in Table 8.1.3 above) were evident for 1900-2000. This was closely followed by the morbidity gains in the

[681] This note applies to all of the tables in this results section, which consider the morbidity, mortality and QALE gain consider the following eras: 1900-1925, 1925-1950, 1950-1975, 1975-2000 and 1900-2000.
[682] For detailed calculations underlying the results in Table 8.13 see Appendix 12.3 and 13.1. For summary results shown in Table 8.1.3 see Appendix 12.4.

second half of the twentieth century: 1950-1975 and 1975-2000. The reasons for the chronology of this trend are largely related to quality of life and prevalence improvements. Hence, Table 8.1.1 implies that between 1950 and 1975 there was the greatest decline in the morbidity burden as a result of reductions in prevalence (17,300 in 1950 declined to 14,098 in 1975) and, more importantly, the improving QALY (the burden, represented by 1-QALY improved from [in QALE terms, a loss of] 0.5000 to only 0.3333 of a healthy life year). The morbidity gain in the following period, 1975-2000, was a function of only reductions in prevalence as there was no QALY improvement. The morbidity gain for 1900-2000, which was the most far reaching, was a result of both improved prevalence and QALY (Table 8.1.1 shows a decline in prevalence from 7,989 to 7,705 enhanced by a decline in the burden of a healthy life year [=1-QALY] from 0.6667 to 0.3333).

Also noteworthy is that relative to the other thesis diseases, the values of the stomach cancer morbidity gains were not substantial. This is largely a result of the failure to significantly increase the quality of life (as reflected by the QALY in Table 8.1 or 1-QALY in Table 8.1.1) for stomach cancer sufferers, especially when this is compared to the other illnesses in the thesis. This is also because of a more limited prevalence relative to breast cancer and especially tuberculosis.

Breast cancer (see Table 8.1.4) experienced a similar phenomenon to stomach cancer, in that the most valuable morbidity gains were evident during the second half of the twentieth century. This result is not surprising, as Part II has outlined, there were few advances in (the key quality of life variable) medical technology associated with cancer before 1950.

Results: Quantitative: Disease

Table 8.1.4: Morbidity gain (morbidity burden change*VSHLY): monetary value of improvements in the burden of morbidity: breast cancer (millions of 1990 international $)[683]

Period	**Morbidity Burden Change 1**	**Morbidity Burden Change 2**	**VSHLY**	**Morbidity Gain T1**	**Morbidity Gain T2**	**Morbidity Gain Average**
1900-1925	-868	-1000	0.21	-182	-210	-196
1925-1950	-2062	-2586	0.32	-660	-828	-741
1950-1975	13287	14952	0.68	9035	10167	9601
1975-2000	11092	11102	1.53	16971	16986	16979
1900-2000	8931	13836	0.51	4555	7056	5806

Table 8.1.4 shows that the most substantial morbidity gains for breast cancer were in the period 1975 to 2000. This is not surprising because as the twentieth century unfolded the morbidity gain for breast cancer became increasingly valuable. Compared to stomach cancer, breast cancer experienced many more far reaching improvements in quality of life (as reflected by the QALY in Table 8.1 or 1-QALY in Table 8.1.1), which explains the greater morbidity gain experienced by breast cancer in the second half of the twentieth century. However, during the period 1900-1950 breast cancer achieved worsening morbidity gains (versus stomach cancer), which was largely as a result of substantially increasing prevalence of breast cancer (from 15,980 in 1900 to 61,477 in 1950) and the failure of the QALY to improve until 1950. This trend also explains why when the twentieth century is considered as a whole, the morbidity gain is of similar magnitude for breast and stomach cancer.

This post 1950 breast cancer morbidity gain is particularly important because, as has been outlined above, this occurred during an era when the prevalence of breast cancer was always increasing (as shown in Table 8.1.1) and therefore the constantly improving QALY burden is the factor which drives the morbidity gain, especially between 1975 and 2000, which experienced considerable increases in prevalence (from 68,953 in 1975 to 87,915 in 2000, shown in Table 8.1.1) that were entirely offset by improvements in the quality of life associated with breast cancer (in Table 8.1.1, the burden, represented by 1-QALY improved from 0.3333 in 1975 to only 0.1667 of a healthy life year in 2000).

[683] For detailed calculations underlying the results in Table 8.14 see Appendix 12.3 and 13.1. For summary results shown in Table 8.1.4 see Appendix 12.4.

Tuberculosis sufferers also experienced considerable morbidity gains (see Table 8.1.5), some of which can be ascribed to the significant improvements in quality of life related to tuberculosis, particularly during the second half of the twentieth century. The substantial decline in the prevalence of tuberculosis also contributed to the morbidity gains outlined in Table 8.1.5.

Table 8.1.5: Morbidity gain (morbidity burden change*VSHLY): monetary value of improvements in the burden of morbidity: tuberculosis (millions of 1990 international $)[684]

Period	**Morbidity Burden Change 1**	**Morbidity Burden Change 2**	**VSHLY**	**Morbidity Gain T1**	**Morbidity Gain T2**	**Morbidity Gain Average**
1900-1925	28168	22021	0.27	7605	5946	6776
1925-1950	18728	17215	0.45	8428	7748	8088
1950-1975	13977	13556	0.87	12160	11794	11977
1975-2000	377	330	1.71	645	565	605
1900-2000	61865	41322	0.51	31551	21074	26313

Table 8.1.5 highlights the tremendous tuberculosis morbidity gains as a result of improvements in both the prevalence and the QALY. This was most pronounced for the period 1900-2000, as would be expected, because the prevalence of tuberculosis declined consistently throughout the twentieth century. Also of relatively very high magnitude was the morbidity gain between 1950 and 1975, which is a likely result of significant improvements in the QALY (in Table 8.1.1, the burden, represented by 1-QALY improved from 0.3333 in 1950 to 0.1667 in 1975) and a significant decline in the prevalence during this period (from 42,024 in 1950 to 7,119 in 1975, shown in Table 8.1.1), both of which were a result of medical developments (see Part II: Chapter 5 and Part III: Chapter 7.2 for an elaboration). Along a less positive vein is the relatively minimal morbidity gain between 1975 and 2000. This is largely a result of minor scope for further improvements, as the QALY nearly reached the ideal level (when considering 1-QALY, the ideal level would be 0 and the tuberculosis 1-QALY had reached 0.1667 by 1975, which can be considered as nearly ideal) and the prevalence declined so that a very marginal proportion of the population were infected with tuberculosis.

[684] For detailed calculations underlying the results in Table 8.1.5 see Appendix 12.3 and 13.1. For summary results shown in Table 8.1.5 see Appendix 12.4.

Results: Quantitative: Disease

When the morbidity gains for the three diseases are compared, the most striking feature is the greater magnitude of the tuberculosis morbidity gain. E.g. if the morbidity gain for these diseases are considered for the twentieth century as a whole (1900-2000), the tuberculosis morbidity gain was about five times greater than the breast cancer morbidity gain and about six times greater than the stomach cancer morbidity gain (26,313 versus 5,806 and 4,183, respectively [all in 1990 international $]). A key reason for the greater magnitude of the tuberculosis morbidity gain relative to the other illnesses in the thesis is the dramatic decline in the prevalence of this disease. For example, in 1900, tuberculosis represented nearly 10 percent of all deaths in the English population. This is a greater proportion than breast cancer and stomach cancer combined, at any point during the twentieth century, and any improvements are going to be more far reaching because of the greater number of healthy life years that are achieved out of reducing the prevalence and morbidity burden associated with tuberculosis. This trend also explains part of the reason for breast cancer experiencing higher morbidity gains versus stomach cancer. Hence, the greater the prevalence of a disease, the greater is the scope for the morbidity gain, as there are more life years that will be effected by any QALY improvements.

Therefore, Tables 8.1.3 to 8.1.5 highlight the value of improvements in morbidity. This is particularly pronounced for tuberculosis, which would be expected as this disease had transformed from being one of the major health threats in 1900 to being virtually eliminated by 2000 (due to the availability of safe and effective antibacterial agents). Breast cancer also experienced important gains associated with improved quality of life (QALY), which were especially far reaching as they were capable of counteracting increases in prevalence. Lastly, although compared to these two diseases stomach cancer was less significant, the associated morbidity gains were still noteworthy and also contribute to the overall conclusions of the thesis in a similar way to breast cancer and tuberculosis, although to a lesser extent.

Now that the morbidity gain for the thesis diseases has been established, the final aspect of the methodology is to combine this with the mortality gain as this provides the overall health gain or in the context of the thesis' methodology and terminology, the QALE gain. In order to form the overall QALE methodology, the morbidity gain (above) will be combined with the mortality gain (below). The methodology and result of the mortality gain calculation will be explained below. With reference to the flow chart example at the

beginning of Chapter 8, this next stage of valuing the morbidity gain relates to stage 8 in the flow chart and WTP mortality in Equation 8.1.

8.1.2 Primary Valuation of Improvements in Mortality

The results below highlight twentieth century improvements in mortality through a very similar process to that which was utilised for morbidity, but instead of considering the burden of illness, the death rate burden is utilised and the change in the death rate burden is valued with the VSL (rather than the VSHLY which was used to value the change in the morbidity burden). Selecting the VSL represents stage 6 in the above flow chart example and included in this outline is Table 8.2, which provides the calculation and result of the VSL values for different eras of the twentieth century.

The first step in identifying the mortality gain is to identify the change in the mortality burden. This is established through considering the change in the death rate. This is a much more simplistic and straightforward process compared to disease as the change in the death rate has been recorded for the entire twentieth century and it is a straightforward statistic (i.e. there is no ambiguity about whether someone is registered as ‘dead’ or remains alive). Hence, once the change in the death rate has been identified, it is possible to value this through applying the VSL to the change (always a decline in the twentieth century) in the death rate. The result of this calculation (decline in death rate*VSL) will yield the mortality gain. This process is highlighted below in Table 8.1.6.

Also in common with the above calculation of the change in the morbidity burden, the change in the death rate burden was considered relative to the age distribution of the population. Therefore, it was necessary to weight these calculations to age profiles at the start and end point (T1 and T2, respectively) of the populations being considered and then identify the mid point between these two sets of results and utilise this as the mortality gain. The summary of this process is shown below in Table 8.1.6 and the detailed calculations for this process are contained in appendices 12.3 and 13.1.

Table 8.1.6: Mortality gain (mortality burden change*VSL): monetary value of improvements in the burden of mortality (millions of 1990 international $)[685]

Period	Mortality burden change T1	Mortality burden change T2	VSL (millions)	Mortality gain T1 (MG1)	Mortality gain T2 (MG2)	Average mortality gain
1900-1925	6201	5968	0.64	3969	3820	3895
1925-1950	4494	4974	0.76	3415	3780	3598
1950-1975	1906	2386	1.16	2211	2767	2489
1975-2000	3781	4034	2.05	7751	8270	8011
1900-2000	13805	17487	0.88	12149	15389	13769

Table 8.1.6 highlights the methodological process and result of valuing the decline in the death rate. I.e. the mortality burden change columns multiplied by the 'VSL' column yields the mortality gain columns. The mid point between these two mortality gain columns can then be identified to yield the 'average mortality gain' result, which will be utilised.

The most substantial mortality gains in the above table are those that exist for the entire twentieth century. This is essentially a result of the fact that this era includes the largest scope for death rate reductions and subsequent mortality gains. Also noteworthy is the value of the mortality gain in 1975-2000, which is larger than any of the preceding eras. This is a result of continued improvements in the life expectancy and also because of an increased value of these improvements. I.e. the VSL for 1975-2000 is nearly double the VSL for the preceding era, 1950-1975, which is also higher than all other VSL values (1975-2000 VSL = 2.05 and 1950-1975 VSL = 1.16 [1990 international $])[686]. The reasons for this difference are explained in Chapter 3, where the rationale and construction of the VSL are detailed.

Lastly, it is important to note that all eras considered in the table experienced important mortality gains, which is essentially a result of twentieth century improvements in mortality that were unprecedented, such that the entire twentieth century mortality gain was worth 13,769,000,000 or approximately fourteen billion (1990 international $).

[685] For detailed calculations underlying the results in Table 8.1.6 see Appendix 12.3. For summary results shown in Table 8.1.6 see Appendix 12.4.
[686] For details of the calculation of the VSL see Appendix 12.1.1.

An improvement in the death rate and a subsequent valuable mortality gain has been recognised in the literature and is therefore not one of the original contributions of the thesis. However, calculating these quantitative gains for morbidity has not, to date, been attempted. Moreover, combining these quantitative morbidity gain evaluations with the mortality gain in a universal health measure, namely, the QALE, is also one of the original key contributions to knowledge. This final aspect of the methodology (combining the mortality and morbidity gain) is achieved below with the QALE gain valuation, where the novelty of the thesis' original QALE methodology is highlighted. This QALE methodology, which combines the WTP morbidity and WTP mortality gain represents stage 10 in the flow chart example provided at the beginning of Chapter 8 and is also the final stage in Equation 8.1.

8.1.3 QALE Gain Valuation

This subsection of results combines the morbidity gain and mortality gain that have been calculated above, in order to estimate the QALE gain (i.e. morbidity gain + mortality gain = QALE gain).

In Table 8.1.7 the QALE gain is considered for the three thesis diseases independently. Hence, 'QALE Gain stomach cancer' comprises the morbidity gain for stomach cancer (calculated above in Table 8.1.3) summed with the mortality gain (calculated in Table 8.1.6), and so forth for each disease. In theory all three disease morbidity gains and the mortality gain could be summed together (along with the thesis QALE gain for blindness, which will be achieved in Chapter 8.3), as well as all disease and disability morbidity in the economy (which will be achieved in Chapter 9). However, for analysis purposes the three diseases and disability will be kept separate and each will be independently summed with the mortality gain. This calculation is shown below in Table 8.1.7.

Table 8.1.7: QALE gain (morbidity gain + mortality gain): monetary value of improvements in the burden of morbidity and mortality for thesis diseases: breast cancer, stomach cancer, tuberculosis (millions of 1990 international $)[687]

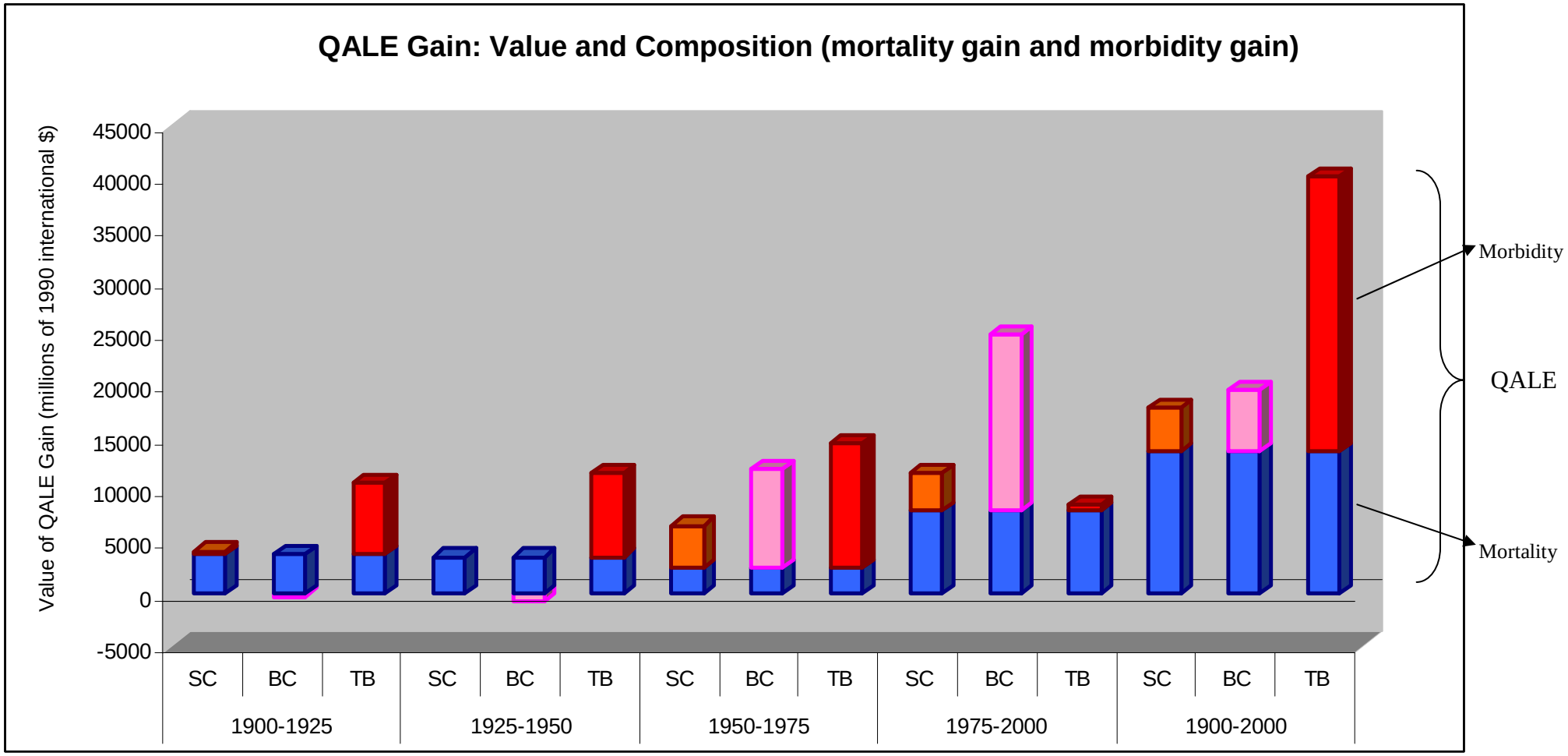
Period	Mortality Gain	Morbidity Gain: Stomach cancer	QALE Gain Stomach cancer	Morbidity Gain: Breast cancer	QALE Gain Breast cancer	Morbidity Gain: Tuberculosis	QALE Gain Tuberculosis
1900-1925	3895	278	4173	-196	3699	6776	10671
1925-1950	3598	-7	3591	-741	2857	8088	11686
1950-1975	2489	4151	6640	9601	12090	11977	14466
1975-2000	8011	3677	11688	16979	24990	605	8616
1900-2000	13769	4183	17952	5806	19575	26313	40082

In Table 8.1.7 the mortality gain is derived from Table 8.1.6, where it was calculated. The morbidity gain for stomach cancer is derived from Table 8.1.3, for breast cancer from Table 8.1.4 and for tuberculosis from Table 8.1.5, where these diseases morbidity gains have been calculated. The QALE gain columns in the above table are the sum of the mortality gain column and the associated disease morbidity gain column. For example, continuing with breast cancer between 1900 and 2000: the morbidity gain has been calculated (in Table 8.1.4) as 5806 (1990 international $) for 1900-2000 and the mortality gain for 1900-2000 is 13769 (1990 international $), the QALE gain is the sum of these two facets = 19575 (5806 + 13769), which is shown in the above table for the QALE gain for breast cancer between 1900 and 2000.

[687] For detailed calculations of the QALE gain in Table 8.1.7 (which is considering the QALE gain as the sum of the morbidity gain [Table 8.1.3, 8.1.4, 8.1.5] and the mortality gain [Table 8.1.6]) see Appendix 12.5.

Hence, Table 8.1.7 considers the value of the morbidity gain (for the specific illness) summed with the mortality gain, which represents the QALE gain. This table illustrates a trend which is expected: for all eras where the morbidity gain was positive, the QALE is higher than the mortality gain and for all eras where there was a worsening in the morbidity gain (i.e. a morbidity loss) the QALE gain is lower than the mortality gain. The direction and magnitude of these combinations can be deduced from the above table and this will be considered in Figure 8.1.1 overleaf, in order to try and indicate the contribution of the morbidity gain and mortality gain towards the overall QALE gain.

Figure 8.1.1: QALE gain: value and composition of the morbidity gain and mortality gain towards QALE gain for thesis diseases: stomach cancer (SC), breast cancer (BC) and tuberculosis (TB), 1900-2000 (millions of 1990 international $)[688]



[688] See Appendix 12.5 for QALE gain and the composition of the morbidity and mortality gain towards the QALE gain.

Results: Quantitative: Disease

Figure 8.1.1 illustrates the contribution of the mortality gain (blue bar connected to the x axis) and the morbidity gain (orange [stomach cancer], pink [breast cancer], red [tuberculosis] bars on top of the blue, mortality bars) to the overall QALE gain (height of both bars for each disease and era). Hence, the height of these bars (composed of blue and orange or pink or red segments) represents the value of the QALE gain. Furthermore, the placement of the morbidity bars also represents the direction (positive or negative) of the morbidity contribution to the overall QALE gain. If the morbidity bar is on top of the mortality gain bar then the morbidity gain has provided a positive contribution to the QALE gain and if the morbidity bar is below the mortality bar the morbidity gain is negative and has caused a reduction in the QALE gain. This latter phenomenon only occurred twice: for breast cancer in 1900-1925 and 1925-1950.

For example, if we continue with twentieth century breast cancer: Table 8.1.7 has calculated the QALE gain by summing the mortality gain (calculated in Table 8.1.6) and the morbidity gain for breast cancer (calculated in Table 8.1.4). The relationship of these three variables is illustrated in Figure 8.1.1 through, first presenting the mortality gain (represented by the blue bar, at a value of 13,769 [shown in Table 8.1.7 and Figure 8.1.1 where it is represented by the height of the blue bar = 0 to 13,769]), second is the morbidity gain which is represented by the pink bar (which sits directly on top of the blue bar and represents a value of 5,806 [shown in Table 8.1.7 and Figure 8.1.1 where it is represented by the height of the pink bar = 13,769 to 19,575, so 19,575 – 13,769 = 5,806]), lastly, the QALE gain is represented by the combined height of the blue and pink bar which indicates a QALE gain value of 19,575 (millions of international $) for breast cancer between 1900 and 2000. This is calculated by summing the mortality gain and the morbidity gain in accordance with the QALE methodology (see Figure 8.1 for a reminder of this methodology). Finally, because the pink bar is on-top-of the blue bar, this highlights that the morbidity gain has provided a positive contribution to the overall QALE gain.

Hence, for ease of review Figure 8.1.1 illustrates the QALE gain segmented for the contribution of the mortality gain and morbidity gain. This illustrates the direction of the morbidity gain (or loss) and the magnitude of the morbidity gain in order to highlight how mortality and morbidity are driving the change in the overall QALE gains.

Through considering the magnitude and direction of the morbidity gain relative to the mortality gain, in order to determine the contribution of improved morbidity to the QALE,

Figure 8.1.1 illustrates two key points. First, that there were only two instances where there were negative contributions from morbidity (breast cancer in 1900-1925 and 1925-1950, shown by the pink bar being below the blue bar in Figure 8.1.1) and there was only one zero contribution (from stomach cancer in 1925-1950, shown by only a blue bar for the mortality gain as there was no morbidity gain, which is represented by a zero contribution). Hence, in 12 out of 15 instances, morbidity provided a positive contribution to mortality. This finding provides a strong justification about the value of twentieth century improvements in morbidity and the need to measure this in conjunction with mortality.

The other striking feature of Figure 8.1.1 is the magnitude of morbidity gains relative to mortality gains in many instances. The reason for this is very important and is essentially because of the difference in the inherent nature of morbidity and mortality, with regards to their influence upon the population. Mortality is a one time only event whereas morbidity has, in theory, an infinite number of possible events (which can even occur concurrently, i.e. co-morbidity), confined only by the one time occurrence of mortality. I.e. while an individual is alive, there is scope for an infinite number of morbidity events but once one mortality event has occurred there is scope for no more as the individual is dead. Furthermore, as the twentieth century unfolded the disease environment evolved such that the most prevalent illness burdens switched to chronic and not infectious diseases, which augmented the persistence of morbidity. This reality provides a subtle but very powerful message about the importance of considering more than just mortality when trying to gauge the health of the nation, as there are many (more) improvements evident for morbidity. This finding helps to bolster one of the foundational claims of the thesis: the need to measure health (morbidity) in some form of model that can calculate mortality and morbidity improvements in tandem.

Furthermore the momentous improvement in tuberculosis also fills a significant historical void. To date, the contribution of improvements in this infectious disease is likely to have been underestimated as it was not possible to make the above quantifications, which yield important results about morbidity. Furthermore, this trend is likely to be consistent with the vast majority of infectious diseases and therefore provides an even greater boost to existing credit for the medical revolution which eliminated this category of morbidity and mortality.

Along a similar vein, the results for breast and stomach cancer are also momentous, largely for the evidence against pessimistic commentaries of the epidemiological transition that they provide. Hence, even though the prevalence of these diseases has increased (to replace infectious diseases) during the twentieth century, the improvements in quality of life associated with these diseases (fostered by advanced medical technology and welfare initiatives) has been significant enough (in the majority of instances) to have created a more favourable morbidity situation over the twentieth century, illustrated by the increase in the morbidity gain (pink and orange bars) in Figure 8.1.1.

Finally, when considering breast and stomach cancer, the magnitude of the QALE gain for these diseases in the period 1900-2000 is noteworthy. This similarity was identified below Table 8.1.4 but deserves an additional consideration. This statistic seems misleading at an initial approximation, especially if it is viewed in conjunction with Table 8.1.1, which highlights the differing prevalence and burden of stomach and breast cancer. However, it is necessary to maintain that the QALE gain is actually considering the compound average change in the morbidity burden [and an identical mortality gain for breast and stomach cancer] which yields relatively similar results that are accurate when the entire twentieth century is considered. A key reason is that the value of the change in the morbidity burden between 1900 and 2000, which, by coincidence are relatively similar for breast cancer and stomach cancer, and also because of the QALY for 1900-2000, although this is higher for breast cancer. These illnesses share the same 1900 QALY and the higher breast cancer QALY by 2000 actually serves to contribute to the coincidental (but not wrong or anomalistic) similarity in the breast cancer and stomach cancer morbidity gains for 1900-2000.

The key feature to note from the above analysis (illustrated in Figure 8.1.1) is the largely positive contribution provided by the morbidity gain (which embodies improvements in the prevalence and/or the burden of morbidity) and the mandate this provides for adopting a fuller notion of health and the indication it provides about the inaccuracy of pessimist claims about the worsening morbidity associated with improving mortality, which is not true when a more rounded view of morbidity is adopted, which has been achieved here for the first time in the literature (for example, if the pessimists were correct then every morbidity bar in Figure 8.1.1 would be below the corresponding mortality bar, as this represents morbidity detracting mortality gains, which is simplistically what the pessimist

school are claiming). The above findings and conclusions will be verified below through a series of sensitivity analyses.

8.1.4 Sensitivity Analyses

In order to highlight the veracity of the above findings and the more general claims of the thesis which are based upon this analysis, it is desirable to test the results through considering different values (Low and High as well as the existing Mid value, utilised above) for the most tenuous variables in the thesis, namely, QALY, VSL and VSHLY[689]. This will generate a broad spectrum of possible results (precisely 378) about the QALE gain.

A final aspect of the sensitivity analysis is age-weighting. Murray's age weighting function will be applied to the range of QALE gain results in order to provide estimate that account for claims in the literature that these gains have been more and less valuable for certain ages. For an elaboration of the appeal and process of age weighting see Chapter 3. This will provide an additional series (containing 378 results) of QALE gain estimates.

Essentially this section will repeat the above methodological procedures (of identifying the morbidity gain then mortality gain and the subsequent QALE gain) but using different QALY, VSL and VSHLY variables. These alternative QALE gain results (as well as those identified in Table 8.1.7) will also be applied to Murray's age-weighting function. This part of the methodology is represented by stage 11 in the flow chart example at the beginning of Chapter 8 (and also draws on stages 5 to 10 as it is repeating the above methodological process).

8.1.4.1 QALY Weights

The QALY was established through an intricate and objective qualitative analysis of the health and welfare related quality of life for different illnesses and eras of the twentieth century (conducted in Part II of the thesis). Developing a QALY for different illnesses and eras is one of the thesis' core contributions to knowledge. The QALYs used here are totally unique and wide reaching as they have been derived from a detailed review of the literature and data relevant to the key aspects of health and welfare related quality of life for the thesis illnesses during the twentieth century. Because of the potential scope for subjectivity

[689] The author of the thesis believes the 'Mid' age-weighted estimates to be the most accurate and plausible, but has provided a series of coherent alternatives in an effort to validate the estimates and overall conclusions of the thesis.

and/or opinion variance in the thesis' analysis and also associated with QALYs in general, a range of QALY estimates will be considered. With reference to the flow chart example at the beginning of Chapter 8, this represents stage 11b of the methodology and will also heavily utilise stage 5 as the QALY is reconsidered. Stages 5 to 10 will also be implemented as the QALE methodology is recalculated using an alternative QALY for the morbidity burden (=prevalence*QALY).

The range of QALY estimates used in this analysis considers the mid QALY estimate, which is depicted by 'Mid' in conjunction with a 'Low' and 'High' estimate. These two estimates were derived through identifying the QALY level below and above the 'Mid' estimate (which has been presented above in Table 8.1). This is likely to be an exaggeration of the variance that a critic would want, but is intended to provide an extreme range in order to highlight the veracity of the thesis claims. The range of QALY estimates, for the diseases considered in the thesis are shown below. For a more detailed explanation about the QALY and QALY value eliciting in the thesis, see Chapter 3 and the flow chart example at the beginning of Chapter 8.

Table 8.1.8: Alternative QALY values (Low, Mid, High) for thesis diseases: breast cancer, stomach cancer, tuberculosis[690]

Year	**Breast Cancer**			**Stomach Cancer**			**Tuberculosis**		
	Low	**Mid**	**High**	**Low**	**Mid**	**High**	**Low**	**Mid**	**High**
1900	0.1667	**0.3333**	0.5000	0.1667	**0.3333**	0.5000	0.1667	**0.3333**	0.5000
1925	0.1667	**0.3333**	0.5000	0.1667	**0.3333**	0.5000	0.3333	**0.5000**	0.6667
1950	0.3333	**0.5000**	0.6667	0.3333	**0.5000**	0.6667	0.5000	**0.6667**	0.8333
1975	0.5000	**0.6667**	0.8333	0.5000	**0.6667**	0.8333	0.6667	**0.8333**	1
2000	0.6667	**0.8333**	1	0.5000	**0.6667**	0.8333	0.6667	**0.8333**	1

Table 8.1.8 illustrates the mid QALY, which has been derived through the thesis' analysis in the 'Mid' column, shown in bold, and this is sandwiched between a lower ('Low') and upper ('High') estimate. This part of the sensitivity analysis is designed to address concerns about the accuracy of the valuation of unhealthy life year estimates, and therefore an exaggerated margin of error has been considered for the value of the QALYs. The alternative positive margin is presented in the 'High' column. I.e. this provides a more generous QALY estimate for the reader who is concerned that the thesis (mid) QALY

[690] See Part II and Chapter 7 and Chapter 3 and see Appendix 12.6.1 for alternative QALY weights.

estimates are too pessimistic, and the negative margin is presented in the ‘Low’ column and addresses observer concerns about the thesis (mid) QALYs being too generous.

Once the values of the QALYs have been estimated it is possible to apply them to the methodology in the same way that has been achieved above for the ‘Mid’ QALY. Hence, the first stage is to estimate the morbidity burden, through the same process outlined previously in this chapter: the morbidity burden is estimated through identifying and combining (multiplying) the prevalence and the QALY (as above, when doing the calculation 1-QALY is utilised) associated with the disease and era, for the age standardised population structure in T1 and T2 (which is later average through identifying the mid-point between T1 and T2). Once this has been achieved it is possible to measure the change in the morbidity burden between the start [T1] and end [T2] point of the period. This change in the morbidity burden can then be valued through combining it with the VSHLY, which will yield the morbidity gain, in this instance for higher and lower QALY variable.

These alternative (higher and lower) QALYs will be utilised in two places in the methodology: i) when estimating the morbidity burden (prevalence * QALY) and ii) when estimating the VSHLY (VSL * QALY).

As in the above methodological process, this morbidity gain (for higher and lower QALYs) can then be combined with the mortality gain (which will remain the same as it was in the previous section because the QALY change only affects the morbidity features of the QALE methodology) in order to estimate the QALE gain. This is achieved below for alternative QALY weights for the QALE gain, where the mortality gain utilised is the same as that presented in Tables 8.1.6 and 8.1.7 and the morbidity gain is considered for the ‘Mid’ QALYs (which are the same as the existing estimate in Tables 8.1.3, 8.1.4, 8.1.5 and 8.1.7) and the ‘Low’ and ‘High’ alternative sensitivity analysis QALYs.

Table 8.1.9: QALE gains for thesis diseases: breast cancer (BC), stomach cancer (SC), tuberculosis (TB) with alternative (Low, Mid, High) QALY values (millions of 1990 international $)[691]

Period	LOW QALY			MID QALY			HIGH QALY		
	QALE Gain SC	QALE Gain BC	QALE Gain TB	QALE Gain SC	QALE Gain BC	QALE Gain TB	QALE Gain SC	QALE Gain BC	QALE Gain TB
1900-1925	4035	3794	7935	4173	3699	10671	4317	3593	13318
1925-1950	3594	3153	9337	3591	2857	11686	3588	2560	13920
1950-1975	5445	9319	11807	6640	12090	14466	7805	14784	17145
1975-2000	10770	21260	8493	11688	24990	8616	12610	28835	8734
1900-2000	16546	17929	32624	17952	19575	40082	19324	21251	47682

Table 8.1.9 is making the same considerations that were made earlier in Table 8.1.7, but in less detail. Hence, the above table presents the final QALE gain result (for different QALY values), whereas Table 8.1.7 made the same calculation (using the mid QALY) but included the mortality gain and the diseases specific 'morbidity gains' as well as presenting the final result, the QALE gain. These detailed calculations for alternative QALY weights are conducted in appendices 12.7.1 and 13.2.1.

The QALE gain results in the above table are generally what would be expected. Hence, the higher the QALY weight (i.e. increasing from 'Low' to 'Mid' to 'High'), the greater the magnitude of the QALE gain. This is for two key reasons; as the QALY approaches one, the burden of the disease declines and provides a greater morbidity (and subsequent) QALE gain and also, the higher the QALY (closer to one), the higher the VSHLY becomes, which also contributes to increasing the value of the morbidity (and subsequent) QALE gain. With reference to the flow chart example and specifically the EuroQol to QALY conversion table (Table 7.1), the higher the QALY, the closer the quality of life for illnesses sufferers was to 'complete quality of life', which would represent a lower morbidity burden.

However, there are exceptions to this trend: breast cancer in 1900-1925 and 1925-1950, and stomach cancer in 1925-1950. These are a result of the morbidity gain being negative

[691] Table 8.1.9 is based on the same type of calculations as Table 8.1.7. See Appendix 12.6.1 for alternative QALY weights. See Appendix 13.2.1 for calculation of QALE gain using alternative QALY weights (see mid VSL, mid VSHLY with low, mid, high QALY).

or zero (in the case of stomach cancer). Hence, under these circumstances, increasing the QALY increases the amount of value lost and the subsequent detraction for the QALE gain. This phenomenon (of a negative or zero morbidity gain) is the same as that which was identified for the mid QALY QALE gain shown in Table 8.1.7.

Also noteworthy is the similarity of the values yielded, regardless of the QALY weight. This adds credibility to the estimates of the thesis as it indicates that, regardless of the QALY value chosen (within a very generous margin) the aggregate conclusion is the same.

Hence, the general results of this QALY sensitivity analysis highlight that when the QALY is higher the contribution to overall health and the relationship with mortality is higher and vice versa when the QALY is lower. In some cases the contribution is negative as a result of a worsening morbidity profile. When the QALE contribution is negative, the higher the QALY the more negative the QALE gain (or loss) becomes.

8.1.4.2 VSL and VSHLY Weights

The thesis has already presented the disputes associated with the identification of the correct VSL and subsequent VSHLY (see Chapter 3). This will be abated as much as possible through considering the QALE gain with a series of additional ('Low' and 'High') weights for the VSL and VSHLY components of the methodology. This aspect of the sensitivity analysis makes the same methodological calculations as above (i.e. morbidity gain + mortality gain = QALE gain), but will do this with alternative VSL (for valuing the mortality gain) and VSHLY (for valuing the morbidity gain) weights, namely, a lower and a higher weight series to enhance the mid weights that have been utilised above.

With reference to the stages in the above flow chart example, this represents stage 6: selecting the VSL and stage 7: calculating the VSHLY, and also an integral aspect of stages 8 and 9: the valuation of the mortality burden change (VSL) and the valuation of the morbidity burden change (VSHLY), respectively. After which stage 10 will be conducted, where the new morbidity gain and mortality gain are summed to estimate the QALE gain (for alternative VSL and VSHLY weights). More specifically, this process relates to stage 11a and 11c, as this represents the sensitivity analysis of the VSL and VSHLY.

Alternative VSL and VSHLY values are derived through considering the low and high estimates of Miller's VSL multiple, provided in Chapter 3: Table 3.3. With reference to the

worked example above in Chapter 8, Table 8.2 illustrates the calculation of the VSL (VSL mid multiple [128] * GDP per capita at mid point). In the VSL sensitivity analysis here, the thesis will be changing the VSL multiple (from 128 to 101 and 154) in order to estimate a lower and higher VSL. The GDP per capita component remains the same.

The VSHLY is a function of the VSL and the QALY (VSL*QALY=VSHLY). With reference to the above worked example in Chapter 8, Table 8.3 shows the construction of the mid VSHLY. The VSHLY calculation involves: i) a new VSL (higher or lower, explained above) and ii) the same, mid QALY for the VSL*QALY=VSHLY calculation.

The results presented in this VSL and VSHLY sensitivity analysis (presented in Table 8.1.10) are derived from an intricate consideration of all the possible combinations of VSL and VSHLY within the QALE methodology (shown in Appendix 13.2.1). Table 8.1.10 (below) presents the QALE gain results for VSL and VSHLY mid values (which are equal to the pre sensitivity analysis results, shown in Table 8.1.7) and the most extreme values (i.e. 'Low' VSL, 'Low' VSHLY and 'High' VSL, 'High' VSHLY).

Table 8.1.10: QALE gains for thesis diseases: breast cancer (BC), stomach cancer (SC), tuberculosis (TB) with alternative VSL and VSHLY values (millions of 1990 international $)[692]

Period	**Low VSL Low VSHLY**			**Mid VSL Mid VSHLY**			**High VSL High VSHLY**		
	QALE Gain SC	**QALE Gain BC**	**QALE Gain TB**	**QALE Gain SC**	**QALE Gain BC**	**QALE Gain TB**	**QALE Gain SC**	**QALE Gain BC**	**QALE Gain TB**
1900-1925	3314	2935	8404	4173	3699	10671	5053	4475	12815
1925-1950	2855	2276	9180	3591	2857	11686	4353	3470	13997
1950-1975	5220	8678	11405	6640	12090	14466	7959	13231	17390
1975-2000	9211	19722	6785	11688	24990	8616	14045	30071	10346
1900-2000	14201	15505	31720	17952	19575	40082	21653	23642	48364

The table above considers the QALE gain with a series of different weights for the valuation of the change in the mortality burden (VSL) and for the change in the morbidity burden (VSHLY). Table 8.1.10 presents QALE gain results which utilise the mid and most extreme (lowest and highest) VSL and VSHLY values. The overall trend is the positive correlation between the value of the VSL and VSHLY and the value of the QALE gain.

[692] Table 8.1.10 is based on the same type of calculations as Table 8.1.7, but with alternative VSL and VSHLY values. See Appendix 12.1.1 for VSL and VSHLY range of values to be included in sensitivity analyses and Appendix 13.2.1 for calculations (see mid QALY with low, mid, high VSL and VSHLY).

This is expected because the higher the value of improvements in these health valuation variables (embodied in the VSL being applied to the change in the mortality burden and VSHLY to the change in the morbidity burden), the higher the QALE gain.

The results in Table 8.1.10 provide a substantial indication about the consistency of the value of improvements in mortality and morbidity, regardless of which VSL and VSHLY variables are utilised. This finding is important as it provides a justification for earlier claims in the thesis that the precise value of the VSL was not paramount because of the nature of the VSL and also the magnitude of improvements in health. This is highlighted by Table 8.1.10, where there is variance in the value of the QALE gain depending on the VSL and VSHLY, but this variance is not great enough to contradict any of the other QALE gain results in the thesis (based on different VSL and VSHLY values).

In order to fully justify the above claims that the values of the VSL and VSHLY will not affect the overall findings of the QALE methodology, it is desirable to briefly consider the QALE gain value that would be yielded according to the VSL elasticities argued by Costa versus Viscusi. Chapter 3: Table 3.4 has highlighted the divergence in opinion about the elasticity of the VSL and subsequent change in the VSL over the twentieth century. The table below summarises the estimate of an alternative VSL and subsequent QALE gain for Costa (who claims a VSL income elasticity of approximately 1.6) versus Viscusi (who claims that the VSL is income inelastic over time in the region of 0.6) versus the mid (unitary elasticity) VSL that has been used in the thesis (derived from Miller’s estimates).

Table 8.1.11: VSL value and subsequent mortality and QALE gain for thesis diseases when assuming different levels of VSL income elasticity: Costa versus Viscusi, 1900-2000 (millions of 1990 international $)[693]

Study	**VSL income elasticity**	**1900-2000 VSL value (millions)**	**Mortality Gain**	**QALE Gain Breast cancer**	**QALE Gain Stomach cancer**	**QALE Gain Tuberculosis**
Costa & Kahn[694]	1.6	0.64	10014	14226	13056	29104
Viscusi & Aldy[695]	0.6	1.18	18675	26530	24285	54275
Thesis (Miller)[696]	1	0.88	13769	19575	17952	40082

Table 8.1.11 is considering the QALE gain that is yielded through including alternative VSL and VSHLY weights. This process is the same as what has been achieved above in Table 8.1.10 for 'Low' and 'High' VSL and VSHLY weights (relative to the thesis 'Mid' estimate, derived from Miller, which is shown in Tables 8.1.7, 8.1.10 and here in Table 8.1.11). Hence, the QALE gain yielded here includes a mortality gain and morbidity gain that have been calculated in the same way as above. The difference is the weight of the mortality and morbidity change valuation variables (VSL and VSHLY). In Table 8.1.11 the VSL and VSHLY have been chosen in accordance with Costa and Viscusi (whereas in Table 8.1.10 the alternative valuation weights [VSL and VSHLY] were selected in accordance with Miller's estimates of a 'Low' and 'High' VSLs).

The results in Table 8.1.11 are not surprising when the mortality and QALE gains are considered as a function of the VSL (and corresponding VSHLY, shown in Appendix 12.7.3). Viscusi's results are consistently higher than the thesis (Miller) and Costa results, respectively. This is because the Viscusi VSL (and subsequent VSHLY [= VSL * QALY]) is higher than the thesis and Costa VSLs. Moreover, the magnitude of difference between these QALE gains is also not surprising, as they reflect the difference in magnitude between the VSLs.

[693] See Appendix 12.7.3 for more detailed calculations
[694] Costa & Kahn, "Changes in the Value of a Statistical Life, 1940-1980", p. 1
[695] Viscusi & Aldy, "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout The World", p. 44
[696] The thesis estimates are based on Miller, where unitary elasticity is implied

Results: Quantitative: Disease

The most noteworthy and important observations, for the thesis, are identified when Table 8.1.11 is compared to Table 8.1.10. Both of these tables provide VSL and VSHLY sensitivity analyses (although the rationale is different). The VSL and VSHLY weight variance is more pronounced for the VSL weights in Table 8.1.11 (this can be highlighted by comparing the VSL weights in Table 8.1.11 with those in Table 8.1.10 and subsequently the QALE gain result range is greater in Table 8.1.11. For example, once again, considering breast cancer for 1900-2000, the (underlying) VSL range in Table 8.1.10 is 0.70 – 1.06 and VSHLY is 0.41 – 0.62 compared to the (underlying) VSL range in Table 8.1.11 is 0.64 – 1.18 and VSHLY is 0.37 – 0.69 in Table 8.1.11. The result is that the range of the QALE gain estimates (for different VSL and VSHLY weights) is broader in Table 8.1.11. In Table 8.1.10 the QALE gain for breast cancer 1900-2000 ranges from 15,505 ('Low' VSL and 'Low' VSHLY) to 23,642 ('High' VSL and 'High' VSHLY) compared to the greater range in Table 8.1.11 of 14,226 (Costa) to 26,530 (Viscusi) in Table 8.1.11.

The most remarkable feature about the above observation is that, although the results in Table 8.1.11 provide a broader range of QALE gain estimates, they still accord with the results in Table 8.1.10 and the general QALE gain conclusions throughout the thesis. Moreover, because of the evident variance in the literature (highlighted by Costa versus Viscusi estimates in Table 8.1.11), it is possible to argue that the thesis VSL selection provides a respectable mid-point between the variance in the literature and a representative estimate.

Therefore, the consistent trend of the above results is that the higher the value of the VSL and subsequent VSHLY, the greater the mortality gain and morbidity gain and ultimate QALE gain (as this is the sum of the mortality and morbidity gain).

Finally, in Table 8.1.10 (and Table 8.1.11) there is a fundamental, clear and consistent trend which is valuable in the context of the thesis because it confirms that, whatever weight (within the thesis' broad range) is adopted for the VSL (and subsequent VSHLY), the QALE gain is still substantial. Hence, the overall conclusions of the thesis remain unchanged and the only alteration is the magnitude of the QALE gain, which always contributes to the same conclusion about the value of twentieth century improvements in health.

8.1.4.3 VSL and VSHLY and QALY Weights

To complete the thesis sensitivity analyses it is desirable to combine the above two versions of sensitivity analysis and consider one aggregate series of different results for considering the full range of values ('Low', 'Mid, 'High') for all methodological variables (QALY, VSL, VSHLY). Once this has been achieved all variables of the methodology will have been considered for all their possible ranges and combinations of ranges and subsequently the QALE will yield the broadest series of estimates possible (within the thesis sensitivity analysis), in order to provide the broadest indication about the value of improvements in health.

With reference to the flow chart example at the beginning of Chapter 8, this represents the combination of stages 11a, 11b and 11c. Also, as was the case in the QALY sensitivity analysis and the VSL and VSHLY sensitivity analysis, stages 8, 9 and 10 will also be utilised as the QALE will be recalculated for alternative QALY, VSL and VSHLY weights. In an effort to simplify the mass of possible results (provided in appendices 12.7.1 and 13.2.1) the table below will provide a summary about the 'Mid' and most extreme results: 'Low' QALY, VSL, VSHLY and 'High' QALY, VSL, VSHLY.

Table 8.1.12: QALE gains for thesis diseases: breast cancer (BC), stomach cancer (SC), tuberculosis (TB) with alternative QALY, VSL and VSHLY values (millions of 1990 international $)[697]

Period	**Low VSL, Low VSHLY, Low QALY**			**Mid VSL, Mid VSHLY, Mid QALY**			**High VSL, High VSHLY, High QALY**		
	QALE Gain SC	QALE Gain BC	QALE Gain TB	QALE Gain SC	QALE Gain BC	QALE Gain TB	QALE Gain SC	QALE Gain BC	QALE Gain TB
1900-1925	3204	3013	6281	4173	3699	10671	5224	4353	16053
1925-1950	2857	2510	7389	3591	2857	11686	4350	3113	16781
1950-1975	4298	7355	9318	6640	12090	14466	9392	17789	20630
1975-2000	8487	16765	6690	11688	24990	8616	15154	34675	10492
1900-2000	13107	14198	25793	17952	19575	40082	23327	25645	57444

[697] Table 8.1.12 is based on the same type of calculations as Table 8.1.7 and 8.1.9 and 8.1.10. See Appendix 12.1.1 for VSL and VSHLY range of values to be included in sensitivity analyses and Appendix 12.6.1 for range of QALY values and Appendix 12.7.1 and 13.2.1 for calculations.

Table 8.1.12, consider the most extreme (highest and lowest) and mid point combinations of the VSL, VSHLY and QALY (for the thesis') sensitivity analysis weights, which was achieved through recalculating the change in the morbidity burden using a lower and higher QALYs (than the mid that was used in the initial part of Chapter 8.1), and then valued, to calculate the morbidity gain, through using a lower and higher VSHLY (comprised of all combinations of a lower and higher VSLs and QALYs, as VSL * QALY = VSHLY). The same considerations were applied to mortality, through recalculating the value of the change in the mortality burden with alternative lower and higher VSL values. This exercise has created a range of QALE gain values, which are a function of the variable weights (i.e. low or high QALY, VSL and VSHLY).

The results from this exercise (in Table 8.1.12) show a general trend, which is expected: as the value of the VSL, VSHLY or QALY weight increases the value of the QALE gain also increases. This result is logical as the more value is attributed to the change in the mortality and morbidity burden, the higher the QALE gain will be.

The level of variance between the results in Table 8.1.12 is greater than for previous QALE gain calculations in the thesis (shown in Table 8.1.6 for the 'Mid' QALE gain and in Table 8.1.8 for the QALE gain with alternative QALYs and in Table 8.1.10 for the QALE gain with alternative VSL and VSHLY weights). This result is what would be expected, because the thesis is considering different weights for a greater number of variables in Table 8.1.12, than in any previous table that is using the alternative thesis variable weights (i.e. Table 8.1.11, which considers alternative author weights is not included in this observation).

The most noteworthy observations, particularly from the perspective of the thesis, is that, regardless of whatever QALYs, VSLs and VSHLYs are utilised, when the twentieth century is considered in its entirety all illnesses provide a valuable contribution to mortality gains, and the overall QALE estimate, which reinforces the value of improved health and substantiates the robustness of the thesis conclusions.

8.1.4.4 Age Weights

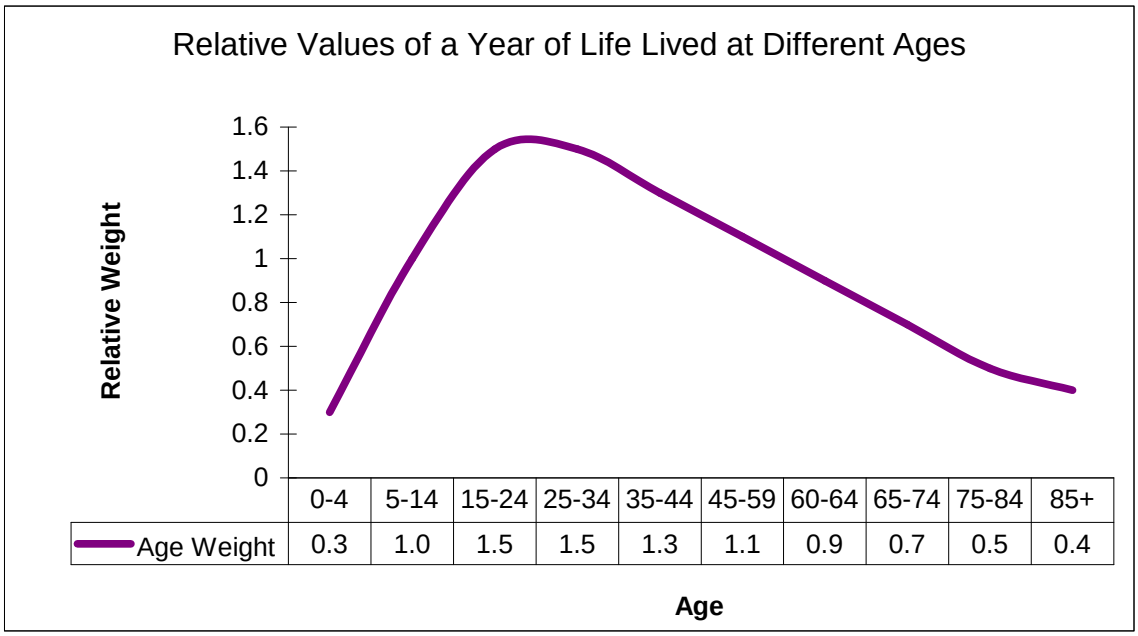
A final process of sensitivity analyses is to consider claims that the value of mortality and morbidity improvements are not constant across all age groups. The rationale and theories surrounding this have been covered in detail in Chapter 3. Figure 8.1.2 (below) illustrates

the weights that will be applied to the QALE gain established above. This calculation represents stage 11d in the flow chart example at the beginning of Chapter 8.

The age weighting function (shown in Figure 8.1.2) will only be applied to the central or mid QALE gain results. This is desirable because it provides an indication of the effect of considering age weighting for the QALE gain without an excess of data that would confuse the conclusions of this exercise. For the more comprehensive reader these calculations have been made for every combination of the QALE gain (i.e. the age weighting function has been applied to all of the sensitivity analyses, above) in appendices 12.8 to 12.10 and 13.3 to 13.5. Furthermore, the summary twentieth century QALE gain results (presented in Chapter 8.3) provides an age weighted equivalent for all QALE gain variable weight combinations for 1900-2000.

The procedure here is to recalculate the (central) QALE gain results of the thesis (shown in Table 8.1.7) with the age weighting function shown in Figure 8.1.2. I.e. so far the QALE gain results have valued mortality and morbidity gains as equal across all age groups of the population, whereas the age weighting function is accounting for differences in the value of morbidity and mortality gains at different ages. E.g. in Figure 8.1.2 a life year at ages 0-4 are only worth 30 percent of a life year versus ages 15-34, which are worth 1.5 times a life years. Therefore, in simplistic terms, the QALE gain would need to be reduced by 70 percent for ages 0-4 and inflated by 50 percent for ages 15-34, and so on.

Figure 8.1.2: Murray's age weight function of DALYs: relative value of a year of life lived at different ages[698]



The application of such a weighting is very straightforward because the weights provided by Murray (1996) are clear and versatile. Moreover, because the thesis has consistently considered the morbidity and mortality gain for the age distribution of the population, Murray's age weighting can be directly applied to the existing data and this will document a reduction or increase in the (mortality gain, morbidity gain and subsequent) QALE gain of the different age groups, by the magnitude outlined in Figure 8.1.2.

[698] Murray, "Rethinking DALYs", in Murray & Lopez, "The Global Burden of Disease", p. 60

Table 8.1.13: Age weighted QALE gains (using mid value QALY, VSL, VSHLY variables) for thesis diseases: breast cancer, stomach cancer, tuberculosis (millions of 1990 international $)[699].

Period	Mortality Gain	Morbidity Gain: Stomach cancer	QALE Gain Stomach cancer	Morbidity Gain: Breast cancer	QALE Gain Breast cancer	Morbidity Gain: Tuberculosis	QALE Gain Tuberculosis
1900-1925	2710	244	2954	-52	2658	1687	4397
1925-1950	2943	84	3027	-738	2205	9633	12576
1950-1975	1415	3145	4560	7854	9269	13540	14955
1975-2000	4862	2554	7416	14298	19160	596	5458
1900-2000	9402	3368	12770	5627	15029	19538	28940

This table provides a summary of the age-weighted QALE gain for the mid QALY, VSL and VSHLY sensitivity analysis variables. I.e. this is essentially the same calculation as in Table 8.1.7 but it is then applied to the age-weighting function illustrated in Figure 8.1.2 to generate the results shown in Table 8.1.13.

Table 8.1.13 relays generally consistent result about the effect of applying an age weighting function to the mortality gain, morbidity gain and QALE gain. All of these observations are derived from comparing Table 8.1.7 with Table 8.1.13. The change in the mortality gain when applied to the age weighting function is an effect which consistently (and relatively significantly) reduced the value of the mortality gain.

[699] Table 8.1.13 is considering the QALE gain, which is the sum of the morbidity gain (Table 8.1.3, 8.1.4, 8.1.5) and the mortality gain (Table 8.1.6), which has been conducted for individual illnesses here. Table 8.1.13 is making the same considerations as Table 8.1.7 but applied to Murray's age weighting function (shown in Figure 8.1.2). See Appendix 12.8, 12.9, 12.10.1, 13.3, 13.4, 13.5 for detailed calculations.

The change in the morbidity gain when applied to the age weighting function is similar to mortality but there are some important exceptions. First is that in five instances in Table 8.1.13 applying the age weighting function created an increase in the value of the morbidity gain compared to the un-weighted equivalent in Table 8.1.7: breast cancer in 1900-1925 and 1925-1950, stomach cancer in 1925-1950, tuberculosis in 1925-1950 and 1950-1975. The episodes related to breast and stomach cancer are quite clearly a result of the morbidity gain being negative (i.e. a change in the morbidity burden that was negative) and therefore when the age weighting function is applied the negative change in the morbidity burden is reduced (as the age weighting function generally reduces the magnitude, as has been outlined above) which creates a more valuable (albeit still negative in most instances morbidity gain). This seemingly perverse effect for tuberculosis between 1925 and 1975 is more ambiguous. It seems most likely that this could be a result of a significant decline in tuberculosis in the 1920s that was much more pronounced for middle/working ages (i.e. approximately ages 20-44, this is illustrated in Chapter 5: Figure 5.6 and 5.8) which receive a greater age weight than all other ages in Murray's age weighting function. This trend was also evident, although to a much lesser extent around the 1960s (illustrated in Chapter 5: Figure 5.8).

Finally, the change in the QALE gain when applying to the age weighting function is also similar to mortality, with only one important exception, which was the continuation of the tuberculosis phenomenon between 1925 and 1975.

A final important observation of the effect of applying an age-weighting function to the thesis QALE gain is the difference in the magnitude this effect has upon the mortality and morbidity gain. Precisely, the change in the morbidity gain for breast and stomach cancer is smaller than for mortality. This result implies that there have been greater improvements in middle aged morbidity associated with cancer than for tuberculosis and mortality, where the most improvements have been at the youngest and oldest ages, which receive a lower value when the age weighting function is applied.

This observation is very important for the thesis as it adds additional weight to the arguments of the thesis concerning the need to include morbidity in health measures. Hence, a comparison of the mortality and morbidity gains in Table 8.1.7 (un-weighted) and Table 8.1.13 (age-weighted) indicates clear improvements in health that are not evident for

mortality (namely an improvements in the age burden of disease) and therefore these subtle morbidity gains need to be represented.

These findings complete the sensitivity analyses of the thesis through reiterating that regardless what values are chosen (within the generous range of thesis variable weights) the QALE gain consistently generates results that confirm the conclusions of the thesis. I.e. the value of health improvements and the subsequent need to measure health is highlighted by all of the above results, regardless of the variable weights. This finding will be reinforced throughout the remainder of Part III of the thesis, when these results are considered in an extended measure of economic development, i.e. for their contribution to economic development (as an appendage to GDP per capita growth, below) and also (in Chapter 9) when the limited number of illnesses analysed in Chapter 8 will be extrapolated forward in order to provide a lowest bound estimate about the value of all twentieth century morbidity (and existing aggregate mortality) improvements.

8.1.5 Value of QALE Gains Relative to GDP

To bring greater significance to the above findings it is possible to consider the QALE gain in conjunction with GDP growth. This exercise is also important because it will highlight the authenticity of the thesis claims to consider wider notions of economic development that include health, e.g. through appending some form of health statistic to national income measures to account for improvements in health and standards of living. Hence, as well as standardising the above QALE gains with economic growth the information below will provide a more accurate account of aggregate twentieth century economic welfare growth.

This analysis will utilise the age-weighted ‘Mid’ or central QALE gain results that have been derived above, in Table 8.1.13, because these are deemed to be the most accurate. Therefore, the mortality, morbidity and QALE gain results presented in Table 8.1.13 will be used here to calculate the compound average rate of growth for each of these variables (see Table 8.1.14) so that the difference in value of growth can be identified between: mortality, morbidity, QALE and GDP. This will enable the two key benefits of this section to first to highlight the extensiveness of health developments relative to GDP, and second, emphasise the demands of the thesis about the need to include health measures in some form of extended national income.

After this has been achieve the QALE growth will be considered in conjunction with GDP growth in order to present a single, rounded growth estimate, where the value of the QALE gains will be added to the value of GDP for the era under consideration and the combined compound average growth rate will be calculated (see Table 8.1.15) in order to provide a first approximation about what the extended national income would have been for twentieth century England.

Table 8.1.14: Compound average growth rates of: GDP per capita, mortality gain, morbidity gain and QALE gain (using mid value QALY, VSL, VSHLY variables and age weighting) for thesis diseases: breast cancer, stomach cancer, tuberculosis (percentage per annum)[700]

Period	**GDP pc Growth**	**Mortality Gain Growth**	**Morbidity Gain Growth**			**QALE Gain Growth**		
			Stomach cancer	**Breast cancer**	**Tuberculosis**	**Stomach cancer**	**Breast cancer**	**Tuberculosis**
1900-1925	0.3	2.2	0.2	0	1.4	2.4	2.1	3.5
1925-1950	1.4	2.0	0.1	-0.4	6.5	2.0	1.4	8.4
1950-1975	2.2	0.7	1.4	3.5	6.0	2.0	4.1	6.4
1975-2000	1.8	1.3	0.7	3.6	0.2	1.8	4.7	1.4
1900-2000	1.4	1.4	0.5	0.8	2.8	1.9	2.2	4.2

Table 8.1.14 considers the compound average growth rate of GDP per capita, mortality, morbidity (by disease) and QALE (by disease). This is achieved through applying the most credible mortality gain, morbidity gain and QALE gain estimates (which are deemed by the author to be the age-weighted mid values presented in Table 8.1.13) to the compounding formula, in order to generate an estimate about average growth per annum for the twentieth century.

Once this has been achieved it is possible to consider the relative value of mortality, morbidity and QALE gains versus GDP gains. This consideration reiterates the need to consider both mortality and morbidity: given the magnitude of average annual growth of mortality gains and morbidity gains. For example, in Table 8.1.14, mortality gain growth was approximately as much as GDP growth during the entire twentieth century, and in the earlier years (1900-1950) mortality growth gains were greater than GDP growth gains.

[700] See Appendix 12.11 for GDP per capita compound average growth rate calculations and see Appendix 12.12.1 and 13.6.1 for mortality and morbidity gain growth calculations.

Table 8.1.14 also highlights the value of morbidity gains relative to mortality and GDP gains, although the relationship is not as consistent as the contribution of mortality to GDP. Tuberculosis experienced the greatest morbidity gain growth for the twentieth century in general, which was exceptionally pronounced between 1925 and 1975, when tuberculosis morbidity gains were contributing more to welfare growth than mortality or GDP. Between 1950 and 1975 breast cancer also experienced important gains which were more pronounced than mortality and GDP growth.

A final crucial point that is reflected in Table 8.1.14 is the magnitude of mortality and morbidity gains (and especially the combination of these facets embodied in the QALE gain) in nearly every single era of the twentieth century relative to GDP growth. This reinforces the need to consider health in some form of extended GDP measure (which will be reiterated below) and also the need to ensure that morbidity (as well as mortality) is included in health measures.

Table 8.1.15 (below) aggregates the results of Table 8.1.4 in order to provide an indication of what a more rounded welfare national income would represent. This is achieved through imputing GDP per capita growth with QALE growth (both shown independently in Table 8.1.14), in order to create a series of ‘Adjusted Growth’ estimates, which essentially represent national income growth adjusted for twentieth century gains in QALE.

Results: Quantitative: Disease

Table 8.1.15: Compound average growth rates of GDP per capita growth adjusted for QALE gain (using mid value QALY, VSL, VSHLY variables and age weighting) for thesis diseases: breast cancer, stomach cancer, tuberculosis (percentage per annum)[701]

Period	**GDP pc Growth**	**QALE Gain Growth**			**'Adjusted Growth' (QALE + GDP pc)**		
		Stomach cancer	**Breast cancer**	**Tuberculosis**	**Stomach cancer**	**Breast cancer**	**Tuberculosis**
1900-1925	0.3	2.4	2.1	3.5	2.7	2.4	3.8
1925-1950	1.4	2.0	1.4	8.4	3.4	2.8	9.8
1950-1975	2.2	2.0	4.1	6.4	4.2	6.3	8.6
1975-2000	1.8	1.8	4.7	1.4	3.6	6.5	3.2
1900-2000	1.4	1.9	2.2	4.2	3.3	3.6	5.6

Table 8.1.15 considers GDP per capita growth per annum and QALE gain growth per annum and (these two indices are summed in order to estimate) 'Adjusted Growth', which represents what GDP per capita per annum growth would be if it was extended to include gains in health or QALE. This exercise provides 'Adjusted Growth' levels that are very noteworthy, as they highlight the significance of overall health improvements to the value of historical economic development. Hence, the findings in Table 8.1.15 justify the need to consider health or QALE gains in an extended form of GDP.

Therefore, the above tables suggest that taking account of QALE gains creates a much more favourable indication of the improvements in economic growth and particularly living standards in twentieth century England. The results highlight the need to rethink the way in which national income and health are considered and computed and specifically the need to provide national income data that incorporates utility and health measures that include morbidity.

These findings will be reinforced in the following chapter when similar considerations and calculations are made for the thesis' disability, blindness. This data provides the first genuine quantitative indication about the value of improvements in disability (to match those made above for disease) for twentieth century England.

[701] See Appendix 12.11 for GDP per capita compound average growth rate calculations and see Appendix 12.12.1 and 13.6.1 for QALE gain growth calculations.

8.2 Disability (Blindness)

8.2.1 QALY Values and Prevalence Estimates

The qualitative account of blindness in twentieth century England has highlighted that there were improvements in the overall quality of life associated with being blind, particularly during the first half of the twentieth century. The process below will derive a quantitative estimate for the value of these improvements. This will be achieved in a very similar process to the methodology that has been outlined above (in Chapter 8 and 8.1). All stages of the flow chart example apply to the calculation of the QALE gain for blindness (in a very similar way as it was calculated for diseases above).

The key benefit of this exercise is to provide an indication of the value of improvements in quality of life associated with disability that can be combined with the QALE gain for disease. This will also provide results which are necessary so that the findings about disability can be represented in the 'Extended Results' (Chapter 9) of the thesis.

The first stage of this quantitative analysis for disability is to calculate the prevalence and summarise the quality of life (QALY) associated with blindness for the eras considered in the thesis. In accordance with the thesis methodology outlined in the flow chart example, this represents stage 5 (identifying the QALY from EuroQol) and also part of stage 9 (compiling illness data). Once both of these data sets have been identified it will be possible to calculate the morbidity burden (prevalence * QALY), which provides the foundation for stage 9 and eventually identifying the morbidity gain and subsequent (when applied to the mortality gain identified in Chapter 8.1) QALE gain. These primary calculations are summarised below in Table 8.2.1 (prevalence) and 8.2.2 (QALY).

Table 8.2.1: Number of blind persons and average number of blind years and corresponding prevalence of blindness in 1900, 1925, 1950, 1975 and 2000[702]

Year	Number of blind persons	Average number of years of blindness	Total blind years	Blind years per annum / Prevalence
1900	10069	23	299747	11990
	8520	8		
1925	10956	19	371244	14850
	18120	9		
1950	10572	68	1366204	54648
	19517	24		
	17890	10		
1975	24900	26	1131447	45258
	53783	9		
2000	108896	11	1197856	47914

Estimating the prevalence of blindness in the economy is more complicated than identifying disease prevalence (which was achieved above in Chapter 8.1). This is largely because of the nature of disabilities (more protracted and ambiguous in duration and cure) and the subsequent way in which disability data is collected and recorded (without any detail or follow-up). Therefore, an alternative approach to estimating the prevalence of blindness is adopted here: the prevalence data above is derived from considering the number of people who are blind and the average number of years that they spent in blindness (which is equivalent to the number of years of life expectancy at the age of onset of blindness). This is necessary as there are no official estimates of the prevalence of blindness.

In Table 8.2.1 prevalence is estimated with two (three in 1950) entries for the number of blind persons and years because there were generally two (three in 1950) most common ages of blindness, which were considered in the estimation of the prevalence of blindness. A detailed explanation of the calculations and results of this unique disability prevalence estimating process is provided in Chapter 4: Table 4.9 and 4.10.

[702] In Table 8.2.1 the 'Number of blind': those registered as blind, 'Average number of blind years': estimated from the age distribution and profile of blindness in the relevant era, for an elaboration and base calculations see Chapter 4: Tables 4.9 and 4.10 and Appendix 12.13.1, 'Total blind years': multiplying the two previous columns and 'Blind years per annum': 'Total blind years'/25 to achiever per annum estimates. Note: these are period estimates, which provide a snapshot of prevalence for the year stated in column 1 of Table 8.2.1.

Results: Quantitative: Disability

A noteworthy feature associated with the prevalence of blindness calculated in Table 8.2.1 is the improvement in the approximate average number of years of blindness during the twentieth century, which declined from nearly 16 years in 1900 and a peak of 34 years in 1950 to about 11 years of blindness by 2000 (calculated by averaging the 'average number of years of blindness' entries in Table 8.2.1 for each year). Although this is only an estimate, because of the problems associated with identifying blind prevalence by age, the results are still important. In contrast to the average number of blind years the 'prevalence' of blindness (used in the thesis) has increased from approximately 11,990 blind years per annum in 1900 to 47,914 in 2000 (shown in Table 8.2.1)[703].

The next key variable, which is also considered consistently across the thesis (although contains slightly different facets for disability and disease) is the quality of these blind life years, QALY[704]. The QALY is the variable that represents the burden of illness through considering its consequence upon a healthy life year, such that the QALY is the fraction (greater than 0 which approximates death) of 1, which represents a full healthy life year. These results about the QALY for blindness during different eras of the twentieth century are shown in Table 8.2.2. In the same way as for diseases, the blind QALY has been derived in Chapter 7 (through EuroQol). Table 8.2.2 also reports 1-QALY, which is the index needed for calculating the morbidity burden, because of the way in which the QALY is considered in the QALE methodology (this has been explained in more detail above in Chapter 8.1: Section 8.1.1).

Table 8.2.2: QALY values for thesis disability: blindness[705]

Year	**QALY**	**(1-QALY)**
1900	0.3333	0.6667
1925	0.5000	0.5000
1950	0.6667	0.3333
1975	0.6667	0.3333
2000	0.6667	0.3333

Table 8.2.2 highlights that the quality of life associated with blindness improved between 1900 and 1950, and that despite marginal improvements in the quality of life during the

[703] It should be noted that the prevalence statistic calculated here (Table 8.2.1) differs from the 'prevalence of blindness in twentieth century England' statistic provided earlier in the thesis (see Figure 4.1 in Chapter 4). The reason for the difference is that the prevalence (here, in Chapter 8.2) considers 'blind years' and not just the number of blind who returned an authentic BD8 form, in order to register as such.
[704] See Chapter 3: Subsection 3.5.1: *Key Variables* for an elaboration
[705] See Appendix 12.6.2 for blind QALY calculations.

second half of the twentieth century (outlined in Chapter 4 and summarised in Chapter 7.1); none of these were far reaching enough to improve blind persons' quality of life into a more superior (EuroQol [in Chapter 7.1] and subsequent) QALY between 1950 and 2000.

The impact of the change in the prevalence (outlined above in Table 8.2.1) and the QALY (shown in Table 8.2.2) will be combined in order to calculate the morbidity burden (prevalence * QALY) and identify the change in the morbidity burden over time, which can subsequently be valued (using the VSHLY) in order to estimate the morbidity gain. This methodological process is the same as the calculation of the disease morbidity gain in Chapter 8.1, except that for disability (blind) the morbidity burden (and subsequent gain) will be calculated in aggregate and not by age, for the age distributional profile of the population as was achieved for diseases in Chapter 8.1 because the data to make this more sophisticated calculation does not exist for blindness.

8.2.2 Morbidity Gain

As in Chapter 8.1 the morbidity gain highlights the value of improved morbidity facilitated by an improvement in the burden of diseases or disability morbidity. An improvement in the morbidity burden can be generated by an improvement in the QALY and/or the prevalence.

In order to highlight the morbidity gain it is necessary to first document the change in the morbidity burden of blindness, which is achieved in Table 8.2.3. (After the change in morbidity burden has been estimated it will be possible to combine this with the associated VSHLY and calculate the morbidity gain).

Table 8.2.3: Calculation of morbidity burden of thesis disability: blindness

Year	**Prevalence**	**(1-QALY)**	**Morbidity burden**
1900	11990	0.6667	7994
1925	14850	0.5000	7425
1950	54648	0.3333	18214
1975	45258	0.3333	15084
2000	47914	0.3333	15970

Results: Quantitative: Disability

Table 8.2.3 considers the number of blind years 'prevalence' (derived from Table 8.2.1) and the associated quality of these years 'QALY' (Table 8.2.2) in order to determine the morbidity burden (prevalence * 1-QALY[706]) of blindness. This calculation is the equivalent of the more detailed process outlined in Chapter 8.1 (Tables 8.1.2.i and 8.1.2.ii) but different because the blind morbidity burden is calculated aggregately with no accounting for the changing age profile structure of the population. Although the results in Table 8.2.3 represent a more approximate estimate of the morbidity gain (versus those identified for diseases in Chapter 8.1) the results are still valuable and indicative.

Table 8.2.3 highlights that there were improvements in the burden of blindness during the twentieth century, although this was not linear, due to the worsening around the middle of the twentieth century and an increase in the prevalence of blindness at the end of the century (which was a result of an increase in the number of blind as the average duration of blindness declined over the twentieth century [shown in column 3 of Table 8.2.1]). This set back in the decline in the prevalence of blindness in 1950 was a result of (accidental) adverse medical intervention (for the administration of oxygen to premature babies), which caused a condition of blindness known as Retrolental Fibroplasia and a subsequent increase in the number of blind at birth. The result of this adverse medical intervention was a worsening in period estimates about the burden of blindness for 1950 (shown in the table above) but was not significant enough (as the effects of such were identified and largely rectified by the 1960s) to show up in the next cohorts estimates[707]. Although, it should be noted that part of the morbidity burden decline between 1950 and 1975 is excessively high due to this increase in the prevalence of blindness in 1950. This occurrence is also noteworthy as it indicates that medical progress has not always been linear during the twentieth century, an important historical detail that is not illustrated by the other morbidity states in the thesis.

The table below provides the next stage of the disability quantitative analysis which is considering the above blind morbidity burden change in conjunction with a value (presented as the VSHLY) of this decline in order to estimate the morbidity gain.

[706] 1-QALY is utilised in the morbidity burden calculation in Table 8.2.3 in accordance with the QALE methodology (this is explained briefly above Table 8.2.3 and in more detail in Chapter 8.1).
[707] Silverman, "Retrolental Fibroplasias: A Modern Parable", Chapters 3, 8, 9, 11

Table 8.2.4: Morbidity gain (morbidity burden change*VSHLY): monetary value of improvements in the burden of morbidity: blindness (millions of 1990 international $)[708]

Period	**Morbidity burden change**	**VSHLY**	**Blind morbidity gain**
1900-1925	569	0.27	154
1925-1950	-10789	0.45	-4855
1950-1975	3130	0.77	2410
1975-2000	-886	1.36	-1205
1900-2000	-7976	0.44	-3509

Table 8.2.4 illustrates the process of calculating the morbidity gain. The first stage of this calculation is to identify the change in the morbidity burden, which is achieved through considering the change between T1 and T2 (shown in the final column of Table 8.2.3) For example, the 1900-1925 morbidity burden change = morbidity burden in T1 (1900 in this example) minus the morbidity burden in T2 (1925 in this example), i.e. 7,994 - 7,425 = 569.

The next stage of the calculation in Table 8.2.4 is to value the change in the morbidity burden, which is yielded by applying the VSHLY to the morbidity burden change (morbidity burden change * VSHLY = morbidity gain). The VSHLY is estimated by considering the VSL and the QALY (VSL*QALY), in the same way as was outlined in detail in Chapter 8.1 (and is also outlined in the flow chart example [stage 7] at the beginning of Chapter 8). For example, continuing with the period 1900-1925, where the identified morbidity change was 569, this is then applied to the VSHLY for blindness in the period 1900-1925 (which is yielded by multiplying the VSL with the QALY for the period 1900-1925 [shown to be 0.4167 in Table 8.3] → 0.64*0.4167 = 0.27 [millions of 1990 international $]). Once the VSHLY has been calculated it is then applied to the change in the morbidity burden in order to estimate the morbidity gain. In the example of blindness between 1900 and 1925, the morbidity gain = the change in the morbidity burden*VSHLY (569*0.27=154), which results in a morbidity gain of 154 (million 1990 international $) for blindness between 1900 and 1925.

[708] See Appendix 12.7.2 (mid estimates) for base calculations underlying the results in Table 8.2.4.

Results: Quantitative: Disability

The above table highlights both positive and negative trends in the morbidity gain. The negative morbidity gains in 1925-50 and 1975-2000 and 1900-2000 are as a potential result of a worsening in the burden (prevalence and/or QALY) of blindness. More precisely a worsening in the prevalence of blindness and not the QALY as this improved in the period 1925-1950 and especially in the period 1900-2000 and was maintained between 1950 and 2000, as Table 8.2.2 highlights.

Therefore, it should be noted that the worsening in the morbidity gain in 1925-1950, 1975-2000 and 1900-2000 is entirely a result of increased prevalence of blindness and despite the extensiveness of this increase it is partially compensated by improvements in the QALY (and also, for the periods 1975-2000 and 1900-2000 it is largely a function of an increase in life expectancy). I.e. if there were no QALY improvements for blind sufferers during the twentieth century and the same substantial increase in blind prevalence then the morbidity gain (or loss as is the case) would have been more than twice as unfavourable (-8780 instead of -3059 (derived through conducting the same calculation in Table 8.2.3 and 8.2.4 but holding the QALY constant at the 1900 level[709]). This dramatises the value of improvements in the quality of life (QALY).

Additionally, if there were no improvements in life expectancy between 1900 and 2000[710] then the prevalence of blindness would have been only 26,135 in 2000 and the subsequent burden of blindness (prevalence [26135] * QALY [0.3333]) would have been 8,711 in 2000, which is only marginally greater than the 1900 burden of 7,994, which would mean that the blind morbidity gain (loss in this case) would be -315 instead of -3509 (the level that is largely a result of increased life expectancy).

Following the same methodological process outlined in the flow chart example and also adhered to in Chapter 8.1, the next stage in estimating the QALE for blindness is to identify the mortality gain (so that this can be summed with the morbidity gain [presented above for blindness in Table 8.2.4] in order to estimate the QALE gain).

[709] Calculation: 47914*0.6667=31944 → 4937-31944=-19954 → -19954*0.44=-8780

[710] Considering life expectancy at 75 for 1900 and 2000. Life expectancy by age data: Figures based on the 'Office of National Statistics/Government Actuaries Department' England and Wales mortality database. This is still subject to revisions. Extract was provided by Mita Saha (Office of National Statistics) on March 17 2006. See Appendix 12.15 for a copy of exert.

8.2.3 Mortality Gain

The mortality gain results utilised here are the same as those that were yielded above in Chapter 8.1 (Table 8.1.6). To recap: the first step in identifying the mortality gain is to identify the change in the death rate or mortality burden. This is established through considering the change in the death rate. Once the change in the mortality burden has been identified it is possible to value this, through applying the VSL to the change (always a decline in the death rate in the twentieth century) in the mortality burden. The result of this calculation (decline in mortality burden * VSL) will yield the mortality gain. These results are shown in the second column of Table 8.2.5. For a more detailed review of the mortality gain, see Chapter 8.1: Section 8.1.2: *Primary Valuation of Improvements in Mortality*.

8.2.4 QALE Gain

Following the same methodological process as in Chapter 8.1, the next stage in estimating the QALE gain is to combine the morbidity gain and the mortality gain. This represents stage 10 in the flow chart example at the beginning of Chapter 8. The QALE gain for blindness is presented below in Table 8.2.5.

Table 8.2.5: QALE gain (morbidity gain + mortality gain): monetary value of improvements in the burden of morbidity and mortality for thesis disability: blindness (millions of 1990 international $)[711]

Period	**Average mortality gain**	**Blind morbidity gain**	**QALE gain Blindness**
1900-1925	3895	154	4049
1925-1950	3598	-4855	-1257
1950-1975	2489	2410	4899
1975-2000	8011	-1205	6806
1900-2000	13769	-3509	10260

Table 8.2.5 presents the calculation and results of the QALE gain for blindness (mortality gain + blind morbidity gain = QALE gain). These blind QALE gain result highlight that in 1925-1950, to a lesser degree in 1975-2000, and in 1900-2000, the morbidity burden associated with blindness worsened and therefore provided a negative contribution to the QALE gain. I.e. the blind morbidity gain was negative and therefore unable to provide extended QALE gains in addition to the mortality gain. This was particularly pronounced

[711] See Appendix 12.7.2 (mid estimates) for base calculations.

for 1925-1950, which was outlined earlier, as the result of Retrolental Fibroplasia. Conversely, in 1900-1925 and 1950-1975 the morbidity burden for blindness improved and was able to provide a positive contribution to the QALE gain. As has been mentioned above, the mortality gain was positive in all the thesis eras of the twentieth century and therefore always provided a positive contribution to the QALE gain.

The relationship between the mortality gain and morbidity gain within the overall QALE gain can be considered in order to provide a more vivid indication of the key contributor to the QALE gain associated with blindness. This comparison is shown below in Figure 8.2.1.

Figure 8.2.1: QALE gain: value and composition of the morbidity gain and mortality gain towards QALE gain for thesis disability: blindness (millions of 1990 international $)[712]

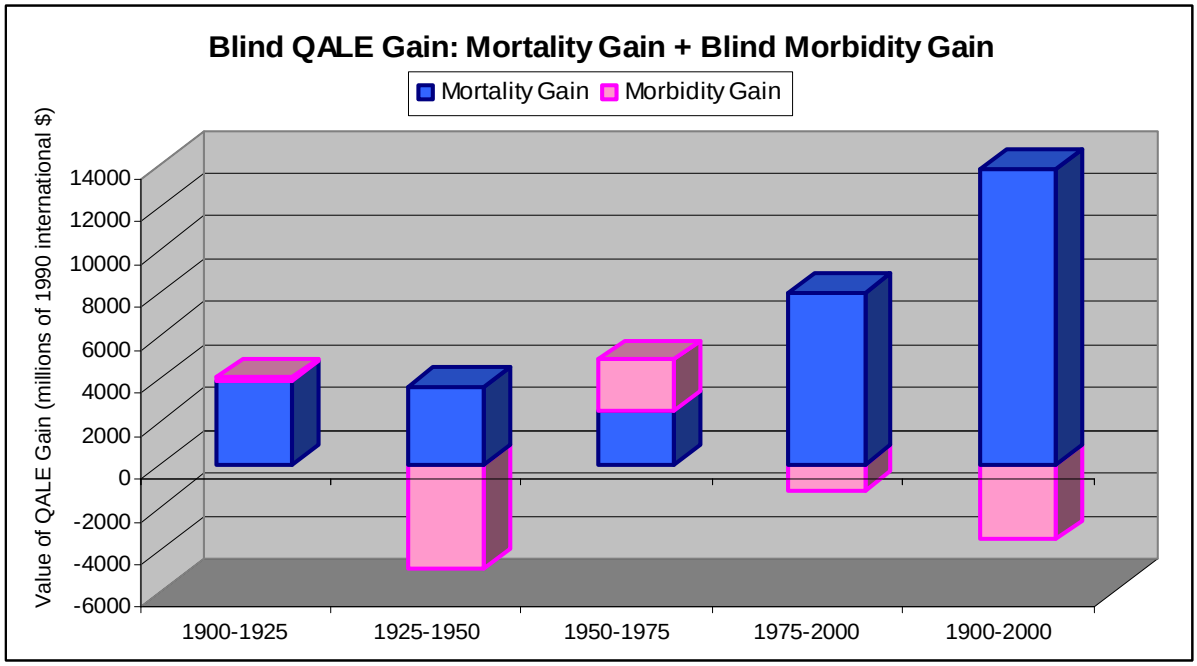


Figure 8.2.1 illustrates the contribution of the mortality gain (blue bar) and the morbidity gain (pink bar) to the overall QALE gain (height of blue bar + pink bar). Furthermore, the placements of the morbidity bars also represent the direction (positive or negative) of the morbidity contribution to the overall QALE gain. If the morbidity gain bar is on top of the mortality gain bar then the morbidity gain has provided a positive contribution to the QALE gain, and if the morbidity bar is below the mortality bar the morbidity gain is negative and has caused a reduction in the QALE gain. This trend was evident in 1925-1950, 1975-2000 and 1900-2000.

[712] See Appendix 12.7.2 for the QALE gain and the composition of the morbidity and mortality gain towards the QALE gain.

Results: Quantitative: Disability

Through considering the magnitude and direction of the morbidity gain relative to the mortality gain, in order to determine the contribution of improved morbidity to the QALE gain, the above figure illustrates a very important finding: in only two instances were there positive contributions of blind morbidity to the blind QALE gain (1900-1925 and 1950-1975 [which was partially a result of the worsening in the blind morbidity gain in the previous era, 1925-1950]). This finding marks a stark contrast to the morbidity gains achieved by the thesis disease morbidity (see Figure 8.1.1 for a direct comparison), where the morbidity gains were usually positive and in certain instances more valuable than the mortality gain. This indicates that, in general, morbidity gains for disabilities were often negative and generally much less valuable than the morbidity gains associated with diseases in twentieth century England. Although, it should be noted that a major driver of blind morbidity increases was prevalence (not the QALY as this improved during the twentieth century) and more specifically, more individual cases of prevalence as the average number of blind years declined per sufferer. These features are not accounted for in the above analysis but should be recognised as they abate a small aspect of the poor performance of the blind morbidity burden in twentieth century England.

The above results and analyses are powerful in highlighting the nature and extensiveness of improvements in blind morbidity. The final exercise below will ensure that the trends above are accurate through applying detailed sensitivity analyses. In keeping with Chapter 8.1 this will contain additional (Low and High) estimates about the VSL, VSHLY and QALY. However, this will not include an age-weighting exercise as the data does not exist to make these considerations. With reference to the flow chart example at the beginning of Chapter 8, the following section will apply the (QALE gain) results from stage 10 (identified above) to stages 11a, 11b, and 11c.

When the same sensitivity analysis stages were applied to the disease QALE gain (in Chapter 8.1 above), each stage (11b then 11a+11b then 11a+11b+11c) was initially conducted separately and then all stages of the QALY, VSL, VSHLY sensitivity analysis were considered in aggregate (see Table 8.1.12). This chapter benefits from this previous gradual and detailed explanation, and will therefore only consider the aggregate sensitivity analysis that comprises all variables: QALY, VSL, VSHLY (i.e. stages 11a, 11b and 11c).

8.2.5 Sensitivity Analyses: QALY and VSL and VSHLY Weights

In this section the sensitivity analysis the morbidity and mortality calculations will be conducted with the extremes (Low and High) and mid-point (Mid) value weights for the methodology variables: QALY, VSL and VSHLY. Once this has been achieved this chapter will yield the broadest possible range of estimates about the value of the QALE gain for blindness. In reference to the flow chart example this process will repeat stages 5 to 10 through applying the sensitivity analysis (stages 11a, 11b, and 11c) alternative weights. The alternative weights that will be used for the blind morbidity gain (QALY and VSHLY) and the mortality gain (VSL) are shown in Table 8.2.6 below.

Table 8.2.6: Alternative (Low and High) and Mid: QALY, VSL, VSHLY weights for QALE gain sensitivity analysis: thesis disability: blindness (millions of 1990 international $)[713]

Year	**QALY**			**VSL**			**VSHLY**		
	Low	Mid	High	Low	Mid	High	Low	Mid	High
1900-1925	0.2500	0.4167	0.5834	0.51	0.64	0.77	0.21	0.27	0.32
1925-1950	0.4167	0.5834	0.7500	0.60	0.77	0.92	0.35	0.45	0.54
1950-1975	0.5000	0.6667	0.8333	0.92	1.16	1.40	0.61	0.77	0.93
1975-2000	0.5000	0.6667	0.8333	1.61	2.05	2.46	1.08	1.36	1.64
1900-2000	0.3334	0.5000	0.6667	0.70	0.88	1.06	0.35	0.44	0.53

Table 8.2.6 presents the values for the different variable weights used in the thesis' sensitivity analysis for the blind QALE gain, where 'Mid' represents the central values that have already been utilised above (in Table 8.2.5) as the mainstream values for the blind QALE gain. 'Low' represents an exaggerated weight for the reader who believes that the thesis has over emphasised the extensiveness and value of the morbidity gain (QALY and VSHLY) and over valued the mortality gain (VSL). For the reader that believes the opposite, i.e. that the thesis' central weights (represented in 'Mid') have underestimated the value of the morbidity and mortality and subsequent QALE gain, the 'High' weights are provided.

As has already been explained, the QALY for blindness during different eras of the twentieth century was established through an intricate review of all relevant information sources and forms the nucleus of the blind analysis in the thesis. Despite this wide survey it

[713] See Appendix 12.1.2 for alternative VSL and VSHLY values and Appendix 12.6.2 for alternative QALY values for thesis disability: blindness and Appendix 13.2.2 for QALE gain calculations.

is impossible to identify a definitive and undisputed QALY value. In recognition of this and to highlight that the thesis results still hold without an identical QALY to the ones utilised above, the analysis below will consider the QALE gains with alternative (Low and High) QALYs. The method for calculating these alternatives (lower and upper bound estimates for the blind QALY) utilises the same process as Chapter 8.1, where a lower and upper bound QALYs are used to represent ‘Low’ and ‘High’ in addition to ‘Mid’, which was identified by the thesis’ detailed analysis.

In common with the QALY, the VSL has endured much criticism and a lack of consensus about a definitively correct value. Therefore, in an attempt to recognise this and overcome any claims of bias the thesis will consider the blind morbidity and QALE gain for a range (Low and High) of VSL and subsequent VSHLY values. This consideration is in alignment with the analyses conducted for diseases in Chapter 8.1.

Hence, the rationale for considering the QALE gain for all combinations of sensitivity analysis is the same for the blind QALE gain as it was for the diseases QALE gains outlined (in more detail) above, in Section 8.1. To this end, Table 8.2.7 will provide the results of calculating these alternative QALY, VSL and VSHLY weights in the QALE gain methodology (for blindness). Also in common with Chapter 8.1, in an effort to simplify the mass of possible results (the full range of which are provided in appendices 12.7.2 and 13.2.2) the tables below will provide a summary about the ‘Mid’ and most extreme results, in order to provide the broadest possible array of estimates. This will be achieved through considering the highest possible QALE gain estimate (High VSL, High VSHLY, High QALY) and the lowest possible (Low VSL, Low VSHLY, Low QALY), in conjunction with the mid estimates (Mid VSL, Mid VSHLY, Mid QALY).

Results: Quantitative: Disability

Table 8.2.7: QALE gains for thesis disability: blindness with alternative QALY, VSL and VSHLY values (millions of 1990 international $)[714]

Period	**Low QALY, Low VSL, Low VSHLY**	**Mid QALY, Mid VSL, Mid VSHLY**	**High QALY, High VSL, High VSHLY**
1900-1925	3212	4049	4897
1925-1950	-915	-1257	-1464
1950-1975	3875	4899	5908
1975-2000	5353	6806	8168
1900-2000	8028	10260	12271

Table 8.2.7 highlights and summarises a general trend for the blind QALE gain: as the value of the QALY, VSL and VSHLY increase so does the value of the QALE gain, with the exception of 1925-1950, which displays the opposite trend, whereby increasing the weight of the variables (i.e. moving from 'Low' to 'High') increases the magnitude of the loss or increasingly reduces the QALE gain. This can be considered as a result of an increasingly valuable opportunity cost of the negative morbidity gain. The general trend in Table 8.2.7 (shown for all other years) is sensible as the QALY, VSL and VSHLY are the valuation aspects of health improvements (QALY and VSHLY for the morbidity gain + VSL for the mortality gain, which all contribute to the QALE gain) and therefore, when the magnitude of these variables increases so do the morbidity and mortality gain, which subsequently increases the QALE gain.

A final noteworthy trend in Table 8.2.7 is the considerable variance between the lowest and highest estimates, i.e. the values in 'Low QALY, Low VSL, Low VSHLY' versus 'High QALY, High VSL, High VSHLY'. This variance in the QALE gain can be interpreted to be a result of the very broad sensitivity analysis (utilised in the thesis), which encompasses a range of twenty seven estimates (instead of one estimate) as a result of considering QALY: Low, Mid, High; VSL: Low, Mid, High; VSHLY: Low, Mid, High, and all possible combinations of these estimates in the QALE methodology.

Despite the wide range of estimates and the considerably lower value of the disability QALE gain (compared to the diseases QALE gains identified in Chapter 8.1) these blind QALE gain results are still important as they provide a broader indication of twentieth

[714] Table 8.2.7 is based on the same type of calculations as Table 8.1.7 and 8.1.11 and 8.1.10. See Appendix 12.1.2 for VSL and VSHLY range of values to be included in sensitivity analyses and Appendix 12.6.2 for range of QALY values and Appendix 12.7.2 and 13.2.2 for calculations.

century health (encompassing disability as well as disease) and, although relatively minimal (and negative in 1925-1950), these blind QALE gains still contribute to the claims and conclusions that have been made throughout the thesis about the importance of twentieth century trends in health and the need to measure this in some form of extended national income.

8.2.6 Value of QALE Gain Relative to GDP

It is desirable to consider these results relative to twentieth century GDP per capita. Chapter 3 and Chapter 8.1 have explained and justified (in more detail) the appeal of considering the QALE results in conjunction with GDP growth. The process conducted in Chapter 8.1 will be applied here in order to highlight the contribution of the QALE gain for disability relative to GDP, and subsequently estimate a more accurate national income index that accounts for disability (in addition to disease which has already been calculated in Chapter 8.1).

Table 8.2.8: Compound average growth rates of GDP per capita, mortality gain, morbidity gain and QALE gain and GDP per capita growth adjusted for QALE gain (using mid value QALY, VSL, VSHLY variables and age weighting mortality) for thesis disability: blindness (percentage per annum)[715]

Period	**GDP pc Growth**	**Mortality Gain Growth**	**Blind Morbidity Gain Growth**	**Blind QALE Gain Growth**	**'Adjusted Growth' (QALE + GDP pc)**
1900-1925	0.3	2.2	0.1	2.3	2.6
1925-1950	1.4	2.0	-3.2	-1.3	0.1
1950-1975	2.2	0.7	1.1	1.7	3.9
1975-2000	1.8	1.3	-0.3	0.9	2.7
1900-2000	1.4	1.4	-0.5	0.9	2.3

Table 8.2.8 considers the compound average growth rate of GDP per capita, and mortality, morbidity and QALE gain growth (for blindness). This is achieved through applying the most credible mortality gain, morbidity gain and QALE gain estimates for blindness (which are deemed by the author to be the mid morbidity gain values, presented in Table 8.2.5 and the age-weighted mid mortality gain values presented in Table 8.1.14) to the compounding formula, in order to generate an estimate about average growth per annum of these variables for the twentieth century.

Once this has been achieved it is possible to consider the relative value of mortality gains, morbidity gains and QALE gains versus GDP gains. This consideration reiterates the limited magnitude of the blind morbidity and QALE gain relative to the morbidity and subsequent QALE gains for the diseases considered in the thesis. For example, in Table 8.2.8, the blind morbidity gain growth was less than the mortality gain in 1900-1925, 1925-1950, 1975-2000 and 1900-2000 (i.e. in every period other than 1950-1975). Furthermore, this detraction was so extensive that the blind QALE gain was less valuable than the mortality gain in 1925-1950, 1975-2000 and 1900-2000.

[715] See Appendix 12.11 for GDP per capita compound average growth rate calculations and see Appendix 12.2 and 12.12.2 and 13.6.2 for QALE gain growth calculations.

Table 8.2.8 also considers ‘Adjusted Growth’ (shown in the final column), which represents what GDP per capita per annum growth would be if it was extended to include gains in health or QALE (i.e. ‘Adjusted Growth’ = GDP per capita growth + QALE gain growth). The above trend of a generally negative blind QALE gain growth is reflected here as the contribution of the blind QALE gain to ‘Adjusted Growth’ is nearly entirely a result of the morbidity gain (as the blind morbidity gain tended to be minimal or negative).

Therefore, the above analysis has highlighted the value of twentieth century improvements in the quality of life associated with blind morbidity. Although when the blind morbidity gain is compared to GDP per capita and the mortality gain (and the disease morbidity gains) the results are less impressive, this should not detract from the previous findings in this chapter, which highlight important improvements in the quality of life for the blind (embodied in the improved QALY [shown in Table 8.2.2] and a decline in the average number of blind years per episode [shown in Table 8.2.1]). Finally, the contrast in the twentieth century history of disability and disease is highlighted by the analysis here, which provides an additional (and not necessarily predictable) important historical detail for the story about the health and welfare related quality of life of the population in twentieth century England.

8.3 Summary Quantitative Results

8.3.1 Summary Presentation of QALE Gains

This final segment of Chapter 8 will provide a summary of the previous analyses (for diseases and disability) and enable the reader to identify and evaluate what they deem to be the most feasible estimates about the value of the QALE gain. With reference to the flow chart example at the beginning of Chapter 8, this represents stage 12, where the reader is able to identify the value of the QALE gain (for tuberculosis, breast and stomach cancer and blindness) associated with what they believe about the value of the QALY, VSL and VSHLY used in the thesis and also whether they agree that Murray's age weighting function should be applied to the results.

Figure 8.3.1 (below) provides a summary flow chart of the QALE methodology sensitivity analyses, which contains different variable weights ('Low', 'Mid', 'High' values for the QALY, VSL and VSHLY), which are utilised to calculate the morbidity (QALY and VSHLY) and mortality (VSL) gains and then combined to form the QALE gain. Figure 8.3.1 has been provided to illuminate the methodological process of the QALE gain sensitivity analysis and help the reader decipher what they believe to be an accurate QALE gain value.

Following on from this flow chart, Table 8.3.1 provides a translation of the flow chart decisions. I.e. if the reader decides that: i) they are not willing to accept the thesis QALY and ii) that this is because they deem the QALY values outlined in the qualitative illness chapters as too high, Table 8.3.1 will translate what QALY value this reader favours (this is shown as the 'Low value' for the QALY [in the second row of Table 8.3.1]). Once the reader has made these considerations for all of the thesis QALE methodology variables (i.e. answered all stages of the flow chart) they will have created their profile of QALY, VSL and VSHLY values for use in the QALE gain methodology.

Table 8.3.2 presents all of the possible combinations for the QALE methodology variables that the reader could have chosen from the flow chart in Figure 8.3.1. Hence, once the reader has chosen their preferred variable weights (from answering the flow chart questions) these are translated into the QALE gain methodology terminology (i.e. from 'flow chart term' to 'methodological term') in Table 8.3.1. Then, Table 8.3.2 presents all

possible QALY, VSL and VSHLY and age weighting combinations, which are numbered to accord with the final two tables (8.3.3 and 8.3.4) in Chapter 8.3.

Once the reader has identified the number that their profile QALE methodological variable weights have been given (shown in Table 8.3.2) they can identify their preferred QALE gain result (in Table 8.3.3 and 8.3.4), through finding the same number on these two tables.

Table 8.3.3 and 8.3.4 provide the series of all possible QALE gains for the thesis sensitivity analysis variable weights. Table 8.3.3 shows all combinations of the QALE gain (i.e. this is the consideration made in Table 8.1.12 and 8.2.7). Table 8.3.4 shows this QALE gain in terms of QALE gain growth per annum for the twentieth century, which can the be appended to GDP per capita growth (referred to as ‘Adjusted Growth’ in Chapter 8.1 and 8.2) to represent an extended national income growth that accounts for improvements in health (or ‘QALE gain growth’ in Tables 8.1.14, 8.1.15 and 8.2.8). To calculate ‘Adjusted Growth’ the QALE gain growth presented in Table 8.3.4 needs to be summed with GDP growth of 1.4 percent for 1900-2000.

This process is presented below, starting with Figure 8.1.3, the flow chart for the QALE gain methodology choice of variables, and then continues in the sequence outlined above.

Figure 8.3.1: QALE gain methodology variable weight options

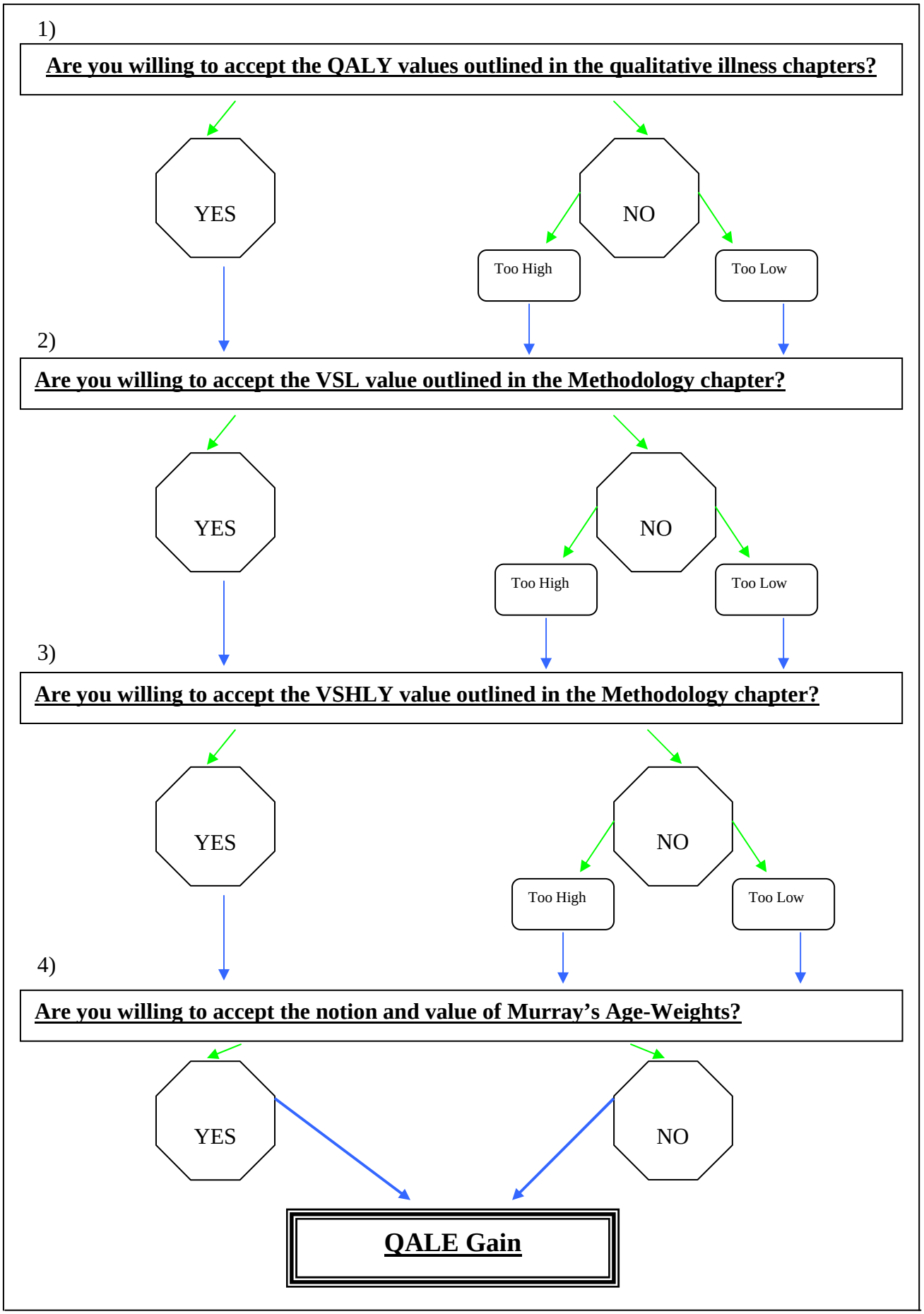


Table 8.3.1: Translation of QALE methodological flow chart outlined in Figure 8.3.1 into QALE methodology variable weights (used in Tables 8.3.2, 8.3.3 and 8.3.4)

Flow Chart Term:	'Yes'	'No' – 'Too High'	'No – Too Low'
Methodological Term:	Mid Value	Low Value	High Value

These terms apply to the first three options in the chart, i.e. the QALY, VSL and VSHLY (which is a combination of the QALY and VSL). The final option in Figure 8.3.1, which is age-weighting, can be deciphered by a simple 'Yes' or 'No' decision. I.e. either the reader agrees with age-weighting ('b' in the tables below) or does not ('a' in the tables below). The blind QALE gain age-weighting only applied to the mortality gain component of the QALE gain (as the data does not exist to perform the age weighting calculations to the blind morbidity gain) and therefore answer 'b' for blind represents the blind QALE gain that comprises: un weighted blind morbidity gain + age weighted mortality gain.

The table below provides the QALE methodology variable weight results of all the potential combinations of answers from the methodological flow chart. Once these have been outlined (in Table 8.3.2) the reader will be able to identify the corresponding QALE gain result (for their selected answers to the flow chart, in Table 8.3.3 and 8.3.4.

Table 8.3.2: All possible QALE methodology (QALY, VSL, VSHLY) variable weights combinations from flow chart in Figure 8.3.1

Combination Number	**QALY**	**VSL**	**VSHLY**	**Age Weights**	
1	Low	Low	Low	No (a)	Yes (b)
2	Low	Low	Mid	No (a)	Yes (b)
3	Low	Low	High	No (a)	Yes (b)
4	Low	Mid	Low	No (a)	Yes (b)
5	Low	Mid	Mid	No (a)	Yes (b)
6	Low	Mid	High	No (a)	Yes (b)
7	Low	High	Low	No (a)	Yes (b)
8	Low	High	Mid	No (a)	Yes (b)
9	Low	High	High	No (a)	Yes (b)
10	Mid	Low	Low	No (a)	Yes (b)
11	Mid	Low	Mid	No (a)	Yes (b)
12	Mid	Low	High	No (a)	Yes (b)
13	Mid	Mid	Low	No (a)	Yes (b)
14	Mid	Mid	Mid	No (a)	Yes (b)
15	Mid	Mid	High	No (a)	Yes (b)
16	Mid	High	Low	No (a)	Yes (b)
17	Mid	High	Mid	No (a)	Yes (b)
18	Mid	High	High	No (a)	Yes (b)
19	High	Low	Low	No (a)	Yes (b)
20	High	Low	Mid	No (a)	Yes (b)
21	High	Low	High	No (a)	Yes (b)
22	High	Mid	Low	No (a)	Yes (b)
23	High	Mid	Mid	No (a)	Yes (b)
24	High	Mid	High	No (a)	Yes (b)
25	High	High	Low	No (a)	Yes (b)
26	High	High	Mid	No (a)	Yes (b)
27	High	High	High	No (a)	Yes (b)

Therefore, in the following two tables, the QALE gain and the QALE gain growth (as an appendage to GDP per annum growth) will be presented for all illnesses and combinations of QALE methodology variable weights for the period 1900-2000. The connectivity

between the above table and those below, which provides the results, is the number (1 to 27) in the first column of all the tables. Additionally, in an attempt to make the referencing easier, the tables below will also contain a column that summarises the combinations. Hence, the column ‘Code’ will provide the first letter (L for Low, M for Mid and H for High) of the level of the QALY, VSL, VSHLY, respectively. For example, No. 1 (in the two tables below) relates to combination 1 in the table above, which represents the result for Low QALY, Low VSL and Low VSHLY, which is summarised in the ‘Code’ column (in the two tables below) as LLL (i.e. Low, Low, Low).

Results: Quantitative: Summary: Disability and Disease

Table 8.3.3: QALE gain results for all combinations of variable weights in methodological flow chart for thesis diseases and disability 1900-2000 (millions of 1990 international $)[716]

No.	Code	Breast cancer		Stomach cancer		Tuberculosis		Blindness	
		a	b	a	b	a	b	a	b[717]
1	LLL	14198	10553	13107	9136	25793	18419	8028	4580
2	LLM	17052	11821	15961	10017	28647	22819	8062	4614
3	LLH	19925	13094	18834	10902	31521	27241	7143	3694
4	LML	15075	11404	13693	9608	29771	21372	8490	5041
5	LMM	17929	12998	16546	10740	32624	26909	7325	3876
6	LMH	20803	14624	19420	11845	35498	32552	6160	2711
7	LHL	15920	12223	14257	10063	33601	24216	8016	4568
8	LHM	18774	14155	17111	11406	36454	30925	6615	3166
9	LHH	21648	16097	19985	12754	39328	37667	5213	1764
10	MLL	15505	12583	14201	11166	31720	20448	11874	7563
11	MLM	18359	13850	17054	12047	34573	24849	10955	6644
12	MLH	21233	15124	19928	12931	37447	29270	10035	5724
13	MML	16733	13434	12123	11638	37281	23402	11382	7071
14	MMM	19575	15029	17952	12770	40082	28940	10260	5893
15	MMH	22460	16654	20807	13875	43009	34582	9052	4741
16	MHL	17914	14253	15925	12093	42637	26246	10909	6598
17	MHM	20768	16185	18779	13436	45491	32955	9507	5196
18	MHH	23642	18127	21653	14784	48364	39697	8105	3794
19	HLL	16819	14399	15299	12982	37657	22265	14659	9379
20	HLM	19673	15667	18152	13864	40528	26665	13740	8460
21	HLH	22547	16940	21026	14748	43402	31087	12821	7540
22	HML	18398	15250	16471	13454	44828	25218	14167	8887
23	HMM	21251	16845	19324	14586	47682	30755	13003	7722
24	HMH	24125	18470	22198	15691	50556	36398	11838	6557
25	HHL	19918	16069	17599	13909	51717	28062	13694	8414
26	HHM	22771	18001	20453	15252	54570	34771	12293	7012
27	HHH	25645	19943	23327	16600	57444	41513	12271	6991

[716] See Appendix 12.7, 12.10, 13.2 and 13.5 for base calculations.
[717] This combines the un-weighted blind morbidity gain with the age-weighted mortality gain. See Appendix 12.10.2 and 13.5.2 for calculations.

Results: Quantitative: Summary: Disability and Disease

Table 8.3.4: QALE gain contribution to compound average GDP per capita growth per annum: results for all combinations of variable weights in methodological flow chart for thesis diseases and disability 1900-2000 (percent pa)[718]

No.	Code	Breast cancer		Stomach cancer		Tuberculosis		Blindness	
		a	b	a	b	a	b	a	b[719]
1	LLL	2.1	1.5	1.9	1.3	3.7	2.7	1.3	0.8
2	LLM	2.5	1.7	2.1	1.5	4.1	3.3	1.2	0.7
3	LLH	2.4	1.9	2.2	1.6	5.5	3.9	1.0	0.5
4	LML	2.6	1.9	2.0	1.6	4.3	3.9	1.2	0.7
5	LMM	2.3	1.9	1.8	1.6	4.7	3.9	1.1	0.6
6	LMH	2.7	2.1	2.4	1.7	6.5	4.7	0.9	0.4
7	LHL	2.3	1.8	2.1	1.5	4.9	3.5	1.2	0.7
8	LHM	2.7	2.0	2.3	1.7	5.3	4.5	1.0	0.5
9	LHH	3.1	2.3	2.5	1.8	5.7	5.5	0.8	0.3
10	MLL	3.1	1.8	2.5	1.7	5.4	3.0	1.7	1.1
11	MLM	2.7	2.0	2.5	1.8	5.0	3.6	1.6	1.0
12	MLH	2.8	2.2	2.6	2.0	5.9	4.2	1.5	0.8
13	MML	2.2	1.9	2.3	1.7	4.6	3.4	1.6	1.0
14	MMM	3.3	2.2	2.6	1.9	5.8	4.2	1.5	0.9
15	MMH	3.1	2.4	2.8	1.6	6.9	5.0	1.3	0.7
16	MHL	2.4	2.1	2.5	1.8	5.4	3.8	1.6	1.0
17	MHM	3.0	2.3	2.7	1.9	6.6	4.8	1.4	0.8
18	MHH	3.4	2.6	3.0	2.1	7.0	5.7	1.2	0.5
19	HLL	2.3	2.1	2.7	1.9	4.6	3.2	2.1	1.4
20	HLM	2.7	2.3	2.9	2.0	5.0	3.9	2.0	1.2
21	HLH	3.3	2.5	3.0	2.1	6.3	4.5	1.9	1.1
22	HML	2.4	2.2	2.8	1.9	5.4	3.7	2.1	1.3
23	HMM	2.8	2.4	3.0	2.1	5.8	4.5	1.9	1.1
24	HMH	3.5	2.7	3.2	2.3	7.3	5.3	2.0	0.9
25	HHL	2.9	2.3	2.9	2.0	7.5	4.1	2.0	1.2
26	HHM	3.3	2.6	3.1	2.2	7.9	5.0	1.8	1.0
27	HHH	3.7	2.9	3.4	2.4	8.3	6.0	1.6	0.8

[718] See Appendix 12.12 and 13.6.1 for base calculations.
[719] This combines the un-weighted blind morbidity gain with the age-weighted mortality gain. See Appendix 12.12.2 and 13.6.2 for calculations.

Results: Quantitative: Summary: Disability and Disease

The above two tables contain the broad set of results for the same calculations that were made for the diseases QALE gain in Tables 8.1.12 and 8.1.13 and disease QALE gain growth in Tables 8.1.14 and 8.1.15 and the disability QALE gain in Table 8.2.7 and disability QALE gain growth in Table and 8.2.8. These estimates exist for all the time periods (in addition to 1900-2000, presented here) considered in the thesis (in Appendix 12.7, 12.10, 12.2 and 13.2, 13.5, 13.6) but for simplicity in answering the key questions of the thesis, about twentieth century improvements in health, only the twentieth century as a whole will be considered here.

In both tables the QALE gain (Table 8.3.3) and QALE gain growth contribution to GDP growth per annum (Table 8.3.4) there are two columns for all illnesses. As has been explained above, these represent the un-weighted (a) and age-weighted (b) estimates. It is important to explain why column b is consistently lower in value and contribution than column a. As was identified in Chapter 8.1, when the age-weighting is applied the QALE gain results become more circumscribed than their un-weighted counterparts, because certain ages receive lower weighting than others: and the QALE gain (improved morbidity + mortality) was often skewed towards the youngest and oldest age groups (which receive a lower weighting than middle ages) in twentieth century England. However, it should be noted that, although the age weighting function does noticeably reduce the QALE gain (both in absolute terms [Table 8.3.3] and in percentage growth per annum [Table 8.3.4]), this reduction does not change the overall conclusions of the thesis.

Another remarkable feature in Table 8.3.3 and 8.3.4 is that, in the ‘blind’ columns, the lowest overall estimate is not yielded by LLL and the highest overall estimate is not yielded by HHH. At a first approximation this result seems counter intuitive and perhaps incorrect. However, this is not the case. The reason for this relationship is the negative morbidity gain for blindness between 1900 and 2000, which means that the interaction between the QALY, VSL and VSHLY variables in the thesis QALE methodology is not consistent or reinforcing. Hence, when the QALE gain is negative: making the QALY and the VSHLY positive increases the magnitude of the loss, as the QALE gain becomes increasingly negative and increasing the VSL would help (as this would make the mortality gain component of the QALE gain methodology more valuable) but this in turn will cause detractions from the morbidity gain (as the morbidity gain = the morbidity burden change * VSHLY and the VSHLY = VSL * QALY).

Results: Quantitative: Summary: Disability and Disease

Table 8.3.3 and 8.3.4 (and the previous analysis) highlight that: however low the values of the QALE methodology variables (within the realms of those provided in the thesis' analysis) the QALE gain still yields significant results which provide an important contribution to economic development and standards of living. This is even true for blindness, which in the above analysis did not experience marked QALE gains. Conversely, this optimistic conclusion about the QALE gain is most applicable to tuberculosis, which is to be expected as this disease experienced a considerable improvement during the twentieth century, along with the general class of infectious diseases which were largely eliminated or much more easily managed during the twentieth century.

The veracity of these claims concerning the contribution of improved health, and morbidity in particular will be reinforced in the following chapter when these results are extended to estimate the significance of twentieth century improvements in the aggregate QALE.

9. Extended Results (Disability and Disease)

9.1 Introduction

In an attempt to add greater significance to the findings of the thesis it is desirable to consider the wider implications of the results from the three diseases and one disability analysed previously (in Chapter 8.1 and 8.2). The objective of this exercise is to highlight the extensiveness of improvements in health through providing a lower bound estimate about the value of aggregate QALE improvements in twentieth century England.

The aggregate QALE gain will be estimated using the following structure. The entire twentieth century profile of diseases will be differentiated into four states: i) infectious (proxied by tuberculosis), ii) non-infectious (proxied by stomach cancer), iii) disabilities (proxied by blindness) and iv) the residual. In order to adhere to the objective of producing lowest bound estimates, these states will be considered with the following constraints.

Infectious diseases will be represented by only tuberculosis and will assume:

(1) *'The only improvements in twentieth century health and welfare related quality of life for infectious diseases were for tuberculosis'*

This is conservative because of the breadth (prevalence) and depth (quality of life) of twentieth century QALE adjustments associated with infectious diseases, such that at no time did tuberculosis represent more than 12 percent of this category. The extensiveness of the twentieth century decline of infectious mortality is illustrated by Figure 2.3.2 (which outlines the burden of disease [infectious and other key categories of mortality] at the start and end of the twentieth century).

Non-infectious diseases will be conservatively estimated through applying the twentieth century stomach cancer quality of life profile to all non-infectious diseases. The non-infectious series of calculations will assume:

(2) *'All non-infectious diseases had the unfavourable quality of life experienced by stomach cancer'*

This is conservative because it is almost certain that the average burden of non-infectious diseases was not as detrimental to quality of life as stomach cancer. The severity of the stomach cancer burden is highlighted in Table 7.3.1 in Chapter 7.3 and Table 8.1.1 in Chapter 8.1. Hence, breast cancer is also classified as a non-infectious disease and therefore comparing the margin in the QALY between these two diseases highlights the conservativeness of the assumptions in this chapter. Moreover, although the QALY improvements associated with breast cancer are impressive (e.g. Table 7.3.1 shows that the breast cancer QALY improved from 0.3 of a healthy life year in 1900 to 0.8 in 2000), these are not necessarily representative of the most far reaching improvement in non-infectious diseases. For example, circulatory diseases affected more of the population (more fractions of healthy life years) and experienced a more sustained decline in prevalence and (likely) improvement in quality of life. Therefore, this provides further justification for the stringency with which the non-infectious component is being estimated in this chapter.

The twentieth century burden of all disabilities will be most conservatively approximated through utilising only blind quality of life to represent all twentieth century quality of life improvements for disabilities:

(3) *'The only disability to experience quality of life improvements in the twentieth century was blindness and therefore this represents all twentieth century quality of life improvements in disabilities'*

This is conservative because of the likelihood that blindness experienced more limited developments in quality of life than an 'average' disability and subsequently, because there is no accounting for a positive, aggregate disability improvement. Moreover, in a similar way to the role of stomach cancer in the non-infectious category, blindness was a greater burden to quality of life than the 'average' disability during the twentieth century.

The final category is the residual, which includes all illnesses that cannot be classified into infectious, non-infectious and disability. The twentieth century fall in the morbidity burden of this category is likely to be sizeable, because of general quality of life improvements and more substantially, because of the decline in the size of this category. The residual has shrunk over the twentieth century as disease classification became more precise: from 192 categories in ICD1 used in 1900 to 5,292 in ICD9 used in 2000. I.e. as illnesses have

become more precisely categorised the residual has declined: the extent of this precision is highlighted by the substantial growth of the number of ICD categories[720].

Because of the ambiguity involved in estimating the residual and the associated negative influence on the justifiability of the extended results, the residual will be eliminated altogether and none of the residual QALE gains will be included, as:

(4) *'No residual illness will be evaluated'*

After the results have been presented for the above assumptions the thesis will highlight that even with these lowest bound estimate assumption constraints, the contribution of improved health was substantial. This will be vividly highlighted in Table 9.4 which calculates the value of twentieth century improvements in health.

The above assumptions (1) to (4), which will be applied throughout this chapter in order to yield the most conservative estimate for the aggregate twentieth century QALE gain are summarised in Table 9.1.

[720] Calculated from: Office of Population, Censuses and Surveys, "Twentieth Century Mortality"

Table 9.1: Definition of aggregate QALE morbidity categories used in extended results

Illness	**Definition / Context**
Infectious	Tuberculosis is used to represent an excessively conservative situation in which tuberculosis was the only infectious disease to improve. The magnitude of improvement would be significantly greater if all infectious diseases were included and profiled with the tuberculosis QALY improvements.
Non-Infectious	Non-infectious diseases have been conservatively estimated through the application of the stomach cancer profile (which is excessively pessimistic compared to the majority of non-infectious diseases) to all twentieth century non-infectious diseases.
Disability	The improved quality of life associated with blindness has been used to represent all disabilities and subsequently, provides a conservative proxy for the value of twentieth century disability gains, especially as blindness can be considered as a particularly adverse disability.
Residual	The residual represents all other causes of death that are not contained in the above three categories. Because of the varied and hybrid nature of this category it is impossible to generate a reliable summary measure and therefore, the residual gains will not be estimated.
TOTAL	This contains the sum of the above categories and therefore represents the entire twentieth century morbidity burden (based on a most conservative forward extrapolation of the thesis illnesses).

In addition to making these stringent assumptions (summarised in Table 9.1) this chapter will utilise the lowest bound estimates possible. In Chapter 8.1 and 8.2 the sensitivity analysis included the estimation of the QALE gain using all 'Low' variables (i.e. 'Low' QALY, 'Low' VSL and 'Low' VSHLY) which represent the variables that will be used here[721].

9.2 Aggregate QALE Gain

Chapters 8.1 and 8.2 have highlighted the importance of considering the QALE gain (mortality gain + morbidity gain) in order to gauge a more accurate account of twentieth century economic development. In the previous chapters the morbidity (and QALE) gain was a direct function of the illness to which it was related. I.e. the morbidity (or QALE) gain for breast cancer between 1900 and 2000 depends on the morbidity burden

[721] See Appendix 12.7.and 13.2 for detailed calculation of the 'Low' combination of all variables in the tuberculosis and blind morbidity gain, respectively. These lowest bound QALE variable weights will be utilised here. Non-infectious disease calculations will also utilise 'Low' QALY, VSL and VSHLY variable weights. It should be noted that because of the interaction between the numbers and variables (which are features not strictly related to the QALE considerations of the thesis) it is possible to find values which are lower than the 'Low' QALY, VSL and VSHLY estimates (for example, see Table 8.3.3 and the subsequent explanation about this phenomenon). Hence, it should be noted that what needs to be evaluated here in order to provide original estimates about the lowest bound approximation of the value of QALE in twentieth century England is the QALE gain that encompasses the lowest variable weights as these are based on the most stringent assumptions about the value of improvements in QALE.

(prevalence * QALY) of breast cancer and the morbidity gain (change in the morbidity burden * VSHLY [= VSL * QALY]) of breast cancer, and (when combined with the mortality gain), the QALE gain for breast cancer between 1900 and 2000. The exercise below makes slightly more ambiguous calculations, where the QALE gain represents more than only one underlying illness. The assumptions that will be used in this chapter, to yield a more aggregate QALE gain estimate are outlined in Table 9.1. For example, in Chapter 8.1, the QALE gain for stomach cancer was (directly and only) related to the morbidity gain for stomach cancer (+ the mortality gain). In the Chapter 9 analysis (below), the stomach cancer morbidity gain profile will be used to proxy all non-infectious diseases, through utilising the stomach cancer variables (QALY, VSHLY) and applying these to the non-infectious prevalence. The results of this process will be a (lower bound) estimate of the QALE gain (stomach cancer morbidity gain variables + non-infectious morbidity gain prevalence = non-infectious morbidity gain → non-infectious morbidity gain + mortality gain = non-infectious QALE gain) for all non-infectious diseases.

This process (of using the QALY and VSHLY profile [for the specific disease, presented in Chapter 8] combined with the more broad disease class prevalence [presented here]) is (the aggregate QALE gain methodology) utilised only for non-infectious diseases. This is because the QALE gain for infectious diseases and disability requires a different approach (due to the nature of these broad morbidity classes).

Chapter 8.1 has highlighted the substantial QALE gains associated with tuberculosis (see Table 8.1.7 and 8.3.3[722]), which can be considered as a typical twentieth century trend for infectious diseases (e.g. Part I of the thesis has indicated the nature of twentieth century morbidity through the epidemiological transition, which represents a decline in infectious morbidity that was replaced by non-infectious). Therefore, Chapter 9 will present the lowest bound ('Low' QALY, 'Low' VSL, 'Low' VSHLY and age-weighted) morbidity and QALE gain for tuberculosis (see Table 8.3.3) as the infectious disease QALE gain. This represents the lowest bound infectious QALE gain for two reasons: because it is the lowest bound tuberculosis QALE gain and also because it is utilising the assumption that:

> *'The only improvements in twentieth century health and welfare related quality of life for infectious diseases were for tuberculosis'*

[722] The values utilised in Chapter 9 are derived from Table 8.3.3 'b' columns (and not Table 8.1.7 or 8.2.7) because Table 8.3.3 considers the age weighted, 'Low' QALY, VSL, VSHLY morbidity and QALE gain, which represents the lowest bound estimate.

This creates a significant underestimate of the potential QALE gain for all types of infectious morbidity (additional to tuberculosis).

The QALE gain for disability, presented here will utilise the QALE gain for blindness from Chapter 8.2. In a similar way to the abovementioned QALE gain for infectious diseases (where only tuberculosis was valued for the entire infectious gain) only the blind QALE gain will be used as the representative of all disability improvements. This is conservative because of the likelihood that blindness experienced more limited developments in quality of life than an 'average' disability and subsequently, because there is no accounting for a positive, aggregate disability improvement (as the 1900-2000 blind morbidity gain is negative). Hence, in a similar way to the role of stomach cancer in the non-infectious category, blindness was a greater burden to quality of life than the 'average' disability during the twentieth century. Therefore, Chapter 9 will present the lowest bound ('Low' QALY, 'Low' VSL, 'Low' VSHLY) morbidity and QALE gain for blindness (see Table 8.3.3[723]) as the disability QALE gain.

Table 9.2 (below) applies the above methodological assumptions to produce estimate about the morbidity, mortality and QALE gain for the hypothetical disease environment that Chapter 9 has generated (summarised in Table 9.1) in order to provide a lower bound estimate about the aggregate QALE gain in twentieth century England.

Table 9.2: Aggregate QALE gain (morbidity gain + mortality gain): monetary value of improvements in the burden of morbidity and mortality for aggregate disease environment, 1900-2000 (millions of 1990 international $)[724]

Illness	**Morbidity Gain**	**Mortality Gain**	**QALE Gain**
Infectious	11047	7372	18419
Non-Infectious	-7467	7372	-813
Disability	-2792	7372	4580
			8160

Table 9.2 is following the thesis QALE gain methodology, where the morbidity and mortality gain are calculated (through valuing [using the VSHLY and VSL] the change in the morbidity and mortality burden) and summed to estimate the QALE gain. The data

[723] Ibid

[724] See Appendix 13.3 (age weighted, Low VSL) for mortality gain calculation. See Appendix 13.4 (age-weighted, Low VSL, VSHLY and QALY estimate) for morbidity gain calculations. See Appendix 12.10 and 13.5 (age-weighted, Low VSL, VSHLY and QALY estimate) for QALE gain calculations.

about the prevalence, QALY, VSL and VSHLY for the disease categories in Table 9.2 are outlined in detail in Appendix 12.14.1. This represents stages 8, 9 and 10 in the flow chart example at the beginning of Chapter 8. Also, the sensitivity analysis stages 11a, 11b, 11c and 11d are utilised as these values represent the QALE gain for the 'Low' thesis variables that have also been age weighted. I.e. the results here, for infectious (which equals tuberculosis) and disability (which equals blindness) are the same as the age weighted, 'Low' QALY, VSL, VSHLY QALE gain results in Table 8.3.3.

Hence, the table above presents the morbidity gain, mortality gain and subsequent QALE gain for the aggregate diseases environment. For infectious diseases this is the same calculation that was made in the first row (column 8 [Tuberculosis: b]) of Table 8.3.3, which represents the tuberculosis QALE gain that utilises the 'Low' QALY, 'Low' VSL and 'Low' VSHLY variables which is then subjected to Murray's age weighting function. Tuberculosis represents all infectious diseases (QALE gains) here because of the conservative assumptions of this chapter (outlined in Table 9.1).

For disability the result in Table 9.2 is also derived from the same calculation that was made in the first row of Table 8.3.3 (column 10 [Blind: b]), which represents the blind QALE gain that utilises the 'Low QALY', 'Low' VSL and 'Low' VSHLY variables. Murray's age weighting function is also applied to the mortality gain (but not to the morbidity gain is not because the data does not exist to make this calculation).

The second row of Table 9.2 represents the non-infectious morbidity, mortality and QALE gain. The mortality gain is derived from the same calculation as for disability and infectious diseases (i.e. through using a 'Low' VSL in the mortality gain methodology and using Murray's age weighting function). The morbidity gain is derived through a slightly different process: this calculation is constructed through combining the prevalence of non-infectious diseases (as the total number of reported diseases that are non-infectious and not in the residual, shown in Appendix 12.14.1) with the stomach cancer variable (QALY and VSHLY) profile, which is thought to be worse than the average variable profile for non-infectious diseases. Hence, the non-infectious morbidity burden is calculated as: non-infectious disease prevalence * stomach cancer ['Low'] QALY) and the subsequent morbidity gain is calculated: change in the morbidity burden (between 1900 and 2000) * ['Low'] VSHLY ('Low' VSL * 'Low' [stomach cancer] QALY).

The mortality gain shown in column three of Table 9.2 represents the lowest possible estimate of the mortality gain throughout all of the thesis' analysis. This was derived through valuing the change in the mortality burden with a 'Low' VSL, and also through subjecting this mortality gain to Murray's age weighting function (Chapter 8.1 has explained why this reduced the mortality gain).

Finally, the QALE gain column is established by summing the morbidity gain and the mortality gain, for each disease category (this accords with the thesis QALE methodology and is explained in detail in Chapter 8.1). The final row of the QALE gain column (Total QALE gain) represents the aggregate QALE gain. This is calculated by summing all the morbidity gains with the mortality gain (i.e. 11047 + -7467 + -2792 + 7372 = 8160), which yields an aggregate QALE gain of 8,160 (thousand 1990 international $) or in excess of 8 billion (1990 international $) for twentieth century England.

Hence, Table 9.2 highlights the extensiveness of improvements in health, which were most significant for infectious causes, as a result of a substantial decline in the prevalence and an improvement in the QALY. Table 9.2 illustrates that the infectious QALE gain was so extensive that it was able to entirely offset the losses for non-infectious and disability. Similarly although not to the same extent, the mortality gain was positive and significant as a result of a substantial decline in the death rate and secondarily, the value (even at a lowest approximation) of this improvement. The other two broad categories of morbidity (non-infectious and disability) reflect a less positive trend: where the QALY has improved but the prevalence has increased (see Table 8.1.1 for tuberculosis and Table 8.2.3 for blind) to such an extent that there is a negative morbidity gain and a subsequently lower QALE gain. This was most noteworthy for non-infectious diseases between 1900 and 2000.

However, even in this lowest bound exercise, from a quality of life perspective, the actual situation was more optimistic than what is implied in the table, because the pessimism is more associated with prevalence than the QALY. It should also be noted that in actuality these improvements would be much more substantial than they are here, as the thesis extended results aggregate QALE gain is trying to calculate the lowest bound value (in order to escape from accusations of bias).

As well as generating an approximate lowest bound value of the aggregate QALE gain (which was substantial, at about 8 billion [1990 international $]), Table 9.2 is also valuable

for reiterating the thesis message about the value of morbidity gains and the need to measure these in tandem with mortality gains. This is most crucial for the morbidity gain associated with infectious diseases, which was more valuable than the mortality gain for the twentieth century, which highlights that only considering mortality would create a serious underestimate of twentieth century health gains.

9.3 Value of Aggregate QALE Gain Relative to GDP

In common with Chapter 8.1, 8.2 and 8.3, the value of the aggregate QALE gain results in Table 9.2 can be further emphasised by comparing them with national income growth in twentieth century England. Also in common with Chapter 8.1, 8.2 and 8.3 this exercise will highlight the authenticity of the thesis claims to consider wider notions of economic development that include health, e.g. through appending some form of health statistic to national income measures to account for improvements in health and standards of living. Hence, as well as standardising the aggregate QALE gains with economic growth the information below will provide a more accurate account of twentieth century economic development.

This analysis will use the mortality, morbidity and subsequent QALE gain values presented in Table 9.2, which will be utilised here to calculate the compound average rate of growth for each of these variables so that the difference in value of growth can be identified for mortality, morbidity, QALE and GDP. After this has been achieved the QALE growth will be considered in conjunction with GDP growth in order to present a single, rounded growth estimate (presented here as ‘Adjusted Growth’), where the growth of the aggregate QALE will be added to the growth of GDP. This will provide a first approximation about what the extended ‘Fisherian’ national income would have been for twentieth century England.

Table 9.3: Compound average growth rates of GDP per capita, mortality gain, morbidity gain and QALE gain and GDP per capita growth adjusted for aggregate QALE gain, 1900-2000 (percentage per annum)[725]

Illness	**GDP pc Growth**	**Mortality Gain Growth**	**Morbidity Gain Growth**	**QALE Gain Growth**	**'Adjusted Growth' (QALE + GDPpc)**
Infectious			1.6	2.7	4.1
Non-Infectious			-1.1	-0.01	1.4
Disability			-0.4	0.8	2.2
TOTAL	1.4	1.1	0.1	1.2	2.6

Table 8.2.8 considers the compound average growth rate of GDP per capita and the mortality, morbidity and QALE gain growth (for infectious and non-infectious diseases and for disability) for the aggregate twentieth century QALE. This is achieved through applying the mortality, morbidity and QALE gain for the morbid states used in Chapter 9 to the compounding formula, in order to generate an estimate about average growth per annum for the twentieth century.

The reason that the mortality gain growth in this table is different to that which was present in Chapter 8 in Tables 8.1.14 and 8.2.8 is because these tables utilised the age-weighted 'Mid' mortality gain and Table 9.3 utilises the age-weighted 'Low' mortality gain. This is also the reason that the infectious morbidity gain does not equal the tuberculosis morbidity gain in Table 8.1.14 and the disability morbidity gain does not equal the blind morbidity gain in Table 8.2.8. The infectious and disability QALE gains presented in Table 9.3 do correspond with the same indices in Table 8.3.4 (tuberculosis in row 1 column 8 [b] and blindness in row 1 column 10 [b]) as these are the same (age weighted 'Low' QALY, VSL, VSHLY) measures.

[725] See Appendix 12.11 for GDP per capita compound average growth rate calculations and see Appendix 12.14.2 and 13.6 for calculations in Table 9.3.

Extended Results: Quantitative: Disability and Disease

It is possible to consider the relative value of mortality gains and morbidity gains and QALE gains versus GDP gains. This consideration reiterates the negative nature of the non-infectious and to a lesser extent the disability morbidity gain. On a more positive note the substantial infectious morbidity gains are evident in Table 9.3, and are calculated to be grater than GDP growth and the mortality gain, which provided a valuable addition to economic development. These results are also reflected in the QALE gain column for each morbid state, i.e. the QALE gain for infectious was substantial at a twentieth century per annum growth of 2.6 percent (nearly double GDP growth for the same era, which was 1.4 percent). The QALE gain for non-infectious diseases was negative as a result of the magnitude of the negative non-infectious morbidity burden and the disability QALE gain was positive but small, also as a result of the negative morbidity gain (which was not as severe as for non-infectious diseases and this is why the disability QALE gain is positive).

Possibly the most important QALE gain in Table 9.3 is the total QALE gain, because this presents the aggregate QALE gain growth contribution to twentieth century GDP growth. This is also noteworthy because of the magnitude of the aggregate QALE gain growth, which was nearly as significant as the compound average rate of GDP growth per annum in twentieth century England (1.2 percent versus 1.4 percent per annum, respectively).

Table 9.3 also considers ‘Adjusted Growth’ (shown in the final column), which represents what GDP per capita per annum growth would be if it was extended to include gains in health or QALE (i.e. ‘Adjusted Growth’ = GDP per capita growth + QALE gain growth). This shows that for infectious diseases and disability there would be an increase in ‘Adjusted Growth’ and for non-infectious a marginal decrease (of 0.01 percent). Moreover, when GDP growth is combined with aggregate QALE gain growth this shows the magnitude of ‘Adjusted Growth’, where accounting for the aggregate QALE gain nearly doubles existing levels of economic growth. This is significant for two key reasons: the relative value of QALE gains compared to GDP gains (where GDP is 1.4 percent and the QALE gain is 1.2 percent) and also, if this per annum growth gain is considered for the entire twentieth century then this represents a lot of growth (e.g. approximately an additional 120 percent of twentieth century economic growth).

Hence, if improved twentieth century health was included in some form of extended national income measure, then growth would have been more like 2.6 percent per annum

rather than the GDP only measure of 1.4 percent per annum, where the difference between these two represents the value of improvements in health in twentieth century England.

9.4 Value of Aggregate Health (QALY) Gains

Finally, an alternative method for illuminating the extensiveness of the twentieth century aggregate QALE gains is to consider the value of the QALE gain with and without the documented QALY improvements in the morbidity part of this methodology. To recap, the QALY is multiplied with the prevalence to estimate the morbidity burden (prevalence * QALY). The change in the morbidity burden is then valued (by applying the VSHLY, which equals the VSL * QALY), to estimate the morbidity gain, which can then be combined with the mortality gain to calculate the QALE gain (see the QALE methodology flow chart worked example at the beginning of Chapter 8 for a more detailed outline of the thesis QALE gain methodology, especially stages 5 to 10).

As has been outlined above, the QALY is utilised to calculate the morbidity burden (prevalence * QALY) and the morbidity gain (change in the morbidity burden * VSHLY [= QALY * VSL]). Also outlined above, is that the QALY (for all thesis illnesses) improved over the twentieth century. It was these improvements (embodied in the QALY) that were very valuable and provided one of the major contributors to the QALE gain. In order to highlight this point, which will also reaffirm the thesis claims (about the extensiveness of health improvements, the value of these QALE gains and the subsequent need to measure them in some form of extended national income), it is necessary to recalculate the morbidity burden change, morbidity gain and subsequent QALE gain, with a constant QALY value (i.e. one that has not improved beyond the 1900 level). This can then be compared to the genuine morbidity burden change, morbidity gain and QALE gain (using the actual dynamic and improving QALY between 1900 and 2000) in order to identify the margin of difference (i.e. how much more valuable the latter QALE gain is than the former), which represents the value of the changing QALY or the value of health improvements in twentieth century England.

Extended Results: Quantitative: Disability and Disease

Table 9.4: Value of twentieth century health improvements for aggregate disease environment, 1990-2000 (millions of 1990 international $)[726]

Illness	**Mortality Gain**	**Morbidity Gain** (with no health improvements)	**Morbidity Gain** (with health improvements)	**QALE Gain**		**Value of health improvements**
Infectious		506	11047	No health improvements	Health improvements	
Non-Infectious		-8811	-7467			
Disability		-23950	-2792			
TOTAL	7372	-32760	788	-25388	8160	**33548**

In Table 9.4 the mortality gain estimate is the same as what has been calculated above (see Table 9.2). The morbidity gain estimate (with health improvements) is also the same as the morbidity gain that was calculated in Table 9.2. The subsequent QALE gain for health improvements (which is the sum of all health improvement morbidity gains and the mortality gain [11047 - 7467 - 2792 {=788} + 7372=8160]) is also the same calculations as in Table 9.2.

The morbidity gain (with no health improvements) has been calculated by conducting the same QALE gain methodological process (morbidity burden [prevalence * QALY] → morbidity burden change between 1900 and 2000 → value of the change in the morbidity burden [change in the morbidity burden * VSHLY = VSL * QALY] = morbidity gain → morbidity gain + mortality gain = QALE gain) but instead of allowing the QALY to improve between 1900 and 2000 (as it did), this version of the QALE gain holds the QALY constant. The results shown as the morbidity and QALE gain (with no health improvements) represent the results of this calculation.

The appeal of calculating the aggregate QALE gain (with no health improvements), achieved through holding the QALY constant (at 1900 levels) is so that the difference between this calculation and the aggregate QALE gain (with health improvements)

[726] In Tale 9.4 the mortality gain and morbidity gain (with health improvements) and subsequent QALE gain (health improvements) are derived from earlier analysis in Table 9.2 and Appendix 12.14.1. See Appendix 12.14.3 for morbidity gain (with no heal improvements) QALE gain (no health improvements) calculations.

calculation can be estimated in an effort to identify the value of twentieth century health gains: the difference between these two results equals the value of twentieth century health improvements. In Table 9.4 the final result, 'value of health improvements' is the difference between the result in the previous two columns (i.e. 33,548 is the magnitude of the difference between -25,388 and 8,160).

Therefore, through holding the QALY aspects of the thesis methodology constant, Table 9.4 highlights the significance of the twentieth century gains in morbidity that has been solely a result of improvements in the health and welfare related quality of life. The result of this exercise provides significant results, where these health and welfare quality of life (or QALY) improvements were worth approximately 33,548 million (1990 international $) or 33.5 billion (1990 international $). This finding in Table 9.4 provides the single biggest evidence for the claims of the thesis: about the value of twentieth century improvements in health and the subsequent need to measure these far reaching developments.

9.5 Summary

The key objective of this extended results chapter was to calculate a lowest bound estimate that is (as far as possible) verified, in order to highlight the authenticity of the thesis claims. Hence, the author recognises that the figures derived here are not precise or even a close approximation about the actual twentieth century aggregate QALE gain (as this would be considerably higher, albeit unjustified). However, what is undeniable is the illustration that improvements in health in twentieth century England were substantial.

Therefore, the previous results reiterate the claims of the thesis about the value of improved health (mortality and morbidity) and the need to include this index in some form of economic development measure. Chapter 9 has substantiated these claims through extending the (thesis morbidity and QALE gain) results in order to indicate a lower bound estimate about the value of the twentieth century aggregate QALE gain. Hence, in this chapter the calculations made stringent assumptions which were applied to the existing thorough findings of the thesis evaluations about health, in order to justify the claims of the thesis. This process has also generated more universal and therefore meaningful findings, which fill an important void in the literature to date.

The key feature to note from the above analysis is the largely positive nature of the aggregate QALE gain (which embodies improvements in the prevalence and/or the burden

of morbidity and improvements in the burden of mortality) and the mandate this provides for adopting a fuller notion of health (rather than only gauging mortality improvements): both because of the need to more accurately propagate improvements in health (namely, infectious diseases) and also because of the need to better specify and understand the dynamics of twentieth century health improvements (i.e. the nature and magnitude of changes in health across all disease classes). This adds weight to the literature that has made similar claims (but with less evidence) about the need to measure health in some form of extended national income. For example, Usher claims that measures of economic development are seriously misleading without accounting for health improvements, wich have been empirically justified here: in simple terms, not accounting for health developments reduces economic development by nearly half (e.g. 1.4 percent versus 2.6 percent). The full implications of such extensive and valuable developments in the QALE will be highlighted in the following chapter: Conclusion.

Moreover, identifying and measuring more detailed aspects of health provide important contributions to the existing literature and debates about the twentieth century health transition in England. The trends in health identified here (i.e. the aggregate QALE gain which has been evaluated in Chapter 9) confirm the accuracy of the epidemiological transition and also provide evidence against the more pessimistic theories associated with the next stage of the epidemiological transition. For example, the 'failure of success' hypothesis (which is explained in detail in Chapter 2: Section 2.1.2.3, and essentially insinuates that the products of twentieth century medical developments have served more to prolong diseases and increase the proportion of morbidity in the economy, instead of diminishing disease and enriching life[727]) marks a contrast with the thesis findings about the existence and value associated with twentieth century health and welfare related quality of life (embodied in the QALY), which is shown in Table 9.4. The thesis has also provided evidence in support of the more optimistic theories (e.g. 'compression of morbidity', 'dynamic equilibrium' and to a lesser extent 'the fourth stage' of the epidemiological transition hypothesis[728]), which will be outlined in detail in the following chapter.

[727] Gruenberg, "The Failure of Success", p. 5
[728] For a detailed explanation of these theories see Part I: Chapter 2.1: Section 2.2.2.3: *Implications of the Epidemiological Transition in General*.

10. Conclusion

The thesis has highlighted both qualitatively and quantitatively that during the twentieth century the English population experienced far reaching improvements in health and that the quality of life implications of these improvements were substantial. In addition to creating a methodology capable of yielding a detailed spectrum of results about the value of improved health, the thesis has also filled a historical void, about the nature of health improvements and their contribution to twentieth century improvements in standards of living and more utility based economic development.

Throughout the thesis, health has been considered as a dual entity, comprising mortality and morbidity. The mortality component accounted for improvements in the death rate or increased life expectancy and the morbidity component accounted for improvements in the prevalence of disease and/or the burden (i.e. the health and welfare related QALY). These two health indices have been combined and simultaneously measured in the thesis QALE methodology in order to provide an accurate and comprehensive health measure which is capable of providing credible answers to the central questions of the thesis: what was the extent and value of improvements in health? And, what has been the impact of these health improvements upon standards of living and economic development in twentieth century England?

One of the thesis' most important contributions to knowledge is the developments of a representational health measurement methodology that can account for historical health improvements, from a health and welfare related quality of life perspective. Blindness, tuberculosis and cancer (breast and stomach) were evaluated in intricate qualitative detail and all aspects that were important for the health and welfare related quality of life were considered and gauged on the thesis' universal measurement spectrum (EuroQol). Once this had been achieved it was possible to derive QALY weights for different eras of the twentieth century for each of these illnesses, which were then utilised as the core morbidity component in the quantitative model. As well as providing a detailed justification for the QALY values, this enabled considerations about the extent to which health (mortality and morbidity, proxied by these three broad illnesses) had improved and the monetary value of these improvements

and the implications of such valuable contributions upon the entire burden of mortality and morbidity in twentieth century England.

Hence, the thesis has transformed the qualitative morbidity analyses into indices which were utilised for the foundation of the quantitative morbidity measurement. This was partnered with mortality in order to provide an original quantitative (monetary) measure of the contribution of improved health (mortality and morbidity) for the thesis illnesses (Chapter 8). These findings then formed the basis of calculations about the entire burden of health (aggregate QALE) in twentieth century England in order to provide conservative estimates about the value of improved health, which has been estimated as extremely valuable and significant in the thesis (Chapter 9). Moreover, the findings of the thesis highlight that even for disabilities and diseases that have experienced a sizeable increased prevalence rate during the twentieth century, there have been improvements related their prognosis, for example, although the calculations are very approximate, there seems to be improvements in the average length of duration of morbidity between 1900 and 2000. E.g. the blind prevalence rate increased substantially but when the approximate average years of blindness are estimated (16 years in 1900 versus 11 in 2000), the implications become much more positive[729]. Furthermore, the burden of all morbidity experienced an improvement in the health and welfare related quality of life for all illnesses in the thesis (highlighted by the 2000 QALY always being more favourable than its 1900 counterpart). This scenario is greatly exaggerated through considering diseases that have experienced an improved burden (QALY) and a decline in the prevalence rate, as the morbidity contribution of a declined prevalence rate and a reduced burden of illness provides very far-reaching contributions. For example, this has been highlighted with the tuberculosis considerations made throughout the thesis.

The findings of the thesis seem sensible, since mortality and morbidity improvements in twentieth century England have been substantial and health gains are valuable to individuals (which is highlighted by the magnitude of the VSL and VSHLY). The calculations of such valuable developments even at the lowest bound, provides considerable weight to claims for measuring health. Hence, the findings that, at a lower bound estimate, the value of twentieth century health improvements of 33,548 million (1990 international $) adds to a new view of

[729] See Chapter 8.2: Table 8.2.1.

the economics of health[730]. This new view is that improvements in health have been a major contributor to economic welfare in twentieth century England and have provided considerable additions to the growth of national income defined on a utility basis.

The above types of claim, which, towards the end of the twentieth century became increasingly more commonplace in the health and developmental literature, are reiterated by the findings of the thesis. The most notorious proponents of these claims and the associated contribution of the thesis to these claims are outlined below.

Usher (1980) propagated the need to measure health in some form of extended national income through claims that the growth of GNP alone significantly understates the extent to which current generations are better off than earlier generations[731]. The thesis has empirically justified Usher's claims through considering the contribution of the aggregate QALE gain to GDP per annum for the twentieth century, to highlight that, in simple terms, not accounting for twentieth century health (mortality and morbidity) developments reduces economic development by nearly half: GDP average growth for the twentieth century was 1.4 percent per annum versus 1.2 percent per annum for QALE (or health) gains, and a subsequent 'Adjusted Growth', which combines GDP growth with the aggregate QALE gains of 2.6 percent per annum[732].

Nordhaus (1999) has claimed that twentieth century improvements in health have provided a substantial contribution to standards of living, which was justified by his study about the WTP for mortality improvements[733]. The thesis has complimented Nordhaus' findings through considering the WTP for morbidity as well as mortality, which has facilitated a more comprehensive bolster for his claims (that were based solely on mortality gains). The most substantial justification that the thesis provides for these claims is the identified value of twentieth century improvements in health improvements, which is shown in Table 9.4, as being in excess of 33 billion 1990 international dollars.

[730] See Chapter 9: Table 9.4.
[731] Usher, "The Measure of Economic Growth", p. 228
[732] See Chapter 9: Table 9.3
[733] Nordhaus, "The Health of Nations: The Contribution of Improved Health to Living Standards", p. 21

Crafts (2005) also conducted a WTP mortality study which allowed him to illustrate that since 1870 increases in life expectancy in the UK have contributed a great deal to the growth of real national income per head defined on a utility basis[734]. The thesis has added weight to these findings through reiterating the WTP mortality gains that Crafts originally identified and also through extending these with original WTP morbidity gains. When these components are combined (WTP mortality + WTP morbidity) the QALE gain is calculated in order to provide a more rounded approximation of the contribution of increased life expectancy and improved health. The thesis has shown that the QALE gain has contributed even more (than mortality only) to the growth of real income per head defined on a utility basis. For example, Table 9.3 highlights that GDP or national income per annum compound average growth was 1.4 percent, the mortality gain contributed an additional 1.1 percent and the morbidity gain 0.1 percent, or the QALE gain (mortality gain + morbidity gain) contributed an additional 1.2 percent to national income (1.4 percent) when defined on a utility basis, which contributes to Crafts' original findings about the contribution of WTP mortality in twentieth century England.

Bradford DeLong (2000) has highlighted the astronomical income that would be necessary to compensate an individual enjoying their health in the year 2000 to return to the health conditions that were evident in 1900, and the chance that no amount of income could compensate for improved health[735]. The thesis has made provisional estimates about the value of improved health, which, given their magnitude –in excess of 33 billion 1990 international dollars – accord with the claims of Bradford DeLong[736].

Hence, the above types of claims, which, towards the end of the twentieth century became increasingly more commonplace in the health and developmental literature, are in tandem with the findings of the thesis and in places the findings of the thesis provide a valuable contribution for confirming the veracity of these theories. This is for two fundamental reasons: first, the thesis provides results about mortality which accord with the implications of the above claims, second, the thesis has gone a step further to provide an initial and, to date, the only existing estimates about the value of morbidity improvements, which provides an

[734] Crafts (2005) "The Contribution of Increased Life Expectancy to Growth of Living Standards in the UK, 1870-1998", p. 10. Retrieved 17 June 2005, from: www.york.ac.uk/res/wpeg/documents/crafts.pdf
[735] Bradford DeLong, "Cornucopia: The Pace of Economic Growth in the Twentieth Century", p. 22
[736] See Table 9.4 'value of improvements in health' result.

important contribution to existing measures and claims, which are generally based upon only mortality improvements.

The findings of the thesis also provide a more involved contribution to one of the most pertinent twentieth century health debates (which is more applicable to the later years of the century), which relates to the collection of theories that have evolved out of the epidemiological transition, which has generated a range of opinions and theories about: the meaning of current trends in health, the outlook for future scenarios, and the general nature of the link between mortality and morbidity (i.e. the 'optimists' versus the 'pessimists').

The (qualitative and quantitative) findings of the thesis provide evidence against the more pessimistic theories associated with the next stage of the epidemiological transition. Hence, 'the failure of success' hypothesis, which represents the pessimist school of thought, essentially contends that: the twentieth century products of medical developments have served more to prolong disease and increase the proportion of the population suffering from disabling and chronic illnesses instead of diminishing disease and enriching life[737]. I.e. for the pessimists, most noteworthy, Kramer (1980)[738] and Gruenberg (1977)[739], medical improvements have merely postponed death and not necessarily improved health and subsequently have prolonged disease and increase the portion of the population suffering from disabling and chronic illness[740]. The thesis highlights the shortcomings of such a theory, through the detailed qualitative analysis which has charted the evolution of improved prognosis and therapy regimes associated with illnesses and most importantly, a significantly improved quality of life (QALY) profile as a result of improved medical technology. The quantitative analysis of the thesis provides a vivid indication about the value of the improving QALY and the subsequent aggregate QALE, which has generated substantial results. Hence, although the pessimist claims about increased prevalence are sometimes correct (e.g. for disability and non-infectious illnesses), their theories and reasoning about the mechanisms that are perpetuating this trend and the result of this trend seem erroneously pessimistic.

[737] Gruenberg, "The Failure of Success", p. 5
[738] Kramer, "The Rising Pandemic of Mental Disorders and Associated Chronic Diseases and Disabilities"
[739] Gruenberg, "The Failure of Success"
[740] Gruenberg, "The Failure of Success", p. 5

Conclusion

A preferable set of theories for according with the findings of the thesis are the more optimistic schools of thought from this epidemiological debate, for example, the theory of 'dynamic equilibrium', which claims that in the long term chronic illness will be confined to the last few years of life so that the proportion of a healthy life will increase. The thesis analysis provides justification of this trend through estimates about the decreasing average number of years spent in blindness (shown quantitatively in Chapter 8.2: Table 8.2.1) and also the later average age of onset of breast cancer and stomach cancer as the twentieth century unfolded (shown qualitatively in Chapter 6: Table 6.4 to 6.7).

Along a similar vein, the theory of the 'compression of morbidity', which envisages the reduction in the national illness burden by postponing the age of onset of chronic infirmity relative to the average life duration, such that the period of morbidity is compressed between an increasing age of onset and a relatively fixed life expectancy also receives some agreement from the thesis[741]. Although this is better considered as a possible next stage, the thesis does show some evidence that this is possible (and perhaps still more viable than the pessimistic theories). This accordance is best highlighted by the compliance of the thesis results with the 'dynamic equilibrium' theory, which can be considered as a first stage of the more exaggerated 'compression of morbidity'. Hence, the thesis has shown a postponement in the average age of onset but this has not been as large as the postponement in the average age of mortality.

Therefore, the findings of the thesis provide additional support for the 'dynamic equilibrium' type hypothesis, which mark a stark contrast with the pessimistic literature and also, but to a much lesser extent the overly optimistic theories ('compression of morbidity and especially the 'fourth stage' hypothesis, which is an even more optimistic theory, outlined in Chapter 2.1: Section 2.1.2.3).

A final contribution of the thesis to contemporary debates and literature is the implications that the thesis' analysis and results have for the possible contribution of the NHS during the second half of the twentieth century. A consistent theme in the qualitative analysis has been the inefficiency of the NHS, most notoriously confirmed by the social class inequality in mortality

[741] Fries, "The Compression of Morbidity: Near or Far", p. 208

and (although in a more ad hoc fashion) morbidity, and that in many instances this has worsened to the disadvantage of the poorer social classes during the second half of the twentieth century. Given that one of the objectives of the NHS was to reduce this gradient and provide universal health care to the British population, this marks a serious shortcoming. This failure of the NHS is exacerbated by its under funding and comparative mediocrity compared to other developed economies. All of these problems contribute to creating an impression of an inefficient health service.

When the output of the NHS is evaluated a very different impression is generated. Current measures of national income or GDP only account for the inputs of the NHS, i.e. as a cost, and do not appraise any of the benefits, returns, or outputs of this spending. The quantitative analysis of the thesis and most precisely, the aggregate QALE gain 'Adjusted Growth' considerations (made in Table 9.3, where GDP growth is summed with aggregate QALE growth or health output growth) provide an estimate of the returns on this spending. When the magnitude of these QALE gains or health benefits are considered relative to GDP and the cost of achieving these gains, it would suggest that the image of an unproductive healthcare system is far off the mark.

It should be noted that not all QALE gains are attributable to the NHS. A suitable approximate would be that fifty percent of the documented QALE gain is attributable to the NHS[742]. Moreover, until a detailed comparative study of health services has been conducted, it is difficult to provide a substantiated appraisal of the NHS.

Nonetheless, the results of this thesis highlight a need to reconsider the healthcare system in an effort to quell the disquiet, which seems rather unfounded if the value and contribution of healthcare spending identified in the thesis is considered. Furthermore, the thesis has provided much detail that indicates the major contribution of technological medical improvements in the reduced mortality and particularly morbidity burden. For example, out of all the (EuroQol) variables considered in the thesis for their effect upon health related quality of life of sufferers, medical developments have provided the largest overall contribution to quality of life improvements. And for all the morbidity and mortality states considered in detail in the thesis,

[742] Cutler et al, "The Value of Medical Spending in the United States, 1960-2000", approximate 50 percent of health gains are due to medical spending. This provides a good estimate for which to consider the contribution of the NHS

the most significant health gains have tended to be during the second half of the twentieth century, after the introduction of the NHS (although this is less consistent for tuberculosis and especially blindness). This reinforces the value of the NHS output and the possible need to reconsider the value of the NHS.

In addition to the benefit of a more detailed historical account about twentieth century standards of living in England the contribution of the thesis is also important because of the initial developments it has made towards a more rounded and accurate health measure, which provides results that have not necessarily been predicted and would have been unidentifiable without the thesis QALE gain methodology. Most noteworthy is the point that: although the prevalence of certain disease groups has increase (namely, chronic, degenerative diseases) and, as a result of the nature of these illnesses and advancements in medical technology, there are more unhealthy life years associated with these later twentieth century illnesses, the quality of life gains associated with these illnesses have often outweighed the increase in their prevalence. Hence, the findings of the thesis not only provide a mandate for including improvements in mortality (previous measures of health) in some form of extended GDP measure, but they also illuminate an endorsement for including a morbidity index in conjunction with mortality.

Therefore, the thesis has provided an important contribution to knowledge regarding the history and value of health improvements. However, there are still numerous facets that could benefit from a more detailed and thorough analysis in order to provide an increasingly indicative estimate towards the actual value of improved health in twentieth century England. Many of these problems are generally insurmountable (and not just in the confines of the thesis), but still ought to be recognised.

The most obvious features for improvement are the methodological variables (QALY, VSL and VSHLY) that were subject to detailed sensitivity analyses. Although the thesis has highlighted that regardless of where in the range the value is selected, the contributions have still been noteworthy, it would be ideal to identify universally accepted values as this would lead to a more agreeable result about improved health.

Conclusion

Although verging on the impossible, it would be ideal to consider a much greater number of illnesses in the same detail as has been conducted as a major component of the thesis. The amount of detail is necessary and desirable in order to yield the most potentially accurate (QALY) results. Failing this, and more realistically, it should be possible to proxy and manipulate the results identified here, in order to provide an estimate of the effect for all illnesses in the epidemiological environment during the twentieth century, which is more accurate than the findings presented in the thesis' Extended Results. Both of these objectives are highly desirable because they would enable a full picture to be provided about morbidity. Additionally, it would be desirable to approximate the influence of co-morbidity for any set of QALE gain results. The thesis has provided a valuable first attempt and most conservative estimate, but it would be ideal to have a much more varied and far-reaching pool of morbidity indices.

Finally, in answering the two nucleus questions of the thesis: what has been the value of health improvements and what is the impact of these health improvements upon standards of living and economic development, the thesis has provided a valuable contribution to knowledge through the following key mechanisms. First, it has provided qualitative and quantitative historical detail. This detail has been utilised to provide a thorough and pragmatic indication about the contribution of improved health to standards of living and economic development in twentieth century England. Second, this has highlighted how valuable and far-reaching health improvements have been and subsequently added weight to existing claims about the need to include health (output) in the national accounts. Third, this thesis has added more optimistic and accurate detail to many of the debates associated with health. Fourth, the findings of the thesis add to the overall scholarship concerning the contribution of reduced illness and increase longevity towards economic gains. This reinforces the work of Nordhaus, Crafts and Hickson[743]. Lastly, the thesis also enhances these works, through making the same considerations, but for morbidity as well as mortality. This has served to reinforce these claims and, if anything strengthens existing arguments about the value of improved health and its far-reaching impact on standards of living and economic development. The findings of the thesis also add weight to claims about the need to consider national income measures that account for health utility.

[743] Nordhaus, "The Health of Nations: The Contribution of Improved Health to Living Standards". Crafts, "The Contribution of Increased Life Expectancy to Growth of Living Standards in the UK, 1870-1998". Hickson, "The Contribution of Improved Life Expectancy to Standards of Living in Twentieth Century Japan"

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12. Appendix

12.1: VSL and VSHLY Values

This Appendix relates to Tables 8.1 and 8.2 in the main thesis.

The VSL represents the value of a statistical life, a term that is fundamental in the methodology for estimating the willingness to pay or value of improved life expectancy. In order to estimate societies willingness to pay it is necessary to establish the amount that a group of people would be willing to pay for a reduction in the current period probability of death. VSL studies estimate the value of fatal risk reduction in the expectation of saving one life (of an unidentified person) in the current period, and therefore estimate the VSL and societies willingness to pay for improved mortality. For example, if people are on average willingness £10 for a safety improvement that will reduce their individual risk of death during the coming year by 1 in 100,000, this risk reduction would mean that on average, in group of 100,000 people there would be 1 fewer premature deaths. These 100,000 people would between them, be willing to pay £10 * 100,000 = £1,000,000 for the prevention of 1 statistical fatality. Hence in this example the VSL is £1,000,000.

The main body of the thesis (Chapter 3) has highlighted the mass of VSL studies and subsequent wide range of estimates of the value of death averted. The thesis has adopted one of the most plausible VSL results that was generated by Miller's detailed statistical analysis and endeavour to identify a best estimate VSL, based on applying this detailed statistical analysis to a range of most credible UK VSL studies.

As well as providing a single VSL estimate the Miller study has also generated associated VSL multiples: a VSL index number (101, 128 and 154, representing a low, mid and high VSL multiple estimate) that is then multiplied with the corresponding GDP per capita value (which is GDP per capita for the mid-point of the period under consideration). This is the approach used here.

After the VSL has been identified, which will be achieved in the thesis through applying the low, mid, and high VSL multiple, this multiple (101, 128, and 154) will multiplied with mid-point GDP per capita for the periods: 1900-1925 (1913 GDP per capita), 1925-1950 (1938 GDP per capita), 1950-1975 (1963 GDP per capita), 1975-2000 (1988 GDP per capita) and 1900-2000 (1950 GDP per capita). Once this process has been conducted the (low, mid, and high) VSL will have been identified for the above eras that the thesis is considering.

This VSL has two primary uses in the thesis methodology: i) to provide a valuation for the decline in mortality (i.e. the change in the mortality burden * VSL = the value of improved mortality or the mortality gain) and, ii) to provide part of the VSHLY.

The VSHLY provides the morbidity equivalent of the VSL, where the VSHLY values the improvement in the burden of illness or the morbidity burden. The VSHLY represents a more tenuous index than the VSL, as the thesis has approximated the VSHLY through combining the VSL and the QALY (VSL*QALY=VSHLY). The QALY was derived from the detailed analysis of the thesis and is illness and era specific. The results for the range of QALYs derived for the thesis illnesses and eras are outlined below in Appendix 12.6.

The result for this VSHLY (as well as the VSL) is shown below, where the range of low, mid and high VSHLY is considered for the thesis morbidity states and eras.

Hence, Appendix 12.1.1 considers the mortality gain (the change in the mortality burden*VSL) that is yielded from different VSL (multiple and era) values. The VSL is derived by multiplying the VSL multiple (identified by Miller) with GDP per capita for the mid point of the period under consideration.

The thesis utilises three VSL multiples: 128*mid point GDP which is used as the central or 'Mid' estimate of the VSL. In the sensitivity analysis the thesis considers a VSL multiple that is lower: 101*mid point GDP, which is referred to as 'Low' and a VSL multiple that is higher: 154*mid point GDP, which is referred to as 'High'.

The VSHLY values have been derived through considering the VSL value in conjunction with the QALY in order to account for the health adjustment for the according illness and era. This has been conducted for the thesis diseases (Appendix 12.1.1) and disability (Appendix 12.1.2). The VSHLY will also be considered with a 'Low' and 'High' VSL estimate, as well as 'Mid'.

Sources

GDP data

- 1900 and 1925: Maddison, "Monitoring the World Economy 1820-1992".
- 1950 and 1975 and 2000: Maddison, "The World Economy: A Millennial Perspective"

Maddison presents these historical GDP estimates in 1990 international $ (and therefore the subsequent VSL in the thesis is represented in this currency).

VSL

- The VSL (multiples) data comes from Miller, "Variations between Countries in Values of Statistical Life".

QALY (for use in the VSHLY)

- The QALY data comes from the thesis, the underlying rational is presented in Part II and the summary results are presented in Chapter 7 and summarised at the beginning of Chapter 8. This data is also summarised in Appendix 12.6.

12.1.1: VSL and VSHLY Values: Disease: Mortality

VSL Values

Low VSL = 101 * Midpoint GDP pc

Period	VSL multiple	Midpoint	GDP pc at Midpoint	VSL	**VSL (Millions)**
1900-1925	101	1913	5032	508232	**0.51**
1925-1950	101	1938	5983	604283	**0.60**
1950-1975	101	1963	9070	916070	**0.92**
1975-2000	101	1988	15988	1614788	**1.61**
1900-2000	101	1950	6907	697607	**0.70**

Mid VSL = 128 * Midpoint GDP pc

Period	VSL multiple	Midpoint	GDP pc at Midpoint	VSL	**VSL (Millions)**
1900-1925	128	1913	5032	644096	**0.64**
1925-1950	128	1938	5983	765824	**0.77**
1950-1975	128	1963	9070	1160960	**1.16**
1975-2000	128	1988	15988	2046464	**2.05**
1900-2000	128	1950	6907	884096	**0.88**

High VSL = 154 * Midpoint GDP pc

Period	VSL multiple	Midpoint	GDP pc at Midpoint	VSL	**VSL (Millions)**
1900-1925	154	1913	5032	774928	**0.77**
1925-1950	154	1938	5983	921382	**0.92**
1950-1975	154	1963	9070	1396780	**1.40**
1975-2000	154	1988	15988	2462152	**2.46**
1900-2000	154	1950	6907	1063678	**1.06**

VSHLY (VSL*QALY) Values
Breast Cancer

QALY

Year	Value	Period	Value
1900	0.3333	1900-1925	0.3333
1925	0.3333	1925-1950	0.4167
1950	0.5000	1950-1975	0.5834
1975	0.6667	1975-2000	0.7485
2000	0.8333	1900-2000	0.5833

Low VSHLY = (101 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.51	0.3333	**0.17**
1925-1950	0.60	0.4167	**0.25**
1950-1975	0.92	0.5834	**0.53**
1975-2000	1.61	0.7485	**1.21**
1900-2000	0.70	0.5833	**0.41**

Mid VSHLY = (128 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.64	0.3333	**0.21**
1925-1950	0.77	0.4167	**0.32**
1950-1975	1.16	0.5834	**0.68**
1975-2000	2.05	0.7485	**1.53**
1900-2000	0.88	0.5833	**0.51**

High VSHLY = (154 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.77	0.3333	**0.26**
1925-1950	0.92	0.4167	**0.38**
1950-1975	1.40	0.5834	**0.81**
1975-2000	2.46	0.7485	**1.84**
1900-2000	1.06	0.5833	**0.62**

VSHLY (VSL*QALY) Values
Stomach Cancer

QALY:

Year	Value	Period	Value
1900	0.3333	1900-1925	0.3333
1925	0.3333	1925-1950	0.4167
1950	0.5000	1950-1975	0.5834
1975	0.6667	1975-2000	0.6667
2000	0.6667	1900-2000	0.5000

Low VSHLY = (101 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.51	0.3333	**0.17**
1925-1950	0.60	0.4167	**0.25**
1950-1975	0.92	0.5834	**0.53**
1975-2000	1.61	0.6667	**1.08**
1900-2000	0.70	0.5000	**0.35**

Mid VSHLY = (128 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.64	0.3333	**0.21**
1925-1950	0.77	0.4167	**0.32**
1950-1975	1.16	0.5834	**0.68**
1975-2000	2.05	0.6667	**1.36**
1900-2000	0.88	0.5000	**0.44**

High VSHLY = (154 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.77	0.3333	**0.26**
1925-1950	0.92	0.4167	**0.38**
1950-1975	1.40	0.5834	**0.81**
1975-2000	2.46	0.6667	**1.64**
1900-2000	1.06	0.5000	**0.53**

12.1.1: VSL and VSHLY Values: Disease: Tuberculosis

VSHLY (VSL*QALY) Values
Tuberculosis

QALY:

Year	Value	Period	Value
1900	0.3333	1900-1925	0.4167
1925	0.5000	1925-1950	0.5834
1950	0.6667	1950-1975	0.7485
1975	0.8333	1975-2000	0.8333
2000	0.8333	1900-2000	0.5833

Low VSHLY = (101 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.51	0.4167	**0.21**
1925-1950	0.60	0.5834	**0.35**
1950-1975	0.92	0.7485	**0.69**
1975-2000	1.61	0.8333	**1.35**
1900-2000	0.70	0.5833	**0.41**

Mid VSHLY = (128 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.64	0.4167	**0.27**
1925-1950	0.77	0.5834	**0.45**
1950-1975	1.16	0.7485	**0.87**
1975-2000	2.05	0.8333	**1.71**
1900-2000	0.88	0.5833	**0.51**

High VSHLY = (154 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
1900-1925	0.77	0.4167	**0.32**
1925-1950	0.92	0.5834	**0.54**
1950-1975	1.40	0.7485	**1.05**
1975-2000	2.46	0.8333	**2.05**
1900-2000	1.06	0.5833	**0.62**

12.1.2: VSL and VSHLY Values: Disability

VSHLY (VSL*QALY) Values
BLIND

QALY:

Year	Value	Period	QALY
1900	0.3333	1900-1925	0.4167
1925	0.5000	1925-1950	0.5834
1950	0.6667	1950-1975	0.6667
1975	0.6667	1975-2000	0.6667
2000	0.6667	1900-2000	0.5000

Low VSHLY = (101 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)VSL Low VSHLY

Period	**VSL (millions) LOW**	QALY	**VSHLY (millions) LOW**
1900-1925	**0.51**	0.4167	**0.21**
1925-1950	**0.60**	0.5834	**0.35**
1950-1975	**0.92**	0.6667	**0.61**
1975-2000	**1.61**	0.6667	**1.08**
1900-2000	**0.70**	0.5000	**0.35**

Mid VSHLY = (128 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	**VSL (millions) MID**	QALY	**VSHLY (millions) MID**
1900-1925	**0.64**	0.4167	**0.27**
1925-1950	**0.77**	0.5834	**0.45**
1950-1975	**1.16**	0.6667	**0.77**
1975-2000	**2.05**	0.6667	**1.36**
1900-2000	**0.88**	0.5000	**0.44**

High VSHLY = (154 * Midpoint GDP pc)* ((T1QALY+T2QALY)/2)

Period	**VSL (millions) HIGH**	QALY	**VSHLY (millions) HIGH**
1900-1925	**0.77**	0.4167	**0.32**
1925-1950	**0.92**	0.5834	**0.54**
1950-1975	**1.40**	0.6667	**0.93**
1975-2000	**2.46**	0.6667	**1.64**
1900-2000	**1.06**	0.5000	**0.53**

12.2: Prevalence, QALY and Morbidity Burden: Disease

This Appendix relates to Table 8.1.1 in the main thesis.

It was necessary to estimate the prevalence of all thesis illnesses for the purpose of the thesis QALE methodology. This is because the burden of illness or the morbidity burden is a function of both how many life years were affected by illness (= prevalence) and also how severely these life years were affected by illness (= QALY). Hence the morbidity burden = prevalence * QALY.

The prevalence of morbidity is approximated through estimating the number of people in the economy with the referenced diseases for any one year. This is a measure that is not readily available in the data for any of the thesis morbidity states.

Therefore it was necessary to estimate the prevalence of morbidity for the key eras considered by the thesis: 1900, 1925, 1950, 1975, and 2000. The data needed to make these morbidity prevalence approximations is: i) a survival rate and, ii) the death rate. This data has been derived and in some case approximated (see below) from the illness specific information in Part II of the thesis. Due to unique idiosyncrasies and illness specific limitations in the data, these calculations were achieved in a slightly different way for each illness in the thesis.

Breast Cancer: Prevalence data only exists for the 1970s onwards and therefore it was necessary to estimate a prevalence figure for the period 1900 to 1970. This process:

1. Assumes 1945 1 year survival rate (1 YSR) for all years preceding 1945.
2. For years after and including 1945, the actual 1YSR for the according year has been used.

The prevalence is calculated through utilising the 1 YSR to identify the percentage of breast cancer deaths (which is the inverse of survival). The mortality data is then utilised to identify the number of deaths that the I YSR percentage of deaths relates to. Once this has been achieved it is possible to calculate the number of survivors (through establishing the number of deaths that the inverse of the survival rate relates to it is possible to subsequently identify the number of survivors represented by the I YSR. Both of these numbers are then summed (due to the inherent assumption that breast cancer mortality for any one year represents prevalence for that year [as an individual needed to first suffer from breast cancer to die of it] and likewise for those who survived from breast cancer.

This was estimated for the age groups utilised in later sections of the thesis quantitative analysis (see Appendix 12.3), which was necessary to facilitate a more detailed analysis and ultimate QALE gain result in the thesis, which is estimated by using age specific prevalence.

Stomach Cancer: The prevalence for stomach cancer was calculated in exactly the same way as for breast cancer. The nature of cancer data is the same for all cancers, especially from the 1970s onward when the national cancer registry was implemented, which is responsible for collating and compiling national cancer data in the UK.

Tuberculosis: Prevalence data exists but only as an aggregate for all ages. Because of the age specific analysis in the thesis it was necessary to identify the age specific prevalence of tuberculosis. This was estimated through identifying the age specific mortality data and combining this with the aggregate prevalence data in order to identify a survival rate for each age group, which could then be applied to the mortality data (in a similar way as for breast and stomach cancer) in order to estimate the age specific prevalence of tuberculosis. Hence, this process:

12.2: Prevalence, QALY and Morbidity Burden: Disease

1. Identify the relationship between notification/prevalence and total deaths which provided an estimate for the survival rate.
2. This rate was then applied to the existing death data for each age group.
3. The 1900 approximate for tuberculosis is based on the 1900 mortality data and the 1915 tuberculosis notification (as the 1900 data does not exist in this format).

When estimating the prevalence for all of the thesis diseases an equal survival rate across all age groups has been assumed. It should be noted that this is not likely to be the case in actuality but there is no alternative due to data limitations. I.e. aggregate prevalence did not exist for cancer before 1970 and for tuberculosis there is no official data about the age specific prevalence or survival rate. However, it is also important to note that this data problem does not affect the aggregate results.

It was also necessary to estimate a prevalence rate for the disability: blindness. This has been conducted through a different methodology (due to the differences in the data and is explained in Chapter 8.2 and Appendix 12.13.1).

Once the prevalence has been identified it is possible to combine this data with the QALY in order to estimate the morbidity burden. Hence, prevalence * QALY = morbidity burden. This is calculated here.

Sources

Survival rate data for cancer:

- 1945: Parliamentary Papers, "Cancer Registration in England and Wales"
- 1966-1971: Office of National Statistics, "Cancer Trends in England and Wales 1971"
- 1971-1990: Coleman, "Cancer Survival Trends in England and Wales, 1971-1995: Deprivation and NHS Region"
- 1998-2000: National Statistics Online, "Cancer Survival: Rates Improved during 1996-2001". Retrieved 24 November 2005, from**:**http://www.statistics.gov.uk/cci/nugget_print.asp?ID=861

Prevalence data for tuberculosis:

- 1920 to 1980: Citron & Raynes & Berrie, "Tuberculosis Today"
- 1990: Watson & Fern & Whitmore, "Notifications of Tuberculosis in England and Wales, 1982-1989"
- 1992 and 2000: Joint Tuberculosis Committee of the British Thoracic Society, "Control and Prevention of Tuberculosis in the UK: Code of Practice 2000"

Mortality data for cancer and tuberculosis:

- Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

QALY

- The QALY data comes from the thesis, the underlying rational is presented in Part II and the summary results are presented in Chapter 7 and summarised at the beginning of Chapter 8. This data is also summarised in Appendix 12.6.

BREAST CANCER

Year	Survival	Deaths
	PERCENT	PRECENT
1900	80	20
1925	80	20
1950	87	13
1975	83	17
2000	87	13

1901 (1911)

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.6667	**0**
5-9	0	0.6667	**0**
10-14	0	0.6667	**0**
15-19	5	0.6667	**3**
20-24	15	0.6667	**10**
25-34	290	0.6667	**193**
35-44	2340	0.6667	**1560**
45-54	4315	0.6667	**2877**
55-64	4295	0.6667	**2864**
65-74	3685	0.6667	**2457**
75+	1035	0.6667	**690**
	15980		

1925

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.6667	**0**
5-9	0	0.6667	**0**
10-14	0	0.6667	**0**
15-19	0	0.6667	**0**
20-24	10	0.6667	**7**
25-34	465	0.6667	**310**
35-44	2955	0.6667	**1970**
45-54	6820	0.6667	**4548**
55-64	6835	0.6667	**4558**
65-74	5725	0.6667	**3817**
75+	4245	0.6667	**2831**
	27055		

1950

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.5000	**0**
5-9	0	0.5000	**0**
10-14	0	0.5000	**0**
15-19	0	0.5000	**0**
20-24	38	0.5000	**19**
25-34	785	0.5000	**392**
35-44	5677	0.5000	**2840**
45-54	12485	0.5000	**6245**
55-64	14969	0.5000	**7488**
65-74	15046	0.5000	**7526**
75+	12477	0.5000	**6241**
	61477		

1975

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.3333	**0**
5-9	0	0.3333	**0**
10-14	0	0.3333	**0**
15-19	0	0.3333	**0**
20-24	41	0.3333	**14**
25-34	735	0.3333	**245**
35-44	4188	0.3333	**1397**
45-54	12006	0.3333	**4005**
55-64	16153	0.3333	**5389**
65-74	18159	0.3333	**6058**
75+	17653	0.3333	**5889**
	68935		

2000

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.1667	**0**
5-9	0	0.1667	**0**
10-14	0	0.1667	**0**
15-19	0	0.1667	**0**
20-24	38	0.1667	**6**
25-34	1108	0.1667	**185**
35-44	4838	0.1667	**808**
45-54	11931	0.1667	**1992**
55-64	15323	0.1667	**2559**
65-74	17831	0.1667	**2978**
75+	36846	0.1667	**6153**
	87915		

STOMACH CANCER

Year	Survival	Deaths
	PERCENT	PRECENT
1900	15	85
1925	15	85
1950	15	85
1975	15	85
2000	25	75

1901 (1911)

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.6667	**0**
5-9	0	0.6667	**0**
10-14	0	0.6667	**0**
15-19	4	0.6667	**3**
20-24	9	0.6667	**6**
25-34	108	0.6667	**72**
35-44	517	0.6667	**345**
45-54	1385	0.6667	**924**
55-64	2378	0.6667	**1586**
65-74	2552	0.6667	**1702**
75+	1036	0.6667	**691**
	7989		

1925

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.6667	**0**
5-9	0	0.6667	**0**
10-14	0	0.6667	**0**
15-19	4	0.6667	**3**
20-24	9	0.6667	**6**
25-34	110	0.6667	**73**
35-44	575	0.6667	**383**
45-54	1758	0.6667	**1172**
55-64	3128	0.6667	**2086**
65-74	3395	0.6667	**2264**
75+	1533	0.6667	**1022**
	10512		

1950

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.5000	**0**
5-9	0	0.5000	**0**
10-14	0	0.5000	**0**
15-19	3	0.5000	**2**
20-24	11	0.5000	**6**
25-34	112	0.5000	**56**
35-44	700	0.5000	**350**
45-54	1808	0.5000	**904**
55-64	3891	0.5000	**1946**
65-74	5796	0.5000	**2899**
75+	4979	0.5000	**2490**
	17300		

1975

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.3333	**0**
5-9	0	0.3333	**0**
10-14	0	0.3333	**0**
15-19	1	0.3333	**0.4**
20-24	7	0.3333	**2**
25-34	41	0.3333	**14**
35-44	162	0.3333	**54**
45-54	865	0.3333	**288**
55-64	2533	0.3333	**845**
65-74	5201	0.3333	**1735**
75+	5287	0.3333	**1764**
	14097		

2000

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	0	0.3333	**0**
5-9	0	0.3333	**0**
10-14	0	0.3333	**0**
15-19	1	0.3333	**0.4**
20-24	3	0.3333	**1**
25-34	25	0.3333	**8**
35-44	105	0.3333	**35**
45-54	317	0.3333	**106**
55-64	868	0.3333	**290**
65-74	2244	0.3333	**749**
75+	4141	0.3333	**1382**
	7704		

TUBERCULOSIS

Year	Survival	Deaths
	PERCENT	PRECENT
1900	85	15
1925	30	70
1950	62	38
1975	82	18
2000	94	6

1901 (1911)

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	38160	0.6667	**25445**
5-9	9933	0.6667	**6624**
10-14	6220	0.6667	**4147**
15-19	4547	0.6667	**3032**
20-24	2680	0.6667	**1787**
25-34	3633	0.6667	**2423**
35-44	2853	0.6667	**1903**
45-54	1693	0.6667	**1129**
55-64	1420	0.6667	**947**
65-74	573	0.6667	**382**
75+	247	0.6667	**164**
	71959		

1925

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	5026	0.5000	**2513**
5-9	1664	0.5000	**832**
10-14	2130	0.5000	**1065**
15-19	5821	0.5000	**2911**
20-24	7268	0.5000	**3634**
25-34	12102	0.5000	**6051**
35-44	9985	0.5000	**4993**
45-54	8762	0.5000	**4381**
55-64	2545	0.5000	**1273**
65-74	1932	0.5000	**966**
75+	370	0.5000	**185**
	57605		

1950

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	1618	0.3333	**544**
5-9	453	0.3333	**152**
10-14	321	0.3333	**108**
15-19	1424	0.3333	**478**
20-24	2924	0.3333	**982**
25-34	7576	0.3333	**2546**
35-44	6747	0.3333	**2267**
45-54	7924	0.3333	**2662**
55-64	7289	0.3333	**2449**
65-74	4582	0.3333	**1539**
75+	1166	0.3333	**392**
	42024		

1975

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	41	0.1667	**7**
5-9	17	0.1667	**3**
10-14	0	0.1667	**0**
15-19	39	0.1667	**6**
20-24	67	0.1667	**11**
25-34	100	0.1667	**17**
35-44	256	0.1667	**43**
45-54	1028	0.1667	**172**
55-64	1544	0.1667	**258**
65-74	2194	0.1667	**366**
75+	1833	0.1667	**306**
	7119		

2000

Age	Prevalence	(1-QALY)	**BURDEN**
0-4	17	0.1667	**3**
5-9	17	0.1667	**3**
10-14	17	0.1667	**3**
15-19	0	0.1667	**0**
20-24	33	0.1667	**6**
25-34	80	0.1667	**13**
35-44	300	0.1667	**50**
45-54	283	0.1667	**47**
55-64	750	0.1667	**125**
65-74	1517	0.1667	**253**
75+	3017	0.1667	**504**
	6031		

12.3: Changes in Mortality and Morbidity Burden

This Appendix relates to Tables 8.1.2.i, 8.1.2.ii, 8.1.3, 8.1.4, 8.1.5, and 8.1.6 in the main thesis.

The change in the mortality and morbidity burden is identified for the age specific burden of mortality and morbidity, i.e. the mortality and morbidity burdens are considered relative to the size of the age group in the era that they relate to. This provides a mortality or morbidity burden rate. The appeal of identifying and utilising such an index is that the mortality or morbidity considerations are standardised to a population and are therefore more meaningful and accurate, which provides a more indicative measure of the change in the burden of mortality or morbidity. This rate will be reversed for the morbidity gain once the calculations have been completed, i.e. the final stage of the morbidity burden change calculation below, 'Actual Morbidity Gain', which converts the morbidity burden back into an actual number. This is desirable as it i) facilitates a more detailed considerations of the change in the morbidity rate and, ii) converts the change in the morbidity burden into the same format as the other (disability and Chapter 9) morbidity burden used in the thesis that lack the data which is necessary to make the type of morbidity rate calculations that are contained here.

The first stage in identifying the change in the mortality or morbidity burden is to identify the rate of mortality or morbidity within the population. This is achieved by dividing the mortality or morbidity burden by the number of population for each age group. This provides an age specific mortality or morbidity burden index. Presented here as the mortality or morbidity burden per 1000.

After this has been achieved the mortality or morbidity burden change will be compared across different eras (and the correspondingly different populations and mortality / morbidity profiles and mortality / morbidity rates). This is achieved through weighting the change in the burden of morbidity to either the start [T1] or end point [T2] population between two eras, which is referred to as the weighted mortality / morbidity rate (WTD MR / WTD MBR). The calculation for the change in the mortality and morbidity rate is conducted for a WTS MR or WTD MBR to T1 and T2: once this has been achieved the average of these two results represents the change in the mortality or morbidity burden.

After this has been achieved the results for the morbidity gain are transformed into actual numbers, i.e. this is shown as 'morbidity burden change' for morbidity. This is achieved by achieved through transforming the morbidity burden from an age rate back into the actual morbidity burden. The 'mortality burden change' considers the mortality rate per million. This represents the mortality indices that are utilised in the main body of the thesis.

These considerations are made for mortality and all diseases morbidity (tuberculosis, breast cancer and stomach cancer) in the thesis for all of the thesis eras: 1900-1925, 1925-1950, 1950-1975, 1975-2000, and 1900-2000. Because of the density of these calculations, they are provided on the attached CD Rom. See Appendix 13.1 for all of these calculations. The thesis will provide an indication of these calculations here: mortality, breast and stomach cancer and tuberculosis for the period 1900 to 2000 are presented below. The reason that 1900-2000 have been selected to provide an example of the methodology contained in Appendix 13.1 is because 1900-2000 is the most referenced era throughout the thesis and especially in the case of breast cancer as this was used to represent a detailed worked example throughout Chapter 8.1.

12.3: Changes in Mortality and Morbidity Burden: Disease

It is noteworthy that these calculations are not made for the thesis disability because the necessary data (prevalence by age) does not exist and it is not possible to generate an accurate estimated about the disability prevalence by age trend. Therefore, blindness will be considered in aggregate which is achieved below in Appendix 12.13.1.

Sources:
See Appendix 13.1 for the full range of these calculations

Death and population data

- Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

Morbidity data

- See Appendix 12.2.

Mortality Burden Change
1900-2000

1901				**2000**			
Age	% Pop	DR/1000	WTD DR	Age	% Pop	DR/1000	WTD DR
0-1	0.02412	178.8	4.31263488	0-1	0.0116673	5.58	0.065103512
1-4	0.089948	20.83	1.8736212	1-4	0.04897759	0.23	0.011264847
5-9	0.107086	4.06	0.43476924	5-9	0.06477421	0.11	0.007125163
10-14	0.102585	2.34	0.24004802	10-14	0.06544862	0.14	0.009162806
15-19	0.099601	3.34	0.33266757	15-19	0.06106495	0.4	0.024425981
20-24	0.095741	4.24	0.40593988	20-24	0.05870452	0.55	0.032287484
25-34	0.161673	5.75	0.92962045	25-34	0.14564562	0.72	0.104864848
35-44	0.123151	9.62	1.18470887	35-44	0.14716016	1.3	0.191308202
45-54	0.089448	15.85	1.41775706	45-54	0.13229423	3.27	0.432602125
55-64	0.059846	29.79	1.78280893	55-64	0.10482839	8.4	0.880558489
65-74	0.033199	61.52	2.04242303	65-74	0.05592209	23.54	1.316406049
75-84	0.012103	129.8	1.5709525	75-84	0.05592209	60.79	3.399503981
85+	0.001499	257.81	0.38657152	85+	0.01939796	164.27	3.186503492

1900-2000 (T1)

Age	% Pop 1901	DR/1000 1901	WTD DR 1901&1901	% Pop 1901	DR/1000 2000	WTD DR 1901&2000	Decrease/1000	**Mortality Burden Change**
0-1	0.02412	178.8	4.31263488	0.02412	5.58	0.13458894	4.17804594	4178.04594
1-4	0.089948	20.83	1.8736212	0.089948	0.23	0.02068809	1.852933114	1852.933114
5-9	0.107086	4.06	0.43476924	0.107086	0.11	0.01177946	0.42298978	422.9897799
10-14	0.102585	2.34	0.24004802	0.102585	0.14	0.01436185	0.225686172	225.6861717
15-19	0.099601	3.34	0.33266757	0.099601	0.4	0.03984043	0.292827141	292.8271408
20-24	0.095741	4.24	0.40593988	0.095741	0.55	0.0526573	0.353282585	353.2825853
25-34	0.161673	5.75	0.92962045	0.161673	0.72	0.11640465	0.8132158	813.2158003
35-44	0.123151	9.62	1.18470887	0.123151	1.3	0.16009579	1.024613073	1024.613073
45-54	0.089448	15.85	1.41775706	0.089448	3.27	0.29249625	1.125260808	1125.260808
55-64	0.059846	29.79	1.78280893	0.059846	8.4	0.50270544	1.280103489	1280.103489
65-74	0.033199	61.52	2.04242303	0.033199	23.54	0.78151232	1.260910705	1260.910705
75-84	0.012103	129.8	1.5709525	0.012103	60.79	0.73573345	0.835219044	835.2190445
85+	0.001499	257.81	0.38657152	0.001499	164.27	0.24631358	0.140257941	140.2579411
							13.80534559	**13805.34559**

12.3: Changes in Mortality and Morbidity Burden: Mortality

1900-2000 (T2)

Age	% Pop 2000	DR/1000 1901	WTD DR 2000&1901	% Pop 2000	DR/1000 2000	WTD DR 2000&2000	Decrease/1000	**Mortality Burden Change**
0-1	0.011667	178.8	2.08611255	0.011667	5.58	0.06510351	2.021009033	2021.009033
1-4	0.048978	20.83	1.02020329	0.048978	0.23	0.01126485	1.00893844	1008.93844
5-9	0.064774	4.06	0.26298328	0.064774	0.11	0.00712516	0.25585812	255.8581195
10-14	0.065449	2.34	0.15314977	0.065449	0.14	0.00916281	0.143986959	143.9869588
15-19	0.061065	3.34	0.20395694	0.061065	0.4	0.02442598	0.179530957	179.5309574
20-24	0.058705	4.24	0.24890715	0.058705	0.55	0.03228748	0.216619663	216.6196635
25-34	0.145646	5.75	0.83746233	0.145646	0.72	0.10486485	0.732597481	732.5974812
35-44	0.14716	9.62	1.41568069	0.14716	1.3	0.1913082	1.22437249	1224.37249
45-54	0.132294	15.85	2.09686351	0.132294	3.27	0.43260212	1.664261386	1664.261386
55-64	0.104828	29.79	3.12283778	0.104828	8.4	0.88055849	2.242279294	2242.279294
65-74	0.055922	61.52	3.44032711	0.055922	23.54	1.31640605	2.123921059	2123.921059
75-84	0.055922	129.8	7.25868756	0.055922	60.79	3.39950398	3.859183578	3859.183578
85+	0.019398	257.81	5.00098901	0.019398	164.27	3.18650349	1.814485521	1814.485521
							17.48704398	**17487.04398**

12.3: Changes in Mortality and Morbidity Burden: Disease: Breast Cancer

Morbidity Burden Change
Breast Cancer
1900-2000

1900

Age	% Pop	MBR per 1000
0-4	0.114068	0
5-9	0.107086	0
10-14	0.102585	0
15-19	0.099601	0.0009236
20-24	0.095741	0.0032028
25-34	0.161673	0.036605
35-44	0.123151	0.3884269
45-54	0.089448	0.9862535
55-64	0.059846	1.4674386
65-74	0.033199	2.2693267
75+	0.013602	1.5554554

2000

Age	% Pop	MBR per 1000
0-4	0.0606449	0
5-9	0.0647742	0
10-14	0.0654486	0
15-19	0.061065	0
20-24	0.0587045	0.001969409
25-34	0.1456456	0.024475432
35-44	0.1471602	0.105797936
45-54	0.1322942	0.290137932
55-64	0.1048284	0.470378472
65-74	0.0559221	0.682198245
75+	0.0753201	1.57410013

1900-2000 (T1)

Age	% Pop 1900	MBR per 1000 1900	WTD MBR 1900&1900	% Pop 1900	MBR/1000 2000	WTD MBR 1900&2000	Decrease per 1000	**Morbidity Burden Change**
0-4	0.114068	0	0	0.114068	0	0	0	**0**
5-9	0.107086	0	0	0.107086	0	0	0	**0**
10-14	0.102585	0	0	0.102585	0	0	0	**0**
15-19	0.099601	0.0009236	9.199E-05	0.099601	0	0	9.19904E-05	**3.887021995**
20-24	0.095741	0.0032028	0.00030663	0.095741	0.00196941	0.0001886	0.000118082	**4.989531439**
25-34	0.161673	0.036605	0.00591805	0.161673	0.02447543	0.003957	0.001961029	**82.86260848**
35-44	0.123151	0.3884269	0.04783501	0.123151	0.10579794	0.0130291	0.034805926	**1470.712206**
45-54	0.089448	0.9862535	0.08821879	0.089448	0.29013793	0.0259524	0.062266418	**2631.045684**
55-64	0.059846	1.4674386	0.08782016	0.059846	0.47037847	0.0281502	0.059669949	**2521.332801**
65-74	0.033199	2.2693267	0.07534013	0.033199	0.68219825	0.0226485	0.052691607	**2226.465416**
75+	0.013602	1.5554554	0.02115779	0.013602	1.57410013	0.0214114	-0.000253612	**-10.71628367**
							0.211351389	**8931**

12.3: Changes in Mortality and Morbidity Burden: Disease: Breast Cancer

1900-2000 (T2)

Age	% Pop 2000	MBR/1000 1900	WTD MBR 2000&1900	% Pop 2000	MBR/1000 2000	WTD MBR 2000&2000	Decrease/1000	**Morbidity Burden Change**
0-4	0.060645	0	0	0.060645	0	0	0	**0**
5-9	0.064774	0	0	0.064774	0	0	0	**0**
10-14	0.065449	0	0	0.065449	0	0	0	**0**
15-19	0.061065	0.0009236	5.6399E-05	0.061065	0	0	5.63989E-05	**2.383115096**
20-24	0.058705	0.0032028	0.00018802	0.058705	0.00196941	0.0001156	7.24037E-05	**3.059393999**
25-34	0.145646	0.036605	0.00533136	0.145646	0.02447543	0.0035647	0.001766622	**74.64800639**
35-44	0.14716	0.3884269	0.05716096	0.14716	0.10579794	0.0155692	0.041591718	**1757.443481**
45-54	0.132294	0.9862535	0.13047564	0.132294	0.29013793	0.0383836	0.092092068	**3891.318088**
55-64	0.104828	1.4674386	0.15382923	0.104828	0.47037847	0.049309	0.104520214	**4416.465061**
65-74	0.055922	2.2693267	0.1269055	0.055922	0.68219825	0.03815	0.088755543	**3750.334394**
75+	0.07532	1.5554554	0.11715698	0.07532	1.57410013	0.1185613	-0.001404325	**-59.33925127**
							0.327450642	**13836**

12.3: Changes in Mortality and Morbidity Burden: Disease: Stomach Cancer

Morbidity Burden Change
Stomach Cancer
1900-2000

1900

Age	% Pop	MBR/1000
0-4	0.114068091	0
5-9	0.10708602	0
10-14	0.102584623	0
15-19	0.099601068	0.000923588
20-24	0.095740538	0.00192166
25-34	0.161673121	0.013655761
35-44	0.12315061	0.085902097
45-54	0.089448395	0.31675294
55-64	0.059845885	0.812624891
65-74	0.033199334	1.571995936
75+	0.013602313	1.557709648

2000

Age	% Pop	MBR/1000
0-4	0.06064489	0
5-9	0.06477421	0
10-14	0.06544862	0
15-19	0.06106495	0.000126219
20-24	0.05870452	0.000295411
25-34	0.14564562	0.001058397
35-44	0.14716016	0.004582831
45-54	0.13229423	0.015439067
55-64	0.10482839	0.053305884
65-74	0.05592209	0.171580418
75+	0.07532006	0.35355215

1900-2000 (T1)

Age	% Pop 1900	MBR/1000 1900	WTD MBR 1900&1900	% Pop 1900	MBR/1000 2000	WTD MBR 1900&2000	Decrease/1000	**Morbidity Burden Change**
0-4	0.114068091	0	0	0.1140681	0	0	0	**0**
5-9	0.10708602	0	0	0.107086	0	0	0	**0**
10-14	0.102584623	0	0	0.1025846	0	0	0	**0**
15-19	0.099601068	0.000923588	9.19904E-05	0.0996011	0.0001262	1.2572E-05	7.94189E-05	**3**
20-24	0.095740538	0.00192166	0.000183981	0.0957405	0.0002954	2.8283E-05	0.000155698	**7**
25-34	0.161673121	0.013655761	0.00220777	0.1616731	0.0010584	0.00017111	0.002036655	**86**
35-44	0.12315061	0.085902097	0.010578896	0.1231506	0.0045828	0.00056438	0.010014517	**423**
45-54	0.089448395	0.31675294	0.028333042	0.0894484	0.0154391	0.001381	0.026952042	**1139**
55-64	0.059845885	0.812624891	0.048632256	0.0598459	0.0533059	0.00319014	0.045442118	**1920**
65-74	0.033199334	1.571995936	0.052189218	0.0331993	0.1715804	0.00569636	0.046492862	**1965**
75+	0.013602313	1.557709648	0.021188455	0.0136023	0.3535522	0.00480913	0.016379327	**692**
							0.14755264	**6235**

12.3: Changes in Mortality and Morbidity Burden: Disease: Stomach Cancer

1900-2000 (T2) Age	% Pop 2000	MBR/1000 1900	WTD MBR 2000&1900	% Pop 2000	MBR/1000 2000	WTD MBR 2000&2000	Decrease/1000	**Morbidity Burden Change**
0-4	0.06064489	0	0	0.0606449	0	0	0	**0**
5-9	0.064774207	0	0	0.0647742	0	0	0	**0**
10-14	0.065448618	0	0	0.0654486	0	0	0	**0**
15-19	0.061064951	0.000923588	5.63989E-05	0.061065	0.0001262	7.7075E-06	4.86913E-05	**2**
20-24	0.058704516	0.00192166	0.00011281	0.0587045	0.0002954	1.7342E-05	9.54682E-05	**4**
25-34	0.145645622	0.013655761	0.001988902	0.1456456	0.0010584	0.00015415	0.001834751	**78**
35-44	0.147160155	0.085902097	0.012641366	0.1471602	0.0045828	0.00067441	0.011966956	**506**
45-54	0.132294228	0.31675294	0.041904586	0.1322942	0.0154391	0.0020425	0.039862086	**1684**
55-64	0.104828392	0.812624891	0.08518616	0.1048284	0.0533059	0.00558797	0.07959819	**3363**
65-74	0.055922092	1.571995936	0.087909302	0.0559221	0.1715804	0.00959514	0.078314166	**3309**
75+	0.075320056	1.557709648	0.117326778	0.0753201	0.3535522	0.02662957	0.09069721	**3832**
							0.302417518	**12779**

12.3: Changes in Mortality and Morbidity Burden: Disease: Tuberculosis

Morbidity Burden Change
Tuberculosis
1900-2000

1900

Age	% Pop	MBR/1000	WTD MBR
0-4	0.114068	6.8400538	0.78023188
5-9	0.107086	1.8967443	0.20311479
10-14	0.102585	1.2395755	0.12716139
15-19	0.099601	0.9334401	0.09297163
20-24	0.095741	0.5723345	0.05479561
25-34	0.161673	0.4595543	0.07429758
35-44	0.123151	0.473831	0.05835257
45-54	0.089448	0.3870282	0.03461905
55-64	0.059846	0.485218	0.0290383
65-74	0.033199	0.3528216	0.01171344
75+	0.013602	0.3697024	0.00502881

2000

Age	% Pop	MBR/1000
0-4	0.0606449	0.000953198
5-9	0.0647742	0.000892432
10-14	0.0654486	0.000883236
15-19	0.061065	0
20-24	0.0587045	0.001969409
25-34	0.1456456	0.001719895
35-44	0.1471602	0.006546902
45-54	0.1322942	0.006845624
55-64	0.1048284	0.022976674
65-74	0.0559221	0.057957071
75+	0.0753201	0.128936529

1900-2000 (T1)

Age	% Pop 1900	MBR/1000 1900	WTD MBR 1900&1900	% Pop 1900	MBR/1000 2000	WTD MBR 1900&2000	Decrease/1000	**Morbidity Burden Change**
0-4	0.114068	6.8400538	0.78023188	0.114068	0.0009532	0.0001087	0.780123148	**32964**
5-9	0.107086	1.8967443	0.20311479	0.107086	0.00089243	9.557E-05	0.203019228	**8579**
10-14	0.102585	1.2395755	0.12716139	0.102585	0.00088324	9.061E-05	0.127070785	**5369**
15-19	0.099601	0.9334401	0.09297163	0.099601	0	0	0.092971627	**3928**
20-24	0.095741	0.5723345	0.05479561	0.095741	0.00196941	0.0001886	0.05460706	**2307**
25-34	0.161673	0.4595543	0.07429758	0.161673	0.0017199	0.0002781	0.074019516	**3128**
35-44	0.123151	0.473831	0.05835257	0.123151	0.0065469	0.0008063	0.05754632	**2432**
45-54	0.089448	0.3870282	0.03461905	0.089448	0.00684562	0.0006123	0.034006722	**1437**
55-64	0.059846	0.485218	0.0290383	0.059846	0.02297667	0.0013751	0.027663242	**1169**
65-74	0.033199	0.3528216	0.01171344	0.033199	0.05795707	0.0019241	0.009789308	**414**
75+	0.013602	0.3697024	0.00502881	0.013602	0.12893653	0.0017538	0.003274973	**138**
							1.464091929	**61865**

12.3: Changes in Mortality and Morbidity Burden: Disease: Tuberculosis

1900-2000 (T2)								
Age	% Pop 2000	MBR/1000 1900	WTD MBR 2000&1900	% Pop 2000	MBR/1000 2000	WTD MBR 2000&2000	Decrease/1000	**Morbidity Burden Change**
0-4	0.060645	6.8400538	0.41481431	0.060645	0.0009532	5.781E-05	0.414756503	**17525**
5-9	0.064774	1.8967443	0.12286011	0.064774	0.00089243	5.781E-05	0.1228023	**5189**
10-14	0.065449	1.2395755	0.08112851	0.065449	0.00088324	5.781E-05	0.0810707	**3426**
15-19	0.061065	0.9334401	0.05700047	0.061065	0	0	0.057000472	**2409**
20-24	0.058705	0.5723345	0.03359862	0.058705	0.00196941	0.0001156	0.033483006	**1415**
25-34	0.145646	0.4595543	0.06693207	0.145646	0.0017199	0.0002505	0.066681576	**2818**
35-44	0.14716	0.473831	0.06972904	0.14716	0.0065469	0.0009634	0.068765598	**2906**
45-54	0.132294	0.3870282	0.0512016	0.132294	0.00684562	0.0009056	0.050295962	**2125**
55-64	0.104828	0.485218	0.05086462	0.104828	0.02297667	0.0024086	0.048456016	**2047**
65-74	0.055922	0.3528216	0.01973052	0.055922	0.05795707	0.0032411	0.016489444	**697**
75+	0.07532	0.3697024	0.02784601	0.07532	0.12893653	0.0097115	0.018134501	**766**
							0.977936079	**41322**

12.4: Valuation of Changes in Mortality and Morbidity Burden: Disease

This Appendix relates to Tables 8.1.3, 8.1.4, 8.1.5, and 8.1.6 in the main thesis.

The next stage in the thesis QALE methodology is to value the change in the mortality and morbidity burdens that have been identified in Appendix 12.3 (and in more detail in Appendix 13.1). This is achieved through identifying the average change in the morbidity burden (between the change weighted to T1 and T2) and then valuing this change. For the change in the mortality burden the VSL is used to value the change and estimate the mortality gain. The change in the burden of morbidity is valued by applying the VSHLY to the change, which yields the morbidity gain (the VSL and VSHLY values that are multiplied by the morbidity and mortality change are contained in Appendix 12.1).

Hence: the mortality gain = change in the burden of mortality * VSL. In the case below, the precise calculation = (change in morbidity burden 1 * VSL = mortality gain 1 and change in morbidity burden 2 * VSL = mortality gain 2) → (mortality gain 1 + mortality gain 2) / 2 = Average mortality gain. This process is the same for the morbidity gain (but using morbidity and the VSHLY instead of mortality and the VSL).

This process is necessary because of the weighting of the change in the burden of morbidity to either the start [T1] or end point [T2] population. Mortality and morbidity change 1 represents the change in mortality and morbidity fixed to T1 and the mortality and morbidity change 2 represents the result fixed to T2. Once these have both been applied to the VSL or VSHLY the corresponding mortality / morbidity gain is derived. This is then average to provide a single estimate, which is the mid-point between the results for the two populations.

The mortality and morbidity gains here utilised the mid VSL and VSHLY values.

Sources:
Mortality and morbidity burden change
- Appendix 12.3 (and in more detail in Appendix 13.1)

VSL and VSHLY values
- Appendix 12.1.1 and 12.1.2.

Summary Mortality Gain

Period	Mortality Burden Change 1	Mortality Burden Change 2	VSL millions	Mortality Gain 1 millions	Mortality Gain 2 millions	**Average Mortality Gain millions**
1900-1925	6201	5968	0.64	3969	3820	3895
1925-1950	4494	4974	0.76	3415	3780	3598
1950-1975	1906	2386	1.16	2211	2767	2489
1975-2000	3781	4034	2.05	7751	8270	8011
1900-2000	13805	17487	0.88	12149	15389	13769

Summary Morbidity Gain
BREAST CANCER

Period	Morbidity Burden Rate Change1	Morbidity Burden Rate Change2	VSHLY millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	**Average Morbidity Gain millions**
1900-1925	-868	-1000	0.21	-182	-210	-196
1925-1950	-2062	-2586	0.32	-660	-828	-741
1950-1975	13287	14952	0.68	9035	10167	9601
1975-2000	11092	11102	1.53	16971	16986	16979
1900-2000	8931	13836	0.51	4555	7056	5806

Summary Morbidity Gain
STOMACH CANCER

Period	Morbidity Burden Rate Change1	Morbidity Burden Rate Change2	VSHLY millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	**Average Morbidity Gain millions**
1900-1925	1116	1510	0.21	234	317	278
1925-1950	121	-164	0.32	39	-52	-7
1950-1975	5598	6611	0.68	3807	4495	4151
1975-2000	2678	2714	1.36	3642	3691	3677
1900-2000	6235	12779	0.44	2743	5623	4183

Summary Morbidity Gain
TUBERCULOSIS

Period	Morbidity Burden Rate Change1	Morbidity Burden Rate Change2	VSHLY millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	**Average Morbidity Gain millions**
1900-1925	28168	22021	0.27	7605	5946	6776
1925-1950	18728	17215	0.45	8428	7748	8088
1950-1975	13977	13556	0.87	12160	11794	19977
1975-2000	377	330	1.71	645	565	605
1900-2000	61865	41322	0.51	31551	21074	26313

12.5: Value of QALE Gain: Disease

This Appendix relates to Tables 8.1.7 in the main thesis.

The next stage in the thesis methodology is to combine the mortality gain and the morbidity gain in order to identify the QALE gain. This is achieved in two stages below. Stage one, which is the top series of data summarises the results generated above, where the mortality and morbidity gains here are the same as those presented as the average mortality and morbidity gain above in Appendix 12.4. The second stage provides the QALE gain, which is achieved by summing the mortality gain and the morbidity gain. This consideration is made for independent diseases below. Hence, it is possible to sum all diseases and mortality (which will be considered later in the Appendices) but for initial analysis purposes the mortality gain will be summed with each individual morbidity gain.

Sources:
Mortality and morbidity gains

- Calculated in Appendix 12.4

12.5: Value of QALE Gain: Disease

QALE Considerations

Period	Deaths Average Mortality Gain millions	Stomach Cancer Average Morbidity Gain millions	Breast Cancer Average Morbidity Gain millions	Tuberculosis Average Morbidity Gain millions
1900-1925	3895	278	-196	6776
1925-1950	3598	-7	-741	8088
1950-1975	2489	4151	9601	19977
1975-2000	8011	3677	16979	605
1900-2000	13769	4183	5806	26313

QALE Gain

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis
1900-1925	4173	3699	10670
1925-1950	3591	2857	11686
1950-1975	6640	12090	14466
1975-2000	11688	24990	8616
1900-2000	17952	19575	40082

12.6: Alternative QALY Weights

This Appendix relates to Tables 8.1 and 8.1.8 in the main thesis.

The QALY represents the index for the burden of morbidity. This QALY provides a standardised index, across all thesis illnesses and all thesis eras. The series of QALYs used in the thesis have been constructed by the detailed qualitative analysis in Part II of the thesis. This was achieved through considering the twentieth century health and welfare history of the thesis morbidity states and summarising this evolution on a standardised spectrum, which considers all illnesses and their level of development, namely, EuroQol. Once all thesis illnesses and eras had received a EuroQol rank (summarised in Chapter 7) these ranks were then converted into QALYs.

Although the QALY values utilised in the thesis and presented below are unique to the thesis, the general concept of a quality adjusted life year is not new. Economists have designed a series of econometric scaling techniques in an attempt to assign a numerical value to health status. These are known as utility ratings, of which the most famous is the QALY. QALYs have become increasingly used in the estimation of cost-effectiveness of different forms of medical intervention.

As well as making similar QALY considerations to the existing literature, the thesis QALY is also subject to a variety of contentions associated with trying to identify the burden of morbidity and summarise this in the QALY. A pertinent problem with the QALY is their potential to misrepresent consumer preferences. Many have highlighted that QALYs fail to accurately represent an individuals' preferences, and the high potential for bias due to the problems of framing effects influencing the results. Although these problems cannot be avoided, they will be recognised and accommodated by the methods of the thesis.

The QALY in this thesis is based upon a comprehensive analysis of the changing health and welfare conditions faced by illness sufferers as the twentieth century unfolded. Moreover, due to the subjective and often controversial nature of illness and welfare measurement, the estimates of the burden of disease in the thesis are presented as a range of possible values, these are shown below.

The key advantages of this approach are that is recognises and therefore to an extent overcomes many of the contentions associates with trying to determine QALY weights. Furthermore, because the results will be presented in a range (shown below for each illness and era considered in the thesis) they will provide a broader consideration about the contributions of improved health. Finally, the analysis that forms the basis of the QALY weights represents a significant proportion of the volume of the thesis, and therefore will be available for the reader to identify the evidence from which the conclusions (QALYs) have been drawn.

Hence, the QALYs presented below, for the illnesses (blindness, breast and stomach cancer and tuberculosis) and eras (1900, 1925, 1950, 1975, and 2000) considered in the thesis have been derived through a detailed qualitative account of the twentieth century health and welfare associated quality of life faced by sufferers of the illness in question. Furthermore, the thesis has created a foundational standardised measurement spectrum (EuroQol), which has generated a consistent account of the effects of the thesis morbidity states upon quality of life at different historical points during the twentieth century. After EuroQol was utilised to summarise all states of thesis morbidity for all eras considered in the thesis, these EuroQol ranks were converted into a QALY indices, which will represent

the changing (diminishing) burden of different illnesses upon quality of life. This will essentially provide the morbidity measure component of the thesis. The results of this process, the QALY indices, are shown below. These are represented by 'Mid'. The other QALY values shown below, 'Low' and 'High' represent the provision of a range of QALY estimate, the appeal of which has been outlined above.

This QALY has two primary uses in the thesis methodology: i) to provide a morbidity burden index (which is combined with the prevalence in order to estimate the morbidity burden) and, ii) to provide part of the VSHLY, which is used to value the change in the morbidity burden and estimate the morbidity gain (VSHLY = VSL * QALY) and (morbidity burden change * VSHLY = morbidity gain).

In the tables below the QALYs are presented in two formats: i) as a QALY for the years or periods the thesis has identified the quality of life through EuroQol (1900, 1925, 1950, 1975, and 2000) and, ii) as a QALY for the eras that the thesis applied the QALE methodology (1900-1925, 1925-1950, 1950-1975, 1975-2000, and 1900-2000). The detailed analysis in the main body of the thesis that has generated EuroQol ranks and subsequent QALY weights has conducted this for specific years. The QALE methodology used throughout Chapter 8 and 9 considers periods. These QALY values are converted into era estimates through identifying the mid value of the QALY between the start and end point of the era under consideration. This is shown below.

Appendix 12.6.1 presents the range of QALY weights for the thesis diseases: breast cancer, stomach cancer, tuberculosis. Appendix 12.6.2 presents the range of QALY weights for the thesis disability: blindness.

Sources:
QALY

- The QALY data comes from the thesis, the underlying rational is presented in Part II and the summary results are presented in Chapter 7 and summarised at the beginning of Chapter 8. This data is also summarised in Appendix 12.6.

12.6.1: Alternative QALY Weights: Disease

Breast Cancer

Low QALY

Period	QALY Value	Era	QALY Value
1900	0.1667	1900-1925	0.1667
1925	0.1667	1925-1950	0.2500
1950	0.3333	1950-1975	0.4167
1975	0.5000	1975-2000	0.5834
2000	0.6667	1900-2000	0.4167

Mid QALY

Period	QALY Value	Era	QALY Value
1900	0.3333	1900-1925	0.3333
1925	0.3333	1925-1950	0.4167
1950	0.5000	1950-1975	0.5834
1975	0.6667	1975-2000	0.7485
2000	0.8333	1900-2000	0.5833

High QALY

Period	QALY Value	Era	QALY Value
1900	0.5000	1900-1925	0.5000
1925	0.5000	1925-1950	0.5834
1950	0.6667	1950-1975	0.7500
1975	0.8333	1975-2000	0.9167
2000	1	1900-2000	0.7500

12.6.1: Alternative QALY Weights: Disease: Stomach Cancer

QALY Weights
Stomach Cancer

Low QALY

Period	QALY Value	Era	QALY Value
1900	0.1667	1900-1925	0.1667
1925	0.1667	1925-1950	0.2500
1950	0.3333	1950-1975	0.4167
1975	0.5000	1975-2000	0.5000
2000	0.5000	1900-2000	0.3333

Mid QALY

Period	QALY Value	Era	QALY Value
1900	0.3333	1900-1925	0.3333
1925	0.3333	1925-1950	0.4167
1950	0.5000	1950-1975	0.5834
1975	0.6667	1975-2000	0.6667
2000	0.6667	1900-2000	0.5000

High QALY

Period	QALY Value	Era	QALY Value
1900	0.5000	1900-1925	0.5000
1925	0.5000	1925-1950	0.5834
1950	0.6667	1950-1975	0.7500
1975	0.8333	1975-2000	0.8333
2000	0.8333	1900-2000	0.6667

12.6.1: Alternative QALY Weights: Disease: Tuberculosis

QALY Weights
Tuberculosis

Low QALY

Era	QALY Value	Era	QALY Value
1900	0.1667	1900-1925	0.2500
1925	0.3333	1925-1950	0.4167
1950	0.5000	1950-1975	0.5834
1975	0.6667	1975-2000	0.6667
2000	0.6667	1900-2000	0.4167

Mid QALY

Era	QALY Value	Era	QALY Value
1900	0.3333	1900-1925	0.4167
1925	0.5000	1925-1950	0.5834
1950	0.6667	1950-1975	0.7485
1975	0.8333	1975-2000	0.8333
2000	0.8333	1900-2000	0.5833

High QALY

Era	QALY Value	Era	QALY Value
1900	0.5000	1900-1925	0.5834
1925	0.6667	1925-1950	0.7500
1950	0.8333	1950-1975	0.9167
1975	1	1975-2000	1
2000	1	1900-2000	0.7500

12.6.2: Alternative QALY Weights: Disability

QALY Weights
Blind

Low QALY

Period		Era	QALY Value
1900	0.1667	1900-1925	0.2500
1925	0.3333	1925-1950	0.4167
1950	0.5000	1950-1975	0.5000
1975	0.5000	1975-2000	0.5000
2000	0.5000	1900-2000	0.3334

Mid QALY

Period		Era	QALY Value
1900	0.3333	1900-1925	0.4167
1925	0.5000	1925-1950	0.5834
1950	0.6667	1950-1975	0.6667
1975	0.6667	1975-2000	0.6667
2000	0.6667	1900-2000	0.5000

High QALY

Period		Era	QALY Value
1900	0.5000	1900-1925	0.5834
1925	0.6667	1925-1950	0.7500
1950	0.8333	1950-1975	0.8333
1975	0.8333	1975-2000	0.8333
2000	0.8333	1900-2000	0.6667

12.7: Value of QALE Gain for Alternative VSL and VSHLY and QALY Weights

This Appendix relates to Tables 8.1.9 and 8.1.10 in the main thesis.

This appendix combines the analysis of the previous Appendices in order to provide a range of QALE gain estimates. Appendix 12.1 considered the range (low, mid, high) of VSL and VSHLY values and Appendix 12.6 considered the range (low, mid, high) of QALY values for all of the thesis illnesses (diseases and disability) and eras. The analysis below will repeat the QALE gain calculation (shown in Appendix 12.4 and 12.5) but for all ranges and subsequent combinations of QALY, VSL and VSHLY weight, for all illnesses and eras.

Hence, the methodological process here is essentially the same as Appendix 12.5, but it applies the series (Low, Mid or actual, High) of variable weights in order to consider the full array of sensitivity analysis QALE gain results, instead of only considering the 'Mid' QALY, VSL and VSHLY estimates, which is the case in Appendix 12.5.

These results represent the foundation of the thesis sensitivity analysis. The desire of making this series of considerations is to provide the reader with the broadest (within the bounds of the thesis analysis) range of results from an exaggeratedly large margin of error. Hence, one of the ways in which the thesis aims to overcome issues of bias and variable selection is to provide the reader with the broadest possible range of estimates. This allows the reader to identify a QALE gain with an alternative (lower or higher) variable value, for the QALY, VSL, VSHLY, or all of these variables, in whichever combination (lower or higher or mid) the reader prefers.

The starting point of the QALE methodological variables has been outlined in detail in the main body of the thesis: the VSL and VSHLY were considered in Chapter 3 and the QALY is substantiated by the qualitative analysis in Part II of the thesis, which is summarised in Chapter7. For the reader who is still unsure of what they believe to be the most accurate weight, these sections should be reconsidered.

Hence, instead of only providing one estimate of the QALE gain for each illness and era, this analysis enables the thesis to provide twenty seven different estimates for each illness and era.

Additionally, this section also considers alternative VSL values that are selected from sources not within the thesis. Appendix 12.7.3 considers the VSL and VSHLY and subsequent QALE gain that is generated by utilising the VSL suggested by Costa and Viscusi. Costa suggests that the VSL over time has an income elasticity of 1.6, which basically means that as income rises, the value of life is increasing at 0.6 times the rate (1) of the income rise. An alternative view is that represented by Viscusi who contends that over time the VSL is income inelastic, such that the VSL is only increasing 0.6 times as much as income over time.

A final appeal of this exercise is to highlight that whichever weights for the QALY, VSL, and VSHLY are preferred by the reader, the overall QALE gain is still significant and still contributes to the same overall conclusions of the thesis. The full veracity of this claim is highlighted best in Appendix 13.2, which provides the full range of twenty seven estimates for each of the four morbidity states and five eras considered in the thesis. Because of the density of this analysis the majority of the results are contained in the CD-Rom Appendices. This Appendix will present the most extreme and mid point estimates, i.e. the

12.7: Value of QALE Gain for Alternative VSL and VSHLY and QALY Weights

QALE gain results using a low, mid, and high QALY, VSL and VSHLY. For all other variable weight combinations, see Appendix 13.2. Appendix 12.7 will also contain the alternative QALE gain for the Viscusi and Costa weights.

Sources:

QALY

- The QALY data comes from the thesis, the underlying rational is presented in Part II and the summary results are presented in Chapter 7 and summarised at the beginning of Chapter 8. This data is also summarised in Appendix 12.6.

VSL

The VSL (multiples) data for the thesis sensitivity analysis, which considers a low, mid, high VSL multiple values, comes from:

- Miller, "Variations between Countries in Values of Statistical Life"

The alternative VSL for different income elasticity comes from:

- Costa & Kahn, "Changes in the Value of a Statistical Life, 1940-1980"
- Viscusi & Aldy, "The Value of a Statistical Life: A Critical Review of Market Estimates Throughout The World"

QALE methodology

- Uses the same as the more detailed methodological outline in Appendix 12.5.

12.7.1: QALE Gain for Alternative VSL and VSHLY and QALY Weights: Disease

LOW QALY
Low VSHLY
Low VSL

BREAST CANCER

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions	Mortality Gain Millions	QALE Gain millions
1900-1925	-934	0.08	-79	3092	3013
1925-1950	-2324	0.15	-351	2861	2510
1950-1975	14120	0.38	5389	1966	7355
1975-2000	11097	0.94	10455	6310	16765
1900-2000	11384	0.29	3283	10915	14198

STOMACH CANCER

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions	Mortality Gain Millions	QALE Gain millions
1900-1925	1313	0.08	111	3092	3204
1925-1950	-22	0.15	-3	2861	2857
1950-1975	6104	0.38	2332	1966	4298
1975-2000	2696	0.81	2178	6310	8487
1900-2000	9507	0.23	2192	10915	13107

TUBERCULOSIS

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions	Mortality Gain Millions	QALE Gain millions
1900-1925	25095	0.13	3189	3092	6281
1925-1950	17972	0.25	4529	2861	7389
1950-1975	13766	0.53	7352	1966	9318
1975-2000	354	1.08	381	6310	6690
1900-2000	51594	0.29	14878	10915	25793

12.7.1: Value of QALE Gain for Alternative VSL and VSHLY and QALY Weights: Disease

MID QALY
Mid VSHLY
Mid VSL

BREAST CANCER

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain Millions	Mortality Gain millions	QALE Gain millions
1900-1925	-934	0.21	-196	3895	3699
1925-1950	-2324	0.32	-741	3598	2857
1950-1975	14120	0.60	9601	2489	12090
1975-2000	11097	1.53	16979	8011	24990
1900-2000	11384	0.51	5806	13769	19575

STOMACH CANCER

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain Millions	Mortality Gain millions	QALE Gain millions
1900-1925	1313	0.21	278	3895	4173
1925-1950	-22	0.32	-7	3598	3591
1950-1975	6104	0.68	4151	2489	6640
1975-2000	2696	1.36	3677	8011	11688
1900-2000	9507	0.44	4183	13769	17952

TUBERCULOSIS

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain Millions	Mortality Gain millions	QALE Gain millions
1900-1925	25095	0.27	6776	3895	10671
1925-1950	17972	0.45	8088	3598	11686
1950-1975	13766	0.87	1977	2489	14466
1975-2000	354	1.70	605	8011	8616
1900-2000	51594	0.51	26313	13769	40082

12.7.1: Value of QALE Gain for Alternative VSL and VSHLY and QALY Weights: Disease

HIGH QALY
High VSHLY
High VSL

BREAST CANCER

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain Millions	Mortality Gain millions	QALE Gain millions
1900-1925	-934	0.39	-362	4715	4353
1925-1950	-2324	0.54	-1248	4362	3113
1950-1975	14120	1.05	14792	2997	17789
1975-2000	11097	2.26	25055	9621	34675
1900-2000	11384	0.79	9003	16643	25645

STOMACH CANCER

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain Millions	Mortality Gain millions	QALE Gain millions
1900-1925	1313	0.39	509	4715	5224
1925-1950	-22	0.54	-12	4362	4350
1950-1975	6104	1.05	6395	2997	9392
1975-2000	2696	2.05	5534	9621	15154
1900-2000	9507	0.70	6684	16643	23327

TUBERCULOSIS

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain Millions	Mortality Gain millions	QALE Gain millions
1900-1925	25095	0.45	11337	4715	16053
1925-1950	17972	0.69	12419	4362	16781
1950-1975	13766	1.28	17633	2997	20630
1975-2000	354	2.46	871	9621	10492
1900-2000	51594	0.79	40802	16643	57444

12.7.2: QALE Gain for Alternative VSL and VSHLY and QALY Weights: Disability

BLIND
Low VSL Low VSHLY Low QALY

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions	Mortality Burden Change	VSL millions	Mortality Gain millions	QALE Gain millions
1900-1925	569	0.21	120	6085	0.51	3092	3212
1925-1950	-10789	0.35	-3776	4734	0.60	2861	-915
1950-1975	3130	0.61	1909	2146	0.92	1966	3875
1975-2000	-886	1.08	-957	3907	1.61	6310	5353
1900-2000	-7976	0.35	-2792	15646	0.69	10820	8028

BLIND
Mid VSL Mid VSHLY Mid QALY

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions	Mortality Burden Change	VSL millions	Mortality Gain millions	QALE Gain millions
1900-1925	569	0.27	154	6085	0.64	3895	4049
1925-1950	-10789	0.45	-4855	4734	0.77	3598	-1257
1950-1975	3130	0.77	2410	2146	1.16	2489	4899
1975-2000	-886	1.36	-1205	3907	2.05	8011	6806
1900-2000	-7976	0.44	-3509	15646	0.88	13769	10260

BLIND
High VSL High VSHLY High QALY

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions	Mortality Burden Change	VSL millions	Mortality Gain millions	QALE Gain millions
1900-1925	569	0.32	182	6085	0.77	4715	4897
1925-1950	-10789	0.54	-5826	4734	0.92	4362	-1464
1950-1975	3130	0.93	2911	2146	1.40	2997	5908
1975-2000	-886	1.64	-1453	3907	2.46	9621	8168
1900-2000	-7976	0.53	-4227	15646	1.05	16498	12271

12.7.3: QALE Gain for Alternative VSL and VSHLY: Costa versus Viscusi

VSL Values: 1900-2000

1900-2000	Costa	0.64
	Viscusi	1.18
	Thesis	0.88

Summary Mortality Gain: Cost Vs Aldy VSLs

Period	Death Rate Change 1	Death Rate Change 2	VSL	Mortality Gain 1	Mortality Gain 2	**Mortality Gain**
Viscusi						
1900-2000	13805.35	17487.04	1.18	16290	21060	18675
Costa						
1900-2000	13805.35	17487.04	0.64	8835	11192	10014
Thesis						
1900-2000	13805.35	17487.04	0.88	12149	15389	13769

12.7.3: QALE Gain for Alternative VSL and VSHLY: Costa versus Viscusi

VSHLY Values
Breast Cancer

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
Viscusi 1900-2000	1.18	0.5833	**0.69**
Costa 1900-2000	0.64	0.5833	**0.37**
Thesis 1900-2000	0.88	0.5833	**0.51**

VSHLY Values
Stomach Cancer

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
Viscusi 1900-2000	1.18	0.5000	**0.59**
Costa 1900-2000	0.64	0.5000	**0.32**
Thesis 1900-2000	0.88	0.5000	**0.44**

VSHLY Values
Tuberculosis

Period	VSL (Millions)	QALY	**VSHLY (Millions)**
Viscusi 1900-2000	1.18	0.5833	**0.69**
Costa 1900-2000	0.64	0.5833	**0.37**
Thesis 1900-2000	0.88	0.5833	**0.51**

12.7.3: QALE Gain for Alternative VSL and VSHLY: Costa versus Viscusi

Author	Period / Illness	Morbidity Burden Change1	Morbidity Burden Change2	VSHLY millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	**Average Morbidity Gain millions**	**Average Mortality Gain millions**	**QALE Gain millions**
Viscusi	Breast Cancer 1900-2000	8931	13836	0.69	6162	9547	7855	18675	26530
Costa	Breast Cancer 1900-2000	8931	13836	0.37	3304	5119	4212	10014	14226
Thesis	Breast Cancer 1900-2000	8931	13836	0.51	4555	7056	5806	13769	19575

Author	Period / Illness	Morbidity Burden Change1	Morbidity Burden Change2	VSHLY millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	**Average Morbidity Gain millions**	**Average Mortality Gain millions**	**QALE Gain millions**
Viscusi	Stomach Cancer 1900-2000	6235	12779	0.59	3679	7540	5610	18675	24285
Costa	Stomach Cancer 1900-2000	6235	12779	0.32	1995	4089	3042	10014	13056
Thesis	Stomach Cancer 1900-2000	6235	12779	0.44	2743	5623	4183	13769	17952

Author	Period / Illness	Morbidity Burden Change1	Morbidity Burden Change2	VSHLY millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	**Average Morbidity Gain millions**	**Average Mortality Gain millions**	**QALE Gain millions**
Viscusi	Tuberculosis 1900-2000	61865	41322	0.69	42687	28512	35600	18675	54275
Costa	Tuberculosis 1900-2000	61865	41322	0.37	22890	15289	19090	10014	29104
Thesis	Tuberculosis 1900-2000	61865	41322	0.51	31551	21074	26312	13769	40082

12.8 and 12.9: Age-Weighting: Mortality and Morbidity

This Appendix relates to Figure 3.3 and 8.1.2 and Tables 8.1.13 and 8.3.3 in the main thesis.

Chapter 3 of the thesis has highlighted that there is evidence about the VSL not being constant across all age groups. I.e. the value of saving one life year of an unidentified person varies with the age of the person. Therefore a more valuable approach for estimating society's willingness to pay would be to include a methodology that considers the potential for different ages to have varying VSL values.

There are disputes in the literature about the standpoint from which age weighting should be conducted. Some theorist have called for equity to be the key function in age weighting: where age weights reflect the feeling that everyone is entitled to some normal span of life and anyone failing to achieve this has been cheated, while anyone living longer is getting more than their fair share. In this type of theory, younger ages receive a higher age weight. At the other end of the spectrum, some theorist claim weights should be related to the value of human capital or income.

The main body of the thesis has emphasised an alternative and more commonly utilised method of age weighting, which considers the relationship between age and efficiency, through reflecting an individuals social role, where: the population is supported by others during infancy and old age but support others during adulthood and therefore greater importance needs to be attached to these years. This form of age weighting is best represented by Murray. This is the age weighting approach that is used in the thesis. Murray's age weight function also provides a desirable mid-point between age-weighting theories.

Murray's age weighting function is not flawless but it does still represent the most attractive model for the purposes of the thesis. The main flaws of Murray's model is the static nature of the age weights for the thesis which considers a historical period of one hundred years, which almost certainly would have experienced some movement in the most valuable ages.

Murray's age weights can be applied to the mortality and the morbidity (except blindness were the data is not age specific) QALE gain calculations in the thesis. This is achieved through adjusting the VSL for Murray's age weight. For example, for mortality in 1900-1925 using a Low VSL, when there is no age weighting the VSL = 0.64 for all age groups (shown in Appendix 12.1.1) when there is an age weight the VSL = 0.64 * 0.3 (age weight for 0-4 age group) = 0.192 (shown in the first row of Appendix 12.8).

These age weighting considerations are made for all the previous QALE gain sensitivity analysis calculations. Appendix 12.8 considers the low, mid, high VSL mortality gain calculations and Appendix 12.9 considers the low, mid, high VSL, VSHLY, QALY morbidity gain calculations. Because of the density of these calculations, the bulk of the age weighted QALE gain calculations and results are provided in Appendix 13.3 (age weighted mortality) and 13.4 (age weighted morbidity). The mid VSL estimates will be shown for the age weighted mortality gain below. In Appendix 12.9 the mid VSL, QALY and subsequent VSHLY will be shown. All other calculations are contained in the CD-Rom Appendices.

12.8 and 12.9: Age-Weighting

Sources:

Mortality and morbidity gain data that is applied to the age weights

- See Appendix 12.7

Age weights

- Murray & Lopez, “The Global Burden of Disease”

12.8: Age-weighting: Mortality

Age-weighting Mortality
VSL MID

1900-1925

Age	Weight	VSL (Mid)	Weighted VSL (Mid)	Fall in DR 1	Fall in DR 2	Mortality Gain 1 million	Mortality Gain 2 million
0-4	0.3	0.64	0.192	3435.94	2594.55	659.70	498.15
5-14	1.0	0.64	0.640	318.72	374.67	203.98	239.79
15-24	1.5	0.64	0.960	175.04	159.48	168.04	153.10
25-34	1.5	0.64	0.960	347.18	328.50	333.29	315.36
35-44	1.3	0.64	0.832	507.87	569.53	422.55	473.85
45-64	1.0	0.64	0.640	1025.55	1398.45	656.35	895.01
65-74	0.7	0.64	0.448	340.82	477.24	152.69	213.80
75+	0.45	0.64	0.288	49.71	66.09	14.32	19.03
				6201	**5968**	**2611**	**2808**

1925-1950

Age	Weight	VSL (Mid)	Weighted VSL (Mid)	Fall in DR 1	Fall in DR 2	Mortality Gain 1 million	Mortality Gain 2 million
0-4	0.3	0.76	0.228	1459.97	1340.78	332.87	305.70
5-14	1.0	0.76	0.760	215.95	154.90	164.12	117.73
15-24	1.5	0.76	1.140	324.12	238.91	369.50	272.36
25-34	1.5	0.76	1.140	310.93	297.17	354.46	338.77
35-44	1.3	0.76	0.988	723.10	813.77	714.42	804.01
45-64	1.0	0.76	0.760	755.27	893.66	574.01	679.18
65-74	0.7	0.76	0.532	398.59	630.10	212.05	335.21
75+	0.45	0.76	0.342	305.81	604.66	104.59	206.79
				4494	**4974**	**2826**	**3060**

1950-1975

Age	Weight	VSL (Mid)	Weighted VSL (Mid)	Fall in DR 1	Fall in DR 2	Mortality Gain 1 million	Mortality Gain 2 million
0-4	0.3	1.16	0.348	279.60	219.64	97.30	76.44
5-14	1.0	1.16	1.160	38.87	46.00	45.09	53.36
15-24	1.5	1.16	1.740	53.66	56.50	93.37	98.32
25-34	1.5	1.16	1.740	119.56	113.17	208.03	196.92
35-44	1.3	1.16	1.508	-237.26	-174.71	-357.78	-263.47
45-64	1.0	1.16	1.160	397.25	403.31	460.81	467.84
65-74	0.7	1.16	0.812	484.39	600.90	393.33	487.93
75+	0.45	1.16	0.522	632.47	846.51	330.15	441.88
				1769	**2111**	**1270**	**1559**

12.8: Age-Weighting: Mortality

1975-2000

Age	Weight	VSL (Mid)	Weighted VSL (Mid)	Fall in DR 1	Fall in DR 2	Mortality Gain 1 million	Mortality Gain 2 million
0-4	0.3	2.05	0.615	144.01	134.03	88.56	82.43
5-14	1.0	2.05	2.050	23.99	19.40	49.19	39.76
15-24	1.5	2.05	3.075	26.89	22.86	82.69	70.28
25-34	1.5	2.05	3.075	4.46	4.69	13.72	14.44
35-44	1.3	2.05	2.665	55.69	71.60	148.40	190.82
45-64	1.0	2.05	2.050	981.72	965.03	2012.52	1978.31
65-74	0.7	2.05	1.435	1143.99	701.74	1641.63	1007.00
75+	0.45	2.05	0.923	1062.04	1436.44	979.74	1325.12
				3443	**3356**	**5016**	**4708**

1900-2000

Age	Weight	VSL (Mid)	Weighted VSL (Mid)	Fall in DR 1	Fall in DR 2	Mortality Gain 1 million	Mortality Gain 2 million
0-4	0.3	0.88	0.264	6030.98	3029.95	1592.18	799.91
5-14	1.0	0.88	0.880	648.68	399.85	570.83	351.86
15-24	1.5	0.88	1.320	646.11	396.15	852.86	522.92
25-34	1.5	0.88	1.320	813.22	732.60	1073.44	967.03
35-44	1.3	0.88	1.144	1024.61	1224.37	1172.16	1400.68
45-64	1.0	0.88	0.880	2405.36	3906.54	2116.72	3437.76
65-74	0.7	0.88	0.616	1260.91	2123.92	776.72	1308.34
75+	0.45	0.88	0.396	835.22	3859.18	330.75	1528.24
				13665	**15673**	**8486**	**10317**

12.9: Age-Weighting: Morbidity

Age-weighting Morbidity
BREAST CANCER
Mid QALY and Mid VSHLY

1900-1925

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.21	0.063	0	0	0.00	0.00
5-14	1.0	0.21	0.210	0	0	0.00	0.00
15-24	1.5	0.21	0.315	7.400679194	6.750870817	2.33	2.13
25-34	1.5	0.21	0.315	-93.1946039	-88.17946381	-29.36	-27.78
35-44	1.3	0.21	0.273	101.3650163	113.6714463	27.67	31.03
45-64	1.0	0.21	0.210	169.1754082	230.9940786	35.53	48.51
65-74	0.7	0.21	0.147	198.8753719	278.4774472	29.23	40.94
75+	0.45	0.21	0.095	-1251.96364	-1541.310123	-118.31	-145.65
						-53	**-51**

1925-1950

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.32	0.096	0	0	0.00	0.00
5-14	1.0	0.32	0.320	0	0	0.00	0.00
15-24	1.5	0.32	0.480	-15.6813014	-12.47244262	-7.53	-5.99
25-34	1.5	0.32	0.480	-59.9331832	-57.28011318	-28.77	-27.49
35-44	1.3	0.32	0.416	-299.648738	-337.2218944	-124.65	-140.28
45-64	1.0	0.32	0.320	-1259.9512	-1519.940858	-403.18	-486.38
65-74	0.7	0.32	0.224	-454.481359	-718.4506349	-101.80	-160.93
75+	0.45	0.32	0.144	27.70783873	59.15467739	3.99	8.52
						-662	**-813**

1950-1975

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Falling MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.68	0.204	0	0	0.00	0.00
5-14	1.0	0.68	0.680	0	0	0.00	0.00
15-24	1.5	0.68	1.020	7.051689492	7.068419936	7.19	7.21
25-34	1.5	0.68	1.020	173.1515881	163.9046285	176.61	167.18
35-44	1.3	0.68	0.884	1233.746114	908.5091965	1090.63	803.12
45-64	1.0	0.68	0.680	5717.188384	5724.110671	3887.69	3892.40
65-74	0.7	0.68	0.476	3405.173296	4224.221886	1620.86	2010.73
75+	0.45	0.68	0.306	2750.560381	3924.677915	841.67	1200.95
						7625	**8082**

1975-2000

Age	Weight	VSHLY (Mid)	Weighted VSL (Mid)	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	1.53	0.459	0	0	0.00	0.00
5-14	1.0	1.53	1.530	0	0	0.00	0.00
15-24	1.5	1.53	2.295	7.506614431	6.433704377	17.23	14.77
25-34	1.5	1.53	2.295	79.33362467	83.49086531	182.07	191.61
35-44	1.3	1.53	1.989	817.5821771	1051.271652	1626.17	2090.98
45-64	1.0	1.53	1.530	5169.091839	5188.104845	7908.71	7937.80
65-74	0.7	1.53	1.071	3054.499154	1873.685355	3271.37	2006.72
75+	0.45	1.53	0.689	1963.623105	2899.268269	1351.95	1996.15
						14358	**14238**

12.9: Age-Weighting: Morbidity: Breast Cancer

1900-2000

Age	Weight	VSHLY (Mid)	Weighted VSLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.51	0.153	0	0	0.00	0.00
5-14	1.0	0.51	0.510	0	0	0.00	0.00
15-24	1.5	0.51	0.765	8.876553434	5.442509095	6.79	4.16
25-34	1.5	0.51	0.765	82.86260848	74.64800639	63.39	57.11
35-44	1.3	0.51	0.663	1470.712206	1757.443481	975.08	1165.19
45-64	1.0	0.51	0.510	5152.378485	8307.783149	2627.71	4236.97
65-74	0.7	0.51	0.357	2226.465416	3750.334394	794.85	1338.87
75+	0.45	0.51	0.230	-10.7162837	-59.33925127	-2.46	-13.62
						4465	**6789**

Age-weighting Morbidity
STOMACH CANCER
Mid QALY and Mid VSHLY

1900-1925

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.21	0.063	0	0	0	0
5-14	1.0	0.21	0.210	0	0	0	0
15-24	1.5	0.21	0.315	0.796183774	0.725302166	0.250797889	0.228470182
25-34	1.5	0.21	0.315	8.443717402	7.989330305	2.659770982	2.516639046
35-44	1.3	0.21	0.273	67.44677644	75.63529215	18.41296997	20.64843476
45-64	1.0	0.21	0.210	587.2936471	802.2982129	123.3316659	168.4826247
65-74	0.7	0.21	0.147	397.9904028	557.2904797	58.50458921	81.92170052
75+	0.45	0.21	0.095	53.82097704	66.25976514	5.08608233	6.261547806
				1116	**1510**	**208**	**280**

1925-1950

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.32	0.096	0	0	0	0
5-14	1.0	0.32	0.320	0	0	0	0
15-24	1.5	0.32	0.480	-2.57562752	-1.963917159	-1.23630121	-0.942680236
25-34	1.5	0.32	0.480	3.253138053	3.109130965	1.561506265	1.492382863
35-44	1.3	0.32	0.416	11.8325508	13.3162423	4.922341134	5.539556798
45-64	1.0	0.32	0.320	391.3878549	468.0769038	125.2441136	149.7846092
65-74	0.7	0.32	0.224	76.02677083	120.1842072	17.02999667	26.92126241
75+	0.45	0.32	0.144	-359.160407	-766.7872703	-51.71909866	-110.4173669
				121	**-164**	**96**	**72**

1950-1975

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.68	0.204	0	0	0	0
5-14	1.0	0.68	0.680	0	0	0	0
15-24	1.5	0.68	1.020	6.307354573	6.602388563	6.433501665	6.734436334
25-34	1.5	0.68	1.020	45.65610678	43.2178954	46.56922892	44.0822533
35-44	1.3	0.68	0.884	303.3666536	223.3939314	268.1761218	197.4802354
45-64	1.0	0.68	0.680	1991.702777	2027.860592	1354.357888	1378.945202
65-74	0.7	0.68	0.476	1766.641911	2191.573461	840.9215498	1043.188968
75+	0.45	0.68	0.306	1484.393607	2118.029056	454.2244438	648.1168912
				5598	**6611**	**2971**	**3319**

1975-2000

Age	Weight	VSHLY (Mid)	Weighted VSL (Mid)	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	1.37	0.411	0	0	0	0
5-14	1.0	1.37	1.370	0	0	0	0
15-24	1.5	1.37	2.055	0.971036667	0.832882807	1.99548035	1.711574169
25-34	1.5	1.37	2.055	6.919597396	7.282198142	14.21977265	14.96491718
35-44	1.3	1.37	1.781	28.74164964	36.95687398	51.18887801	65.82019256
45-64	1.0	1.37	1.370	763.9095819	746.5698186	1046.556127	1022.800651
65-74	0.7	1.37	0.959	984.771448	604.0767232	944.3958187	579.3095776
75+	0.45	1.37	0.617	893.1640807	1318.747102	550.6356558	813.0075883
				2678	**2714**	**2609**	**2498**

12.9: Age-Weighting: Morbidity: Stomach Cancer

1900-2000

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.44	0.132	0	0	0	0
5-14	1.0	0.44	0.440	0	0	0	0
15-24	1.5	0.44	0.660	9.934779215	6.091409269	6.556954282	4.020330118
25-34	1.5	0.44	0.660	86.05815066	77.52675782	56.79837943	51.16766016
35-44	1.3	0.44	0.572	423.1599142	505.6595231	242.0474709	289.2372472
45-64	1.0	0.44	0.440	3058.989918	5047.752165	1345.955564	2221.010953
65-74	0.7	0.44	0.308	1964.539632	3309.137658	605.0782068	1019.214399
75+	0.45	0.44	0.198	692.1027505	3832.378863	137.0363446	758.8110148
				6235	**12779**	**2393**	**4343**

Age-weighting Morbidity
TUBERCULOSIS
Mid QALY and Mid VSHLY

1900-1925

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.27	0.081	26125.87925	19449.66491	2116.19622	1575.422858
5-14	1.0	0.27	0.270	10644.00358	11734.69911	2873.880966	3168.368759
15-24	1.5	0.27	0.405	-1369.13356	-1213.610125	-554.4990905	-491.5121006
25-34	1.5	0.27	0.405	-3357.63795	-3176.951255	-1359.843368	-1286.665258
35-44	1.3	0.27	0.351	-2089.95076	-2343.685564	-733.5727165	-822.6336328
45-64	1.0	0.27	0.270	-1615.2181	-2181.72411	-436.1088871	-589.0655097
65-74	0.7	0.27	0.189	-224.146506	-313.8636331	-42.36368961	-59.32022666
75+	0.45	0.27	0.122	54.28464825	66.83059732	6.595584762	8.119917574
				28168	**22021**	**1870**	**1503**

1925-1950

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.44	0.132	2034.03609	2035.673289	268.4927639	268.7088741
5-14	1.0	0.44	0.440	1642.834566	995.6207823	722.8472092	438.0731442
15-24	1.5	0.44	0.660	5313.084531	3921.539499	3506.635791	2588.216069
25-34	1.5	0.44	0.660	4063.123997	3883.261156	2681.661838	2562.952363
35-44	1.3	0.44	0.572	3532.128437	3975.024396	2020.377466	2273.713954
45-64	1.0	0.44	0.440	1993.287763	2146.536845	877.0466159	944.4762119
65-74	0.7	0.44	0.308	111.573124	176.376391	34.3645222	54.32392841
75+	0.45	0.44	0.198	37.98332589	81.09226457	7.520698526	16.05626838
				18728	**17215**	**10119**	**9147**

1950-1975

Age	Weight	VSHLY (Mid)	Weighted VSL (Mid)	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.87	0.261	570.8908145	464.7444803	149.0025026	121.2983094
5-14	1.0	0.87	0.870	274.2410447	326.3057436	238.5897089	283.8859969
15-24	1.5	0.87	1.305	1538.696728	1620.621496	2007.99923	2114.911053
25-34	1.5	0.87	1.305	2692.867169	2549.057724	3514.191656	3326.52033
35-44	1.3	0.87	1.131	2357.787479	1736.233723	2666.657639	1963.680341
45-64	1.0	0.87	0.870	5034.308945	4961.43806	4379.848782	4316.451112
65-74	0.7	0.87	0.609	1359.797661	1686.870694	828.1167755	1027.304252
75+	0.45	0.87	0.392	147.9352809	211.0836518	57.91666247	82.63924969
				13977	**13556**	**13842**	**13237**

1975-2000

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	1.71	0.513	3.829283096	3.356569916	1.964422228	1.721920367
5-14	1.0	1.71	1.710	-4.185318867	-3.361278263	-7.156895262	-5.74778583
15-24	1.5	1.71	2.565	10.58021691	8.993643574	27.13825638	23.06869577
25-34	1.5	1.71	2.565	5.353274348	5.633796633	13.7311487	14.45068836
35-44	1.3	1.71	2.223	6.07897602	7.816529444	13.51356369	17.37614495
45-64	1.0	1.71	1.710	268.2953826	271.2939947	458.7851043	463.9127309
65-74	0.7	1.71	1.197	107.1861569	65.7499388	128.3018298	78.70267675
75+	0.45	1.71	0.770	-19.85980144	-29.32278196	-15.28211721	-22.56388072
				377	**330**	**621**	**571**

12.9: Age-Weighting: Morbidity: Tuberculosis

1900-2000

Age	Weight	VSHLY (Mid)	Weighted VSHLY	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.51	0.153	32963.83056	17525.39089	5043.466076	2681.384806
5-14	1.0	0.51	0.510	13947.83794	8614.582243	7113.39735	4393.436944
15-24	1.5	0.51	0.765	6235.885789	3823.347706	4770.452629	2924.860995
25-34	1.5	0.51	0.765	3127.668735	2817.606638	2392.666582	2155.469078
35-44	1.3	0.51	0.663	2431.599597	2905.666277	1612.150533	1926.456742
45-64	1.0	0.51	0.510	2605.842774	4172.730281	1328.979815	2128.092443
65-74	0.7	0.51	0.357	413.6437681	696.7556916	147.6708252	248.7417819
75+	0.45	0.51	0.230	138.382849	766.2670101	31.75886384	175.8582788
				61865	**41322**	**22441**	**16634**

12.10: Age-Weighted Value of QALE Gain

This Appendix relates to Tables 8.1.13 in the main thesis.

After the mortality gain and morbidity gain have been applied to Murray's age weighting function (as has been achieved in Appendix 12.8 and 12.9) they can be combined in order to identify the age-weighted QALE gain. This is achieved here.

The analysis below will repeat the QALE gain calculation for all ranges and subsequent combinations of QALY, VSL and VSHLY weight (shown in Appendix 12.7), for all illnesses and eras, applied to Murray's age weighting function.

Appendix 12.10.1 will present the age weighted QALE gain for the thesis diseases, which combined the age weighted mortality gain and individual age weighted morbidity gains. Appendix 12.10.2 considers the age weighted disability gain, this comprises the age weighted mortality gain and a un-weighted morbidity gain (as the data does not exist to make age weighted morbidity gain calculations for blindness).

Because of the density of this analysis the majority of the results are contained in the CD-Rom Appendices. This Appendix will present the most extreme and mid point estimates, i.e. the age weighted QALE gain results using a low, mid, and high QALY, VSL and VSHLY. For all other variable weight combinations, see Appendix 13.5.

Sources:

- QALE methodology outlined in Appendix 12.7 is used here, but applied to Murray's age weighting function.

Age weights

- Murray & Lopez, "The Global Burden of Disease"

12.10.1: Age-Weighted Value of QALE Gain: Disease

Low VSL and Low VSHLY and Low QALY

	Deaths		**Stomach Cancer**		**Breast Cancer**		**Tuberculosis**	
Period	Mortality Gain 1 millions	Mortality Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions
1900-1925	2081	2238	84	113	-21	-21	880	707
1925-1950	2231	2416	45	34	-312	-384	5795	5238
1950-1975	1007	1237	1669	1864	4280	4536	8497	8126
1975-2000	3940	3698	1538	1472	8841	8768	391	359
1900-2000	6654	8089	1254	2276	2525	3839	12689	9406

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis
1900-1925	2258	2138	2953
1925-1950	2363	1975	7840
1950-1975	2889	5530	9434
1975-2000	5324	12623	4194
1900-2000	9136	10553	18419

12.10.1: Age-Weighted Value of QALE Gain: Disease

Mid VSL and Mid VSHLY and Mid QALY

	Deaths		**Stomach Cancer**		**Breast Cancer**		**Tuberculosis**	
Period	Mortality Gain 1 millions	Mortality Gain 2 millions	Morbidity Gain 1 Millions	Morbidity Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions
1900-1925	2611	2808	208	280	-53	-51	1870	1503
1925-1950	2826	3060	96	72	-662	-813	10119	9147
1950-1975	1270	1559	2971	3319	7625	8082	13842	13237
1975-2000	5016	4708	2609	2498	14358	14238	621	571
1900-2000	8486	10317	2393	4343	4465	6789	22441	16634

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis
1900-1925	2954	2658	4397
1925-1950	3027	2205	12576
1950-1975	4560	9269	14955
1975-2000	7416	19160	5458
1900-2000	12770	15029	28940

12.10.1: Age-Weighted Value of QALE Gain: Disease

High VSL and High VSHLY and High QALY

	Deaths		Stomach Cancer		Breast Cancer		Tuberculosis	
Period	Mortality Gain 1 millions	Mortality Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions	Morbidity Gain 1 millions	Morbidity Gain 2 millions
1900-1925	3141	3378	384	517	-98	-94	3129	2514
1925-1950	3421	3704	161	121	-1111	-1364	15892	14365
1950-1975	1533	1882	4577	5112	11746	12450	20379	19488
1975-2000	6020	5650	3908	3741	21187	21011	894	822
1900-2000	10125	12310	3825	6941	6924	10527	34797	25794

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis
1900-1925	3710	3164	6081
1925-1950	3704	2325	18691
1950-1975	6552	13806	21641
1975-2000	9660	26934	6693
1900-2000	16600	19943	41513

12.10.2: Age weighted Value of QALE Gain: Disability

Low VSL Low VSHLY Low QALY

Period	Morbidity Gain millions	Mortality Gain millions	QALE Gain millions
1900-1925	120	2160	2280
1925-1950	-3776	2324	-1452
1950-1975	1909	1122	3031
1975-2000	-957	3819	2862
1900-2000	-2792	7372	4580

Mid VSL Mid VSHLY Mid QALY

Period	Morbidity Gain millions	Mortality Gain millions	QALE Gain millions
1900-1925	154	2710	2864
1925-1950	-4855	2943	-1912
1950-1975	2410	1415	3825
1975-2000	-1205	4862	3657
1900-2000	-3509	9402	5893

High VSL High VSHLY High QALY

Period	Morbidity Gain millions	Mortality Gain millions	QALE Gain millions
1900-1925	182	3260	3442
1925-1950	-5826	3563	-2263
1950-1975	2911	1708	4619
1975-2000	-1453	5835	4382
1900-2000	-4227	11218	6991

12.11: GDP per capita Compound Average Growth Rates

This Appendix relates to Tables 8.1.14, 8.1.15, 8.2.8, 8.3.4, and 9.3 in the main thesis.

This Appendix identifies the growth rate of national income for the eras of the thesis. This is necessary to address one of the main questions of the thesis, about the value of improved QALE and what this contributes to twentieth century welfare economic development.

Hence, the main body of the thesis has consistently emphasised the need to consider more comprehensive or utility inclusive measures of national income. This exercise is conducted in the following Appendix (12.12). However, in order to illuminate the veracity of this thesis claim it is desirable to compare GDP growth and GDP+QALE gain growth. GDP growth is calculated here (and QALE gain growth is calculated in Appendix 12.12).

The compound average growth rates of GDP pc (which is the average rate of annual growth within a defined period) was calculated using the natural exponential function $S = Pe^{rt}$, for all five eras considered in the thesis: 1900-1925, 1925-1950, 1950-1975, 1975-2000 and 1900-2000.

Sources:

GDP per capita data

- Maddison, “Monitoring the World Economy 1820-1992”
- Maddison, “The World Economy: A Millennial Perspective”

Compound average growth rate methodology

- Dowling, “Mathematical Methods for Business and Economics”

12.11: GDP per capita Compound Average Growth Rates

GDP per capita Compound Average Rate of Growth per annum

Period	GDPpc at Start (t1)	GDPpc at End (t2)	t	t2 / t1	Ln	Ln / t	Growth
1900-1925	4593	4912	25	1.069454	0.067148	0.002686	**0.3**
1925-1950	4912	6907	25	1.406148	0.340854	0.013634	**1.4**
1950-1975	6907	11845	25	1.714927	0.53937	0.021575	**2.2**
1975-2000	11845	18714	25	1.579907	0.457366	0.018295	**1.8**
1900-2000	4593	18714	100	4.074461	1.404739	0.014047	**1.4**

Period	Midpoint	GDPpc at Midpoint
1900-1925	1913	5032
1925-1950	1938	5983
1950-1975	1963	9070
1975-2000	1988	15988
1900-2000	1950	6907

12.12: Value of QALE Gain relative to GDP per capita Growth

This Appendix relates to Tables 8.1.15, 8.2.8, and 9.3 in the main thesis.

The main body of the thesis has consistently emphasised the need to value and standardise health improvements in twentieth century England. As a result of this objective the QALE gain methodology was developed in such a way that the QALE gain results generated by this methodology could be compared to the national income measures. Following on from this, another consistent objective of the thesis has been to provide a more thorough and accurate account of economic developments from a standards of living or welfare economics perspective, i.e. a Fisharian measure of economic development, which essentially represents a more rounded utility national income. Hence, the thesis will ultimately generate an alternative estimate for national income growth that includes the value of the QALE gain.

This extended GDP or utility national income is estimated by calculating the compound average rate of GDP per capita growth and the compound average rate of QALE gain growth. These are then summed to provide 'Adjusted Growth', which represents utility national income or Fisharian growth, or extended GDP growth.

In addition the mortality and morbidity gain growth are also considered here (as this provides the foundation level and a more detailed indication of the QALE gain growth construction). Finally, another layer of detail is that these considerations are made for the un-weighted and age-weighted QALE gain results shown above in Appendix 12.7 and 12.9, respectively.

Appendix 12.12 provides the mortality gain and morbidity gains. Appendix 12.12.1 presents the QALE gain growth for the thesis diseases and Appendix 12.12.2 makes the same consideration but for the thesis disability. Because of the density of the calculations in Appendix 12.12.1 and 12.12.2, the bulk of these results will be presented in Appendix 13.6.1 and 13.6.2 in the CD-Rom Appendices. The most extreme (low and high) and mid calculations for the un-weighted and age-weighted calculations will be shown here and the complete set of calculations are contained in the CD-Rom appendices.

Sources:

GDP per capita data

- Maddison, "Monitoring the World Economy 1820-1992"
- Maddison, "The World Economy: A Millennial Perspective"

Compound average growth rate methodology

- Dowling, "Mathematical Methods for Business and Economics"

QALE Gain numbers

- Appendix 12.7 (un-weighted)
- Appendix 12.10 (age-weighted)

12.12: Value of QALE Gain relative to GDP per capita Growth: Mortality and Morbidity

Mortality Gains - UN-WEIGHTED as a proportion of GDP Growth

Period	Death Rate Change 1	Death Rate Change 2	VSL millions	Mortality Gain 1 millions	Mortality Gain 2 millions
1900-1925	6200.828	5968.488	0.64	3969	3820
1925-1950	4493.741	4973.952	0.76	3415	3780
1950-1975	1906.388	2385.516	1.16	2211	2767
1975-2000	3780.775	4034.04	2.05	7751	8270
1900-2000	13805.35	17487.04	0.88	12149	15389

Period	t	Mortality Gain 1 millions	Mortality Gain 2 millions	Midpoint	GDPpc at Midpoint	Mortality Gain 1		Mortality Gain 2	
						AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)			
1900-1925	25	3969	3820	1913	5032	79	3.2	76	3.0
1925-1950	25	3415	3780	1938	5983	57	2.3	63	2.5
1950-1975	25	2211	2767	1963	9070	24	1.0	31	1.2
1975-2000	25	7751	8270	1988	15988	48	1.9	52	2.1
1900-2000	100	12149	15389	1950	6907	176	1.8	223	2.2

12.12: Value of QALE Gain relative to GDP per capita Growth: Mortality and Morbidity

Mortality Gains - AGE-WEIGHTED as a proportion of GDP Growth

Period	t	**VSL (LOW)** Mortality Gain 1	Mortality Gain 2	Midpoint	GDPpc at Midpoint	Mortality Gain 1		Mortality Gain 2	
						AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)			
1900-1925	25	2081	2238	1913	5032	41	1.7	44	1.8
1925-1950	25	2231	2416	1938	5983	37	1.5	40	1.6
1950-1975	25	1007	1237	1963	9070	11	0.4	14	0.5
1975-2000	25	3940	3698	1988	15988	25	1.0	23	0.9
1900-2000	100	6654	8089	1950	6907	96	1.0	117	1.2

Period	t	**VSL (MID)** Mortality Gain 1	Mortality Gain 2	Midpoint	GDPpc at Midpoint	Mortality Gain 1		Mortality Gain 2	
						AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)			
1900-1925	25	2611	2808	1913	5032	52	2.1	56	2.2
1925-1950	25	2826	3060	1938	5983	47	1.9	51	2.0
1950-1975	25	1270	1559	1963	9070	14	0.6	17	0.7
1975-2000	25	5016	4708	1988	15988	31	1.3	29	1.2
1900-2000	100	8486	10317	1950	6907	123	1.2	149	1.5

Period	t	**VSL (HIGH)** Mortality Gain 1	Mortality Gain 2	Midpoint	GDPpc at Midpoint	Mortality Gain 1		Mortality Gain 2	
						AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)			
1900-1925	25	3141	3378	1913	5032	62	2.5	67	2.7
1925-1950	25	3421	3704	1938	5983	57	2.3	62	2.5
1950-1975	25	1533	1882	1963	9070	17	0.7	21	0.8
1975-2000	25	6020	5650	1988	15988	38	1.5	35	1.4
1900-2000	100	10125	12310	1950	6907	147	1.5	178	1.8

12.12: Value of QALE Gain relative to GDP per capita Growth: Mortality and Morbidity

Breast Cancer Morbidity Gain Age-Weighted

Period	t	Morbidity Gain 1	Morbidity Gain 2	Midpoint	GDPpc at Midpoint	Morbidity Gain 1 AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)		Morbidity Gain 2 AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)	
1900-1925	25	-53	-51	1913	5032	-1	0.0	-1	0.0
1925-1950	25	-662	-813	1938	5983	-11	-0.4	-14	-0.5
1950-1975	25	7625	8082	1963	9070	84	3.4	89	3.6
1975-2000	25	14358	14238	1988	15988	90	3.6	89	3.6
1900-2000	100	4465	6789	1950	6907	65	0.6	98	1.0

Stomach Cancer Morbidity Gain Age-Weighted

Period	t	Morbidity Gain 1	Morbidity Gain 2	Midpoint	GDPpc at Midpoint	Morbidity Gain 1 AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)		Morbidity Gain 2 AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)	
1900-1925	25	208	280	1913	5032	4	0.2	6	0.2
1925-1950	25	96	72	1938	5983	2	0.1	1	0.0
1950-1975	25	2971	3319	1963	9070	33	1.3	37	1.5
1975-2000	25	2609	2498	1988	15988	16	0.7	16	0.6
1900-2000	100	2393	4343	1950	6907	35	0.3	63	0.6

Tuberculosis Morbidity Gain Age-Weighted

Period	t	Morbidity Gain 1	Morbidity Gain 2	Midpoint	GDPpc at Midpoint	Morbidity Gain 1 AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)		Morbidity Gain 2 AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)	
1900-1925	25	1870	1503	1913	5032	37	1.5	30	1.2
1925-1950	25	10119	9147	1938	5983	169	6.8	153	6.1
1950-1975	25	13842	13237	1963	9070	153	6.1	146	5.8
1975-2000	25	621	571	1988	15988	4	0.2	4	0.1
1900-2000	100	22441	16634	1950	6907	325	3.2	241	2.4

12.12: Value of QALE Gain relative to GDP per capita Growth: Mortality and Morbidity

Blind Morbidity Gain Un-Weighted

Period	t		Midpoint	GDPpc at Midpoint	Morbidity Gain AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)	
1900-1925	25	154	1913	5032	3	0.1
1925-1950	25	-4855	1938	5983	-81	-3.2
1950-1975	25	2410	1963	9070	27	1.1
1975-2000	25	-1205	1988	15988	-7.5	-0.3
1900-2000	100	-3509	1950	6907	-51	-0.5

12.12.1: Value of QALE Gain relative to GDP per capita Growth: Disease

QALE Gain as a % of GDP pc - Un-Weighted -Low VSLs

Low VSL and Low VSHLY with Low QALY

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis	t	Midpoint	GDPpc at Midpoint	QALE Gain Stomach Cancer		QALE Gain Breast Cancer		QALE Gain Tuberculosis	
							AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)					
1900-1925	3204	3013	6281	25	1913	5032	63.7	2.5	59.9	2.4	124.8	6281
1925-1950	2857	2510	7389	25	1938	5983	47.8	1.9	41.9	1.7	123.5	7389
1950-1975	4298	7355	9318	25	1963	9070	47.4	1.9	81.1	3.2	102.7	9318
1975-2000	8487	16765	6690	25	1988	15988	53.1	2.1	104.9	4.2	41.8	6690
1900-2000	13107	14198	25793	100	1950	6907	189.8	1.9	205.6	2.1	373.4	25793

Mid VSL and Mid VSHLY with MID QALY

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis	t	Midpoint	GDPpc at Midpoint	QALE Gain Stomach Cancer		QALE Gain Breast Cancer		QALE Gain Tuberculosis	
							AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)					
1900-1925	4175	3694	10626	25	1913	5032	83.0	3.319	73.4	2.9	211.2	8.4
1925-1950	3591	2856	11607	25	1938	5983	60.0	2.401	47.7	1.9	194.0	7.8
1950-1975	6613	10995	14452	25	1963	9070	72.9	2.917	121.2	4.8	159.3	6.4
1975-2000	11688	25008	8613	25	1988	15988	73.1	2.924	156.4	6.3	53.9	2.2
1900-2000	17933	22460	40135	100	1950	6907	259.6	2.596	325.2	3.3	581.1	5.8

High VSL and High VSHLY with High QALY

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis	t	Midpoint	GDPpc at Midpoint	QALE Gain Stomach Cancer		QALE Gain Breast Cancer		QALE Gain Tuberculosis	
							AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)					
1900-1925	5224	4353	16053	25	1913	5032	103.8	4.2	86.5	3.5	319.0	12.8
1925-1950	4350	3113	16781	25	1938	5983	72.7	2.9	52.0	2.1	280.5	11.2
1950-1975	9392	17789	20630	25	1963	9070	103.6	4.1	196.1	7.8	227.5	9.1
1975-2000	15154	34675	10492	25	1988	15988	94.8	3.8	216.9	8.7	65.6	2.6
1900-2000	23327	25645	57445	100	1950	6907	337.7	3.4	371.3	3.7	831.7	8.3

12.12.1: Value of QALE Gain relative to GDP per capita Growth: Disease

QALE Gain as a % of GDP pc - Age-Weighted -Low VSLs

Low VSL and Low VSHLY with Low QALY

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis	t	Midpoint	GDPpc at Midpoint	QALE Gain Stomach Cancer		QALE Gain Breast Cancer		QALE Gain Tuberculosis	
							AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)					
1900-1925	2258	2138	2953	25	1913	5032	44.9	1.8	42.5	1.7	58.7	2.3
1925-1950	2363	1975	7840	25	1938	5983	39.5	1.6	33.0	1.3	131.0	5.2
1950-1975	2889	5530	9434	25	1963	9070	31.8	1.3	61.0	2.4	104.0	4.2
1975-2000	5324	12623	4194	25	1988	15988	33.3	1.3	79.0	3.2	26.2	1.0
1900-2000	9136	10553	18419	100	1950	6907	132.3	1.3	152.8	1.5	266.7	2.7

Mid VSL and Mid VSHLY with MID QALY

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis	t	Midpoint	GDPpc at Midpoint	QALE Gain Stomach Cancer		QALE Gain Breast Cancer		QALE Gain Tuberculosis	
							AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)					
1900-1925	3005	2647	4396	25	1913	5032	59.7	2.4	52.6	2.1	87.4	3.5
1925-1950	3035	2131	12576	25	1938	5983	50.7	2.0	35.6	1.4	210.2	8.4
1950-1975	4592	9349	14954	25	1963	9070	50.6	2.0	103.1	4.1	164.9	6.6
1975-2000	7371	18700	5458	25	1988	15988	46.1	1.8	117.0	4.7	34.1	1.4
1900-2000	12931	15028	28939	100	1950	6907	187.2	1.9	217.6	2.2	419.0	4.2

High VSL and High VSHLY with High QALY

Period	QALE Gain Stomach Cancer	QALE Gain Breast Cancer	QALE Gain Tuberculosis	t	Midpoint	GDPpc at Midpoint	QALE Gain Stomach Cancer		QALE Gain Breast Cancer		QALE Gain Tuberculosis	
							AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)					
1900-1925	3710	3164	6081	25	1913	5032	73.7	2.9	62.9	2.5	120.9	4.8
1925-1950	3704	2325	18691	25	1938	5983	61.9	2.5	38.9	1.6	312.4	12.5
1950-1975	6552	13806	21641	25	1963	9070	72.2	2.9	152.2	6.1	238.6	9.5
1975-2000	9660	26934	6693	25	1988	15988	60.4	2.4	168.5	6.7	41.9	1.7
1900-2000	16600	19943	41513	100	1950	6907	240.3	2.4	288.7	2.9	601.0	6.0

12.12.2: Value of QALE Gain relative to GDP per capita Growth: Disability

Blind QALE Gain: Un-weighted

Low VSL Low VSHLY Low QALY			Midpoint	GDPpc at Midpoint	QALE Gain Blindness	
Period		QALE Gain				
	t	millions			**AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)**	
1900-1925	25	3212	1913	5032	63	2.5
1925-1950	25	-915	1938	5983	2	0.1
1950-1975	25	3875	1963	9070	37	1.5
1975-2000	25	5353	1988	15988	35	1.4
1900-2000	100	8028	1950	6907	130	1.3

Mid VSL Mid VSHLY Mid QALY			Midpoint	GDPpc at Midpoint	QALE Gain Blindness	
Period		QALE Gain				
	t	millions			**AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)**	
1900-1925	25	4049	1913	5032	81	3.2
1925-1950	25	-1257	1938	5983	-20	-0.8
1950-1975	25	4899	1963	9070	54	2.2
1975-2000	25	6806	1988	15988	42	1.7
1900-2000	100	10260	1950	6907	148	1.5

High VSL High VSHLY High QALY			Midpoint	GDPpc at Midpoint	QALE Gain Blindness	
Period		QALE Gain				
	t	millions			**AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)**	
1900-1925	25	4897	1913	5032	99	4.0
1925-1950	25	-1464	1938	5983	-52	-2.1
1950-1975	25	5908	1963	9070	73	2.9
1975-2000	25	8168	1988	15988	49	2.0
1900-2000	100	12271	1950	6907	158	1.6

12.12.2: Value of QALE Gain relative to GDP per capita Growth: Disability: Blindness

Blind QALE Gain using Age-weighted Mortality Gain

Low VSL Low VSHLY Low QALY

Period	t	QALE Gain millions	Midpoint	GDP at Midpoint	QALE Gain Blindness **AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)**	
					total	per annum
1900-1925	25	2280	1913	5032	44	1.8
1925-1950	25	-1452	1938	5983	-7	-0.3
1950-1975	25	3031	1963	9070	28	1.1
1975-2000	25	2862	1988	15988	19	0.8
1900-2000	100	4580	1950	6907	80	0.8

Mid VSL Mid VSHLY Mid QALY

Period	t	QALE Gain millions	Midpoint	GDP at Midpoint	QALE Gain Blindness **AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)**	
					total	per annum
1900-1925	25	2864	1913	5032	57	2.3
1925-1950	25	-1912	1938	5983	-31	-1.3
1950-1975	25	3825	1963	9070	42	1.7
1975-2000	25	3657	1988	15988	23	0.9
1900-2000	100	5893	1950	6907	86	0.9

High VSL High VSHLY High QALY

Period	t	QALE Gain millions	Midpoint	GDP at Midpoint	QALE Gain Blindness **AS A PROPORTION OF GDPpc (TOTAL / PER ANNUM)**	
					total	per annum
1900-1925	25	3442	1913	5032	70	2.8
1925-1950	25	-2263	1938	5983	-65	-2.6
1950-1975	25	4619	1963	9070	59	2.4
1975-2000	25	4382	1988	15988	25	1.0
1900-2000	100	6991	1950	6907	81	0.8

12.13: Blind Quantitative Estimates

12.13.1: Years in Blindness

This appendix provides the calculations and base data for the number of years of blindness estimates in the thesis.

12.13.2: Cohort Effect Calculations

This appendix utilises the results of Appendix 12.13.1 in order to calculate the extent of cohort effect distortions.

12.13.3: Blind Income Details

This appendix provides the collection of more detailed tables that were used to construct a summary in Table 4.2 in the main thesis.

12.13.1: Years in Blindness

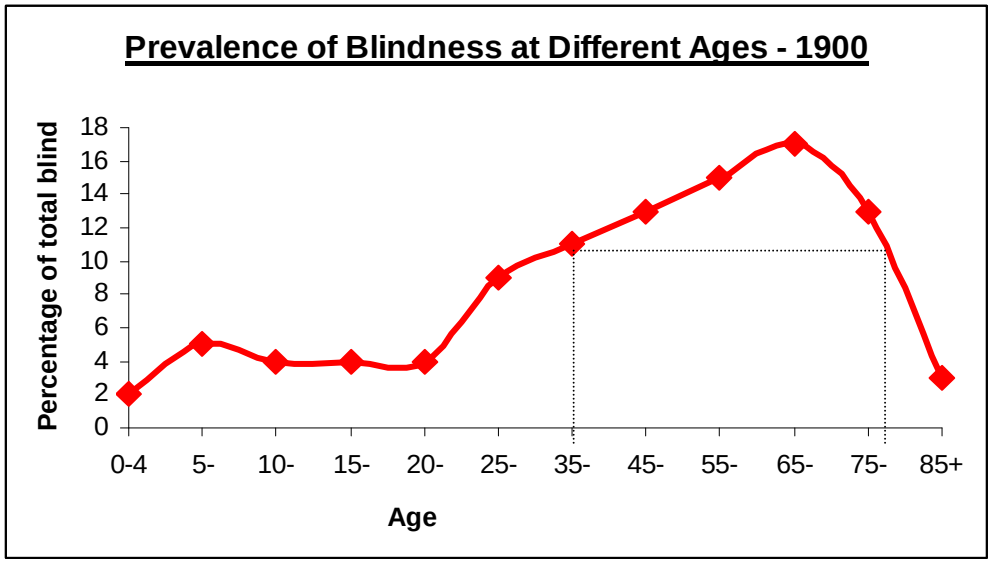
- In order to highlight the trends of an increased age at onset of blindness and the subsequent fewer years of blindness it is necessary to estimate the average number of years spent in blindness at different times during the twentieth century (namely, 1900, 1925, 1950, 1975 and 2000).

- This is exceptionally difficult to estimate as a result of the following:
    - The ideal method of estimating the average number of years spent in blindness would be to consider the most common age of onset of blindness in conjunction with average life expectancy (at the average age of onset) for each period during the twentieth century. This is not possible for the following reasons:
        1. The main problem is related to the data for blindness, whereby the data for the prevalence of blindness is only provided in 5 to 10 year blocks, so it is impossible to determine the exact age within a block. For example, in 1901 the greatest numbers of blind persons were aged between 65 and 74.
        2. This problem is exacerbated by the nature of life expectancy data, whereby life expectancy is only an average. This problem is evident throughout the twentieth century. The calculations will overcome this problem as far as possible through using age specific life expectancy, for the average age of onset of blindness.
    - Therefore, the combination of these problems means that it is only possible to provide an indication of the likely number of years spent in blindness at different times during the twentieth century. This will be achieved below.
- Along a more positive vein, this exercise still vividly highlights the improved trend in the age of onset of blindness (as it is possible to illustrate the increase in the average age of blindness and therefore the decline in the number of years spent in this less than optimal state).

- The average age of onset of blindness and the subsequent average number of years spent in blindness will be estimates through graphically identifying the most common ages of blindness for the eras considered by the thesis (1900, 1925, 1950, 1975 and 2000). This will often contain three reference points, where blindness was most common.
- Once the most common ages of blindness have been identified this information will be considered in conjunction with life expectancy in order to identify the number of average years spent in blindness.
- After the profile of estimates about the average number if years in blindness has been constructed, consideration will be made about double counting and cohort effects.

12.13.1: Years in Blindness

1900



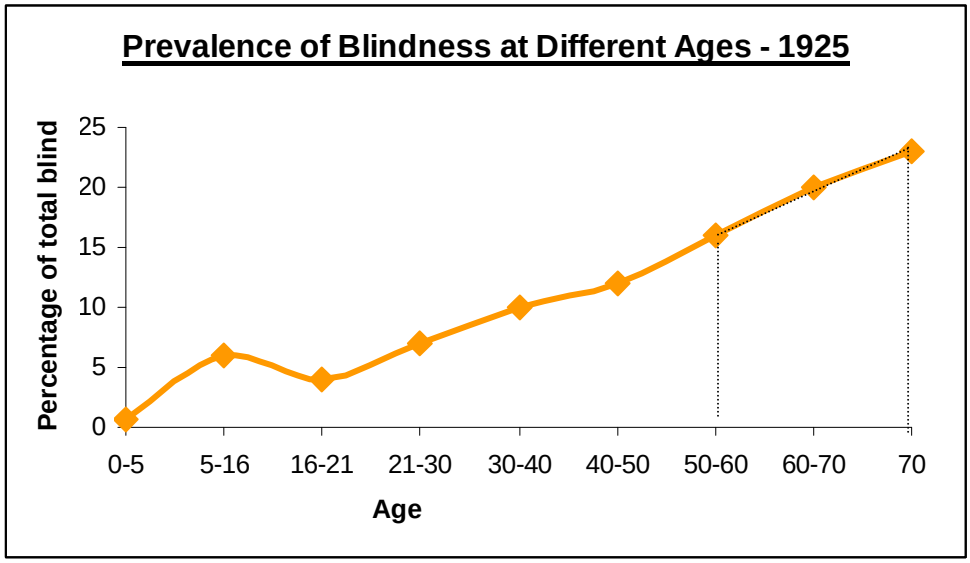
- The graph above illustrates the percentage of blind in each age group in 1901. Between the ages of 35 and approximately 77 were the most cases of blindness. Hence, the most common age of onset of blindness is somewhere between these two ages.
- This implies that during this time, there were two trends for the prevalence of blindness.
    1. The **onset of blindness during middle age, where an individual would be blind for 23 years (using 45 as age of onset of blindness and life expectancy [LE_{45}] in 1900 of 23).**
    2. The second trend was the onset of blindness very late in life, at an age greater than life expectancy, which implies that very few years were spent in blindness on average. This is particularly apparent in the graph above as there is a decline in blindness after age 65 and a substantial decline above the age of 85. This is not likely to be due to improvements in the prevalence, but instead as a result of there being much fewer people at these ages as a result of a relatively low age of life expectancy. **The onset of blindness here is averaged at 71 years of age, where an individual would be blind for 8 years (using 71 as age of onset of blindness and life expectancy [LE_{71}] in 1900 of 9).**

1900:
Row 2: most common ages of onset of blindness and subsequent average number of blind years per registered blind person
Row 3: Percent of blind represented in above age group
Row 4: Number of blind and subsequent total number of blind years for most common ages of onset of blindness (total number of blind years [299747] = {number of blind 1 [10069] * average years of blindness 1 [23] = average years of blindness 1 [231587]} + {number of blind 2 [8520] * average years of blindness 2 [8] = average years of blindness 2 [68160]})

	Average most common age of blindness 1 (Age)	Average most common age of blindness 2 (Age)	LE_x Life Expectancy at age of onset of blindness (Years) 1	2	Average years of blindness 1 (Years)	Average years of blindness 2 (Years)	Total number of blind years for most common ages of onset
	35-55 → 45	65-77 → 71	23	8	23	8	
Percent of blind	39	33					
Number of blind	10069	8520			231587	68160	299747

12.13.1: Years in Blindness

1925

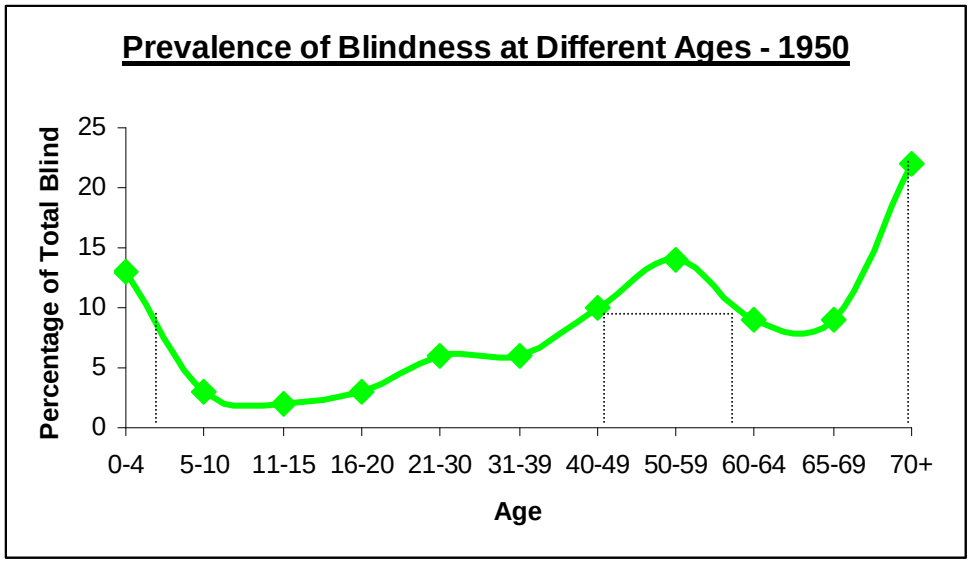


- In 1925 the incidence of blindness above age 16 was a direct function of age; as age increased so did the prevalence of blindness. This means that there was an increasing number of old aged blind and a proportionate decrease in the number of younger blind.
- However as a result of increased life expectancy it was still possible that a blind persons spent many years in this state.
    1. If this onset of blindness occurred at about age 50. **The onset of blindness here is averaged at 55 years of age, where an individual would be blind for 19 years (using 55 as age of onset of blindness and life expectancy [LE_{55}] in 1925 of 19).**
    2. However many more of the blind were increasingly likely to obtain this disability towards the end of their life and therefore spend a much smaller fraction of time with this burden. For example, the most common ages for contracting blindness were those aged 70 and older. **The onset of blindness here is estimated to be 70 years of age, where an individual would be blind for 9 years (using 70 as age of onset of blindness and life expectancy [LE_{70}] in 1925 of 9).**

1925:
Row 2: most common ages of onset of blindness and subsequent average number of blind years per registered blind person
Row 3: Percent of blind represented in above age group
Row 4: Number of blind and subsequent total number of blind years for most common ages of onset of blindness (total number of blind years [371244] = {number of blind 1 [10956] * average years of blindness 1 [19] = average years of blindness 1 [208164]} + {number of blind 2 [18120] * average years of blindness 2 [9] = average years of blindness 2 [163080]})

	Average most common age of blindness 1 (Age)	Average most common age of blindness 2 (Age)	LE_x Life Expectancy at age of onset of blindness (Years) 1	2	Average years of blindness 1 (Years)	Average years of blindness 2 (Years)	Total number of blind years for most common ages of onset
	50	70	19	9	19	9	
Percent of blind	26	43					
Number of blind	10956	18120			208164	68160	371244

1950



- This is a very unusual and unexpected pattern. This unexpected increase was a result of a condition known as Retrolental Fibroplasia and was caused through problems with the administration of oxygen to premature babies, which caused blindness.
- There are three trends of interest here, although only 1 of them seems to be in alignment with the situation during the rest of the twentieth century (increase in the old aged blind).
    1. There is a comparatively high number of blind from birth or very young childhood in 1950. About 12 percent of the 1950 blind cohort spent virtually their entire life in blindness. **The onset of blindness here is at birth: subsequently an individual would be blind for 68 years (using 0 as age of onset of blindness and life expectancy [LE_0] in 1950 of 68).**
    2. A slightly more common trend was the onset of blindness during middle age. From the ages of 40 to 60 there were a sizeable number of individuals contracting blindness. These individuals represent about 24 percent of the blind population in 1950. **The onset of blindness here is averaged at 50 years of age, where an individual would be blind for 24 years (using 50 as age of onset of blindness and life expectancy [LE_{50}] in 1950 of 24).**
    3. Finally, there was an evident trend of very old aged blind. There were about 22 percent of the blind contracting this impairment after age 70. **The onset of blindness here is estimated at 70 years of age, where an individual would be blind for 10 years (using 70 as age of onset of blindness and life expectancy [LE_{70}] in 1950 of 10).**

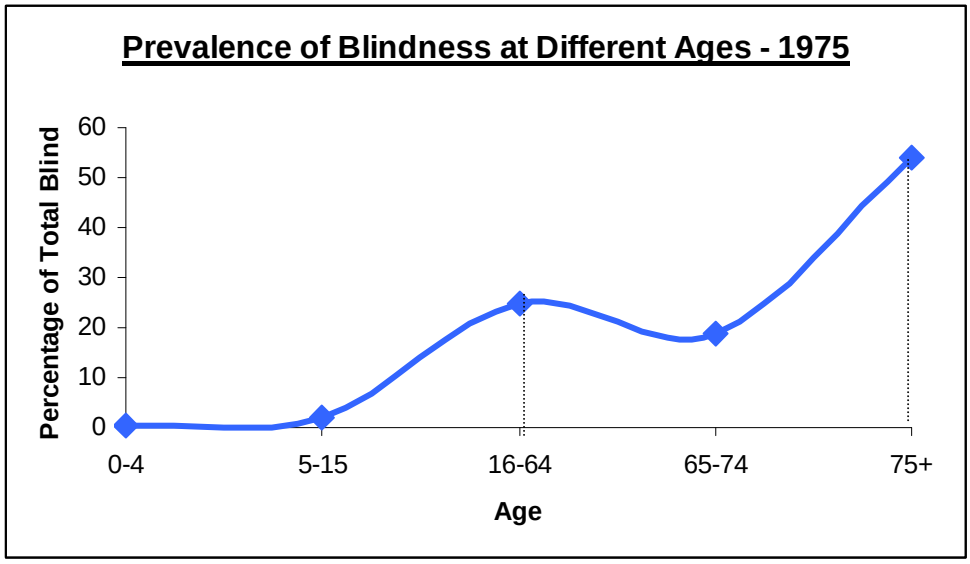
12.13.1: Years in Blindness

1950:
Row 2: most common ages of onset of blindness and subsequent average number of blind years per registered blind person
Row 3: Percent of blind represented in above age group
Row 4: Number of blind and subsequent total number of blind years for most common ages of onset of blindness (total number of blind years [1366204] = {number of blind 1 [10572] * average years of blindness 1 [68] = average years of blindness 1 [718896]} + {number of blind 2 [19517] * average years of blindness 2 [24] = average years of blindness 2 [468408]} + {number of blind 3 [17890] * average years of blindness 3 [10] = average years of blindness 3 [178900]})

	Average most common age of blindness 1 (Age)	Average most common age of blindness 2 (Age)	Average most common age of blindness 3 (Age)	LE_x Life Expectancy at age of onset of blindness (Years)			Average years of blindness 1 (Years)	Average years of blindness 2 (Years)	Average years of blindness 3 (Years)	Total number of blind years for most common ages of onset
				1	2	3				
	0 → 0	40-60 → 50	70 → 70	68	24	10	68	24	10	
Percent of blind	13	24	22							
Number of blind	10572	19517	17890				718896	468408	178900	1366204

12.13.1: Years in Blindness

1975



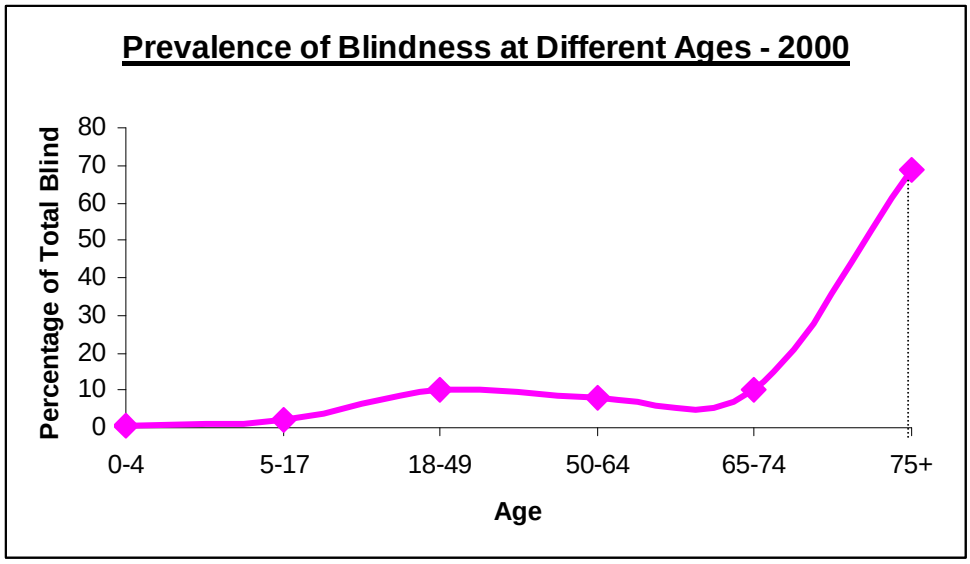
- In 1975 there were two main ages of blind onset.
    1. There were a considerable number (which represents about 25 percent of the blind population in 1975) of blind persons at working age, somewhere between 16 and 64. Due to the format of the data for this period it is not possible to stipulate whether the onset of blindness was at the beginning or end of this age group, although from what is available (18-49=10% and 50-64 = 8%) it seems best to adopt a conservative estimate for the average age (during working age) of onset of blindness, for example 50 years old. Hence, **the onset of blindness here is estimated at 50 years of age, where an individual would be blind for 26 years (using 50 as age of onset of blindness and life expectancy [LE_{50}] in 1975 of 26).**
    2. The second trend which is very noteworthy, in relation to all the previous years, is the substantial increase in the number of blind aged 75 and older. Approximately 54 percent of the blind population contracted this impairment in the last few years of life. **The onset of blindness here is estimated at 75 years of age, where an individual would be blind for 9 years (using 75 as age of onset of blindness and life expectancy [LE_{75}] in 1975 of 9).**

1975:
Row 2: most common ages of onset of blindness and subsequent average number of blind years per registered blind person
Row 3: Percent of blind represented in above age group
Row 4: Number of blind and subsequent total number of blind years for most common ages of onset of blindness (total number of blind years [1131447] = {number of blind 1 [24900] * average years of blindness 1 [26] = average years of blindness 1 [647400} + {number of blind 2 [53783] * average years of blindness 2 [9] = average years of blindness 2 [484047]})

	Average most common age of blindness 1 (Age)	Average most common age of blindness 2 (Age)	LEx Life Expectancy at age of onset of blindness (Years) 1	2	Average years of blindness 1 (Years)	Average years of blindness 2 (Years)	Total number of blind years for most common ages of onset
	50	75+	26	9	26	9	
Percent of blind	25	54					
Number of blind	24900	53783			647400	484047	1131447

12.13.1: Years in Blindness

2000



- The graph above highlights what would appear to be a considerable improvement in the conditions surrounding blindness. This is result of a massive increase in the average age of onset of blindness.
    1. There is only one situation above, and this represents 69 percent of the blind population who were aged 75 and older in 2000. This means that the vast majority of the blind population were only in this less than optimal state for the last years of life. Hence by 2000 there has been an important reduction in the disabling and adverse effects of blindness, as it is now an old age disability rather than a burden that could strike at any age during the most productive years of life. **The onset of blindness here is estimated at 75 years of age, where an individual would be blind for 11 years (using 75 as age of onset of blindness and life expectancy [LE_{75}] in 2000 of 11).**

2000:
Row 2: most common ages of onset of blindness and subsequent average number of blind years per registered blind person
Row 3: Percent of blind represented in above age group
Row 4: Number of blind and subsequent total number of blind years for most common ages of onset of blindness (total number of blind years [1197856] = {number of blind 1 [108896] * average years of blindness 1 [11] = average years of blindness 1 [1197856]})

	Average most common age of blindness 1 (Age)	LE_x Life Expectancy at age of onset of blindness (Years)	Average years of blindness 1 (Years)	Total number of blind years for most common ages of onset
	75+ → 75	11	11	
Percent of blind	69			
Number of blind	108896		1197856	1197856

- The above situations and the distribution and average years of blindness for 1900, 1925, 1950, 1975 and 2000 are summarised in the tables below: summary table for estimates of the average number of years spent in blindness during the twentieth century and the percentage and number of blind this represented.

Average number of years spent in blindness during the twentieth century.

Year	**Average most common age of onset of blindness 1 (Age)**	**Average most common age of onset of blindness 2 (Age)**	**Average most common age of onset of blindness 3 (Age)**	**LEx Life Expectancy at age of onset of blindness (Years)**			**Average number of years of blindness 1 (Years)**	**Average number of years of blindness 2 (Years)**	**Average number of years of blindness 3 (Years)**
1900	35-55 → 45	65-77 → 71		LE_{45} = 23	LE_{71} = 8		23	8	
1925	55 → 55	70+ → 70		LE_{55} = 19	LE_{70} = 9		19	9	
1950	0 → 0	40-60 → 50	70+ → 70	LE_{0} = 68	LE_{50} = 24	LE_{70} = 10	68	24	10
1975	50 → 50	75+ → 75		LE_{50} = 26	LE_{75} = 9		26	9	
2000	75+ → 75			LE_{75} = 11			11		

Sources:

Calculated from: 1901 Census and 1925, 1950, 1975, 2000 Local Authority Blind Returns

Life expectancy by age data: Figures based on the 'Office of National Statistics/Government Actuaries Department' England and Wales mortality database. See Appendix 12.15 for a copy of exert.

Percent and number of blind population represented and subsequent number of blind years from most common ages of onset of blindness

12.13.1: Years in Blindness

Year	Blind at common age of blindness onset 1		Blind at common age of blindness onset 2		Blind at common age of blindness onset 3		Total number of blind years (from life expectancy at most common ages of onset)
	Percent	Number	Percent	Number	Percent	Number	
1900	39	10069	33	8520			(10069*23=231587) + (8520*8=68160) = **299,747**
1925	26	10956	43	18120			(10956*19=208164) + (18120*9=163080) = **371,244**
1950	13	10572	24	19517	22	17890	(10572*68=718896) + (19517*24=468408) + (17890*10=178900) = **1,366,204**
1975	25	24900	54	53783			(24900*26=647400) + (53783*9=484047) = **1,131,447**
2000	69	108896					(108896*11) = **1,197,856**

The table highlights the developments that have been made in preventing or delaying the onset of blindness. As a result, by 2000 the most common age of onset of blindness (by a significant majority 69 percent) was older than 75. When an individual contracts blindness at this age they are only burdened with it for a few years. This has meant there has actually been a reduction in the average number of blind years.

12.13.2: Cohort Effect Calculations

- There was a substantial increase in the average age of blindness during the twentieth century.
- It is crucial to determine whether this is a genuine effect or a result of the ageing of many blind. I.e. the increase in the 75+ population with blindness in 2000 could be a result of the 1975, 50+ age group getting older.
- This will be achieved through the following process:
    1. Identifying the age group where blindness was most prevalent in time T.
    2. Determining the age group that this would have been in the previous (-25 years) time period = T-1.
    3. Identifying the number of blind people in the most common age group in time T.
    4. Identifying the number of blind people in the most common age group in time T-1.
    5. Calculating what percentage of the number in T-1 as a percentage of T to identify the proportion of the blind in T that had also been in T-1.
- E.g. for T = 1925 where the most common age group was 16-64. Period T-1 = 1900 and T-1 population age would be -9 to 39, i.e. 0-39 (as in 25 years time this would be the 16-25 age group). The number of blind aged between 0 and 39 in 1900 was 8676 and the number aged 16-64 in 1925 was 24723. Therefore 8676/24723*100 = 35.09 → 35 percent of the 1925 blind are carried over from 1900. Hence, the cohort effect is 35 percent.
- These calculations have been done for all of the necessary years and are shown in the table below.

Year (T)	Most common age group	Period T-1	Age in period T-1	Number of blind in age group in T	Number of blind in age group in T-1	Cohort effect (%) % in T that are from T-1
1900	16-64	-	-	-	-	-
1925	16-64	1900	0-39	24723	8676	35
1950	16-64	1925	0-39	39472	12027	30
1974	65+	1950	40-60	71117	19800	28
1975		1950				(25)*
1976	75+	1950	50-60	54638	11394	21

* This is a mid point between the 1974 and 1976 estimates that is necessary due to data constraints for 1975

12.13.3: Blind Income Details

This Appendix provides the detailed tables about blind: pensions, benefits and wages, that underlie Table 4.2 in Chapter 4 of the thesis.

Table 12.13.3.1: The rate of weekly pension payable to the blind aged 50 to 70 years old under 1920 Blind Persons Act

Means of claimant or pensioner				**Weekly pension rate**
Annual means of the claimant:				
Do not exceed £26.25				50p
Exceed £26.25, but do not exceed £36.50				40p
"	£36.50	"	£36.75	30p
"	£36.75	"	£42.00	20p
"	£42.00	"	£47.25	10p
"	£47.25	"	£49.88	5p
Annual means of more than £49.88				No pension

Sources:
Parliamentary Bills, "Allowances under the 1920 Blind Persons Act"

Table 12.13.3.2: Weekly pensions and other payments to soldiers and sailors blinded in the Great War

Rank	**Degree of blindness**	**Weekly pension**	**Other payments**
Senior Officer	Complete loss of sight		£300 p.a. +
		25p for retraining	
Officer	Complete loss of sight		£175 p.a.
		25p for retraining	
NCO and Men	Complete loss of sight	£1.38 - £2.13	
		25p for retraining	
	Loss of vision in 1 eye	£0.69 - £1.06	
		25p for retraining	
	Alternative pension based on pre-war earnings	£1.38 - £3.75	
		25p for retraining	

Sources:
Parliamentary report of the departmental committee on the welfare of the blind

Table 12.13.3.3: 1948 National Assistance Act: weekly rates of allowance (excluding rents) for blind persons.

Type of blind individual	**Weekly rate of allowance (excluding rent)**
Husband and wife:	
1. Of whom 1 is blind	£2.75
2. Of whom, both are blind	£3.25
Single person:	
1. Aged 21 or over	£1.95
2. Aged 18 years or over but less than 21	£1.50
3. Aged 16 years or over but less than 18	£1.25

Sources:
Beacon 1948: National Assistance Rates

12.13.3: Blind Income Details

Table 12.13.3.4: Average wages for blind home workers in various industries 1924 and 1933

Trade	**General average earnings per week (1924)**	**General average earnings per week (1933)**
Basket makers	88p	90p
Boot repairers	13p	79p
Carpenters	£1.02	40p
Chair caners	50p	58p
Fancy basket makers	36p	57p
Hand knitters	36p	31p
Machine knitters	40p	52p
Mat makers	43p	89p
Music teachers	£1.00	97p
Piano tuners	£1.20	£1.37
Straw basket makers	56p	-
Weavers	84p	35p
Wood choppers	75p	90p

Sources:
Beacon 1924: Home Industries for the Blind and Beacon 1933: The Social and Economic Value of Home working Schemes

Table 12.13.3.5: Comparison of average weekly wages between blind and mainstream industries 1924, 1933 and 1935.

Industry	**Male / Female weekly wage average 1924**	**Male / Female weekly wage average 1933 and 1935**
Blind Home workers average	65p	71p (1933)
Unskilled mainstream industry	£1.99 (33%)	£2.09 (1935) (34%)
Semi-skilled mainstream industry	£2.25 (29%)	£2.35 (1935) (30%)

Sources:
Blind home worker wage data: Beacon 1924 and 1933 and mainstream industry wage data; Routh, "Occupation and Pay in Great Britain 1906-79"

Table 12.13.3.6: Comparison of average weekly wages between blind workshops, blind in mainstream industries and the able bodied population 1935, 1944 and 1955.

Industry	**Male/Female weekly wage average 1938**	**Male/Female weekly wage average 1944**	**Male/Female weekly wage average 1955**
Blind Workshop		£3.49	
Blind in mainstream industry		£3.27	
Able bodied in mainstream industry	£2.72		£7.70

Sources:
1938: Chapman, "Wages and Salaries in the United Kingdom 1920-1938" and 1944: TNA: MH 55 (1089) and 1955: Routh, "Occupation and Pay in Great Britain 1906-79"

12.14 Extended Results

This Appendix relates to Tables 9.2, 9.3, and 9.4 in Chapter 9 of the thesis. The calculations here represent variations of those that are presented throughout the thesis. The QALE gain methodology is consistent here but the data that is used differs from that which has been presented above. This is because Chapter 9 (and this corresponding appendix) considers the entire burden of morbidity through extrapolating forward the findings about the thesis disease and disability morbidity (contained in the previous appendices).

12.14.1: Lowest Bound Estimates for Key Morbidity Categories

Appendix 12.14.1 presents the lowest morbidity gain estimates for the extended results illnesses. This utilises the methodology applied in Appendix 12.3 and 12.4. The values that have been utilised here are the Low VSL and VSHLY (presented in Appendix 12.1) and the Low QALY (presented in Appendix 12.6).

The infectious morbidity gain is equal to the lowest tuberculosis morbidity gain (as a result of the conservative assumption of Chapter 9: that tuberculosis was the only infectious disease to experience a morbidity gain), shown in Appendix 13.4. The disability morbidity gain is also equal to the lowest blind morbidity gain in the thesis. This is equal to the blind morbidity gain shown in Appendix 12.7.2 (for an explanation see Table 9.1).

The non-infectious morbidity gain is an additional calculation to the previous appendix. This applied the ('Low') stomach cancer VSHLY and QALY profile to the aggregate number of non-infectious deaths in twentieth century England. This is because of the conservative assumptions in Chapter 9 (see Table 9.1 for details).

Sources:

- Appendix 12.2 for VSL and VSHLY information (utilising Low)
- Appendix 12.3 for the change in the morbidity burden calculations
- Appendix 12.4 for the morbidity gain calculation
- Appendix 12.10 for the age-weighted mortality, morbidity and QALE gain calculations Death and population data
- Office of National Statistics, "Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause"

12.14.2: Value of QALE Gain relative to GDP per capita growth: Extended Results: Disease and Disability

After the morbidity gain has been identified (in Appendix 12.14.1) this can be combined with the mortality gain calculation, which represents the lowest age-weighted estimated (presented in Appendix 12.8) to estimate the QALE gain, or given the underlying assumptions of this appendix, the aggregate QALE gain.

GDP per capita growth has been calculated in Appendix 12.11. Appendix 12.12 calculated the mortality, morbidity and QALE gain relative to GDP growth. This same methodology and process is conducted here, but for the different morbidity and QALE gain values that were yielded in Appendix 12.14.1.

12.14: Extended Results

Sources:
GDP per capita data
- Maddison, “Monitoring the World Economy 1820-1992”
- Maddison, “The World Economy: A Millennial Perspective”

Compound average growth rate methodology
- Dowling, “Mathematical Methods for Business and Economics”

QALE Gain numbers
- Appendix 12.14.1

12.14.3: Value of morbidity (QALY) improvements

This appendix readdresses the considerations made in Appendix 12.14.1, but holds all QALY gain (from 1900 onwards) constant in order to identify the value of the twentieth century (1900-2000) health improvements. This is then compared to actual QALE gain (including 1900-2000 QALY improvements) identified in Appendix 12.14.2, in order to identify the value of health improvements for the most conservative twentieth century disease environment.

Sources:
- Appendix 12.3 for the change in the morbidity burden calculations
- Appendix 12.4 for the morbidity gain calculations
- Appendix 12.10 for the age-weighted mortality, morbidity and QALE gain calculations

12.14.1: Lowest Bound Estimates for Key Morbidity Categories

Infectious Morbidity Gain

Period	Morbidity Gain
1900-2000	**11047**

Tuberculosis Morbidity Gain (Low VSHLY, Low QALY, Age-Weighted)
1900-2000

Age	Weight	VSHLY (Mid)	Weighted VSL (Mid)	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2	Average Mortality Gain
0-4	0.3	0.288375099	0.08651253	32963.83056	17525.39089	2851.78437	1516.1659	
5-14	1	0.288375099	0.288375099	13947.83794	8614.582243	4022.209147	2484.231007	
15-24	1.5	0.288375099	0.432562649	6235.885789	3823.347706	2697.411273	1653.83741	
25-34	1.5	0.288375099	0.432562649	3127.668735	2817.606638	1352.912672	1218.79139	
35-44	1.3	0.288375099	0.374887629	2431.599597	2905.666277	911.5766069	1089.298341	
45-64	1	0.288375099	0.288375099	2605.842774	4172.730281	751.4601679	1203.311508	
65-74	0.7	0.288375099	0.201862569	413.6437681	696.7556916	83.49919381	140.6488941	
75+	0.45	0.288375099	0.129768795	138.382849	766.2670101	17.9577755	99.43754621	
				61864.69201	41322.34673	12688.81121	9405.721995	**11047**

12.14.1: Extended Results: Lowest Bound Estimated for Key Morbidity Categories

Non-Infectious Morbidity Gain

Period	Morbidity Gain
1900-2000	-**7467**

Stomach Cancer				Non-Infectious Disease					
Year	Deaths percent	(1-QALY)	Survival percent	Year	Deaths Percent	Survival/Prevalence percent	(1-QALY)	Burden	Morbidity Burden Change
1900	85		15	1900	85	15			
2000	75		25	2000	75	25			
		0.6667		1900	97760	17252	0.6667	11502	
		0.3333		2000	391518	130506	0.3333	43498	-31996

Summary Morbidity Gain

Summary Morbidity Gain

Period	Morbidity Burden Change	Low VSL	Low QALY 1900-2000	VSHLY Millions	Morbidity Gain millions
1900-2000	-31996	0.7	0.3334	0.2334	-7467

Sources: non-infectious deaths are calculated as all deaths not included in infectious and residual. Death data provided by: Office of National Statistics (2003) “Twentieth Century Mortality: 100 Years of Mortality Data in England and Wales by Age, Sex, Year and Underlying Cause”

Disabled Morbidity Gain

Blind Morbidity Gain

Low VSL Low VSHLY Low QALY

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions
1900-2000	-7976	0.35	**-2792**

12.14.2: Value of QALE Gain relative to GDP per capita growth: Extended Results: Disease and Disability

Mortality						
Period	t	Mortality Gain	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	7372	1950	6907	107	1.1
Infectious						
Period	t	Morbidity Gain: Infectious	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	11047	1950	6907	160	1.6
	t	QALE Gain: Infectious	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	18419	1950	6907	267	2.7
Non-Infectious						
Period	t	Morbidity Gain: Infectious	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	-7467	1950	6907	-108	-1.1
	t	QALE Gain: Infectious	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	-95	1950	6907	-1.4	-0.01
Disability						
Period	t	Morbidity Gain: Infectious	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	-2792	1950	6907	-40	-0.4
	t	QALE Gain: Infectious	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	6353	1950	6907	80	0.8
Total						
Period	t	Morbidity Gain: Total	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	788	1950	6907	11	0.1
	t	QALE Gain: Total	Midpoint	GDPpc at Midpoint	Gain as a proportion of GDP (total/per annum)	
1900-2000	100	8160 (7372+11047-7467-2792)	1950	6907	118	1.2

12.14.3: Value of morbidity (QALY) improvements

Infectious Morbidity Gain

Morbidity Gain Health Improvements	Morbidity Gain No Heath Improvements	Value of Infectious Health Improvements
11047	**506**	**10541**

Infectious Morbidity Gain: (Health Improvements)

Period	Morbidity Gain
1900-2000	11047

Infectious Morbidity Gain: (No Health Improvements)

Period	Morbidity Gain
1900-2000	506

Summary Morbidity Gain

Period	Morbidity Rate Change 1	Morbidity Rate Change 2	Low VSL	Low QALY 1900-2000	VSHLY millions	Morbidity Gain 1 millions
1900-2000	5045	3631	0.7	0.1667	0.11669	589

Age-Weighted Morbidity Gain
Tuberculosis 1900-2000
1900-2000: Constant QALY

Age	Weight	VSHLY (Mid)	Weighted VSL (Mid)	Fall in MBR 1	Fall in MBR 2	Morbidity Gain 1	Morbidity Gain 2
0-4	0.3	0.1167	0.03501	32951.40206	17518.78321	1153.628586	613.3326002
5-14	1	0.1167	0.1167	13926.55707	8601.366887	1625.229211	1003.779516
15-24	1.5	0.1167	0.17505	6214.341868	3810.13778	1087.820544	666.9646183
25-34	1.5	0.1167	0.17505	3091.205943	2784.758592	541.1156002	487.4719916
35-44	1.3	0.1167	0.15171	2329.368277	2783.503855	353.3884614	422.2853698
45-64	1	0.1167	0.1167	2353.35804	3751.769479	274.6368832	437.8314982
65-74	0.7	0.1167	0.08169	169.9526224	286.2740021	13.88342972	23.38572323
75+	0.45	0.1167	0.052515	-83.28082287	-461.1506962	-4.373492413	-24.21732881

12.14.3: Value of morbidity (QALY) improvements

Morbidity Burden Change

Tuberculosis

1900-2000: Constant QALY

1900			2000		
Age	% Pop	MBR/1000	Age	% Pop	MBR/1000
0-4	0.114068091	6.840053763	0-4	0.06064489	0.003531775
5-9	0.10708602	1.896744266	5-9	0.064774207	0.003306627
10-14	0.102584623	1.239575549	10-14	0.065448618	0.003272554
15-19	0.099601068	0.933440059	15-19	0.061064951	0
20-24	0.095740538	0.572334497	20-24	0.058704516	0.007294835
25-34	0.161673121	0.459554291	25-34	0.145645622	0.007057392
35-44	0.12315061	0.473830985	35-44	0.147160155	0.026192846
45-54	0.089448395	0.387028213	45-54	0.132294228	0.027517142
55-64	0.059845885	0.485218015	55-64	0.104828392	0.091925078
65-74	0.033199334	0.35282165	65-74	0.055922092	0.23167149
75+	0.013602313	0.369702435	75+	0.075320056	0.514598878
				0.971807728	0.916368616

Age	% Pop 1900	MBR/1000 1900	WTD MBR 1900&1900	% Pop 1900	MBR/1000 2000	WTD MBR 1900&2000	Decrease/1000	**Morbidity Rate Change**
0-4	0.114068091	6.840053763	0.780231877	0.114068091	0.003531775	0.000402863	0.779829014	**32951**
5-9	0.10708602	1.896744266	0.203114795	0.10708602	0.003306627	0.000354093	0.202760701	**8568**
10-14	0.102584623	1.239575549	0.127161391	0.102584623	0.003272554	0.000335714	0.126825677	**5359**
15-19	0.099601068	0.933440059	0.092971627	0.099601068	0	0	0.092971627	**3928**
20-24	0.095740538	0.572334497	0.054795613	0.095740538	0.007294835	0.000698411	0.054097201	**2286**
25-34	0.161673121	0.459554291	0.074297577	0.161673121	0.007057392	0.001140991	0.073156586	**3091**
35-44	0.12315061	0.473830985	0.058352575	0.12315061	0.026192846	0.003225665	0.05512691	**2329**
45-54	0.089448395	0.387028213	0.034619052	0.089448395	0.027517142	0.002461364	0.032157688	**1359**
55-64	0.059845885	0.485218015	0.029038302	0.059845885	0.091925078	0.005501338	0.023536964	**995**
65-74	0.033199334	0.35282165	0.011713444	0.033199334	0.23167149	0.007691339	0.004022105	**170**
75+	0.013602313	0.369702435	0.005028808	0.013602313	0.514598878	0.006999735	-0.001970927	**-83**
					0.916368616	0.916368616	**1.442513547**	**60953**

12.14.3: Value of morbidity (QALY) improvements

Age	% Pop 2000	MBR/1000 1900	WTD MBR 2000&1900	% Pop 2000	MBR/1000 2000	WTD MBR 2000&2000	Decrease/1000	**Morbidity Rate Change**
0-4	0.06064489	6.840053763	0.41481431	0.06064489	0.003531775	0.000214184	0.414600126	**17519**
5-9	0.064774207	1.896744266	0.122860107	0.064774207	0.003306627	0.000214184	0.122645922	**5182**
10-14	0.065448618	1.239575549	0.081128506	0.065448618	0.003272554	0.000214184	0.080914322	**3419**
15-19	0.061064951	0.933440059	0.057000472	0.061064951	0	0	0.057000472	**2409**
20-24	0.058704516	0.572334497	0.03359862	0.058704516	0.007294835	0.00042824	0.03317038	**1402**
25-34	0.145645622	0.459554291	0.066932071	0.145645622	0.007057392	0.001027878	0.065904193	**2785**
35-44	0.147160155	0.473830985	0.069729041	0.147160155	0.026192846	0.003854543	0.065874498	**2784**
45-54	0.132294228	0.387028213	0.051201599	0.132294228	0.027517142	0.003640359	0.047561239	**2010**
55-64	0.104828392	0.485218015	0.050864624	0.104828392	0.091925078	0.009636358	0.041228266	**1742**
65-74	0.055922092	0.35282165	0.019730525	0.055922092	0.23167149	0.012955554	0.00677497	**286**
75+	0.075320056	0.369702435	0.027846008	0.075320056	0.514598878	0.038759616	-0.010913608	**-461**
						0	**0.92476078**	**39075**

12.14.3: Value of morbidity (QALY) improvements

Non-Infectious

Morbidity Gain Health Improvements	**Morbidity Gain No Heath Improvements**	**Value of Infectious Health Improvements**
-7467	**-8811**	**1344**

Infectious Morbidity Gain: (Health Improvements)

Period	Morbidity Gain
1900-2000	-7467

Infectious Morbidity Gain: (No Health Improvements)

Period	Morbidity Gain
1900-2000	-8811

Period	Morbidity Burden Change	Low VSL	Low QALY 1900-2000	Low VSHLY	Morbidity Gain millions
1900-2000	-75506	0.7	0.1667	0.11669	-8811

Non-Infectious Disease Year	Deaths percent	Survival/Prevalence Percent	(1-QALY)	Burden	Morbidity Burden Change
1900	85	15			
2000	75	25			
1900	97760	17252	0.6667	11502	
2000	391518	130506	0.6667	87008	-75506

12.14.3: Value of morbidity (QALY) improvements

Disability

Morbidity Gain Health Improvements	Morbidity Gain No Heath Improvements	Value of Disability Health Improvements
-2792	**-23950**	**22111**

Blind Morbidity Gain

Low VSL Low VSHLY Low QALY

Period	Morbidity Burden Change	VSHLY millions	Morbidity Gain millions	
1900-2000	-7976	0.35	-2792	
Blind Year	Prevalence percent	(1-QALY)	Burden	Morbidity Burden Change
1900	11990	0.6667	7994	
2000	47914	0.6667	31944	-23950

12.15: Life Expectancy by Age

This Appendix relates to Tables 4.10 and 4.11 and 8.2.1 in the main thesis.

This Appendix contains life expectancy by age estimates for the reference years used in the thesis (1900, 1925, 1950, 1975, and 2000). These figures are based on the ONS/GAD England & Wales mortality database.

It should be noted that this database is subject to further revision and that this dataset should be used for internal research/work purposes only. This dataset represents the best available estimates for life expectancy by age.

Sources:
Life expectancy by age data:

- Figures based on the 'Office of National Statistics/Government Actuaries Department' England and Wales mortality database. Extract was provided by Mita Saha (Office of National Statistics) on March 17 2006.

12.15: Life Expectancy by Age

England and Wales: Period expectation of life (years)

	Males					Females				
Age	**1900**	**1925**	**1950**	**1975**	**2000**	**1901**	**1925**	**1950**	**1975**	**2000**
0	43.72	56.38	66.09	69.70	75.68	48.83	60.24	70.61	75.85	80.41
1	51.49	60.44	67.38	69.93	75.14	55.46	63.39	71.47	75.91	79.81
2	53.33	60.86	66.65	69.01	74.17	57.17	63.77	70.73	74.98	78.84
3	53.46	60.49	65.76	68.05	73.19	57.24	63.39	69.84	74.01	77.85
4	53.18	59.88	64.84	67.09	72.21	56.94	62.79	68.91	73.04	76.86
5	52.71	59.17	63.90	66.12	71.22	56.46	62.08	67.97	72.07	75.87
6	52.09	58.40	62.97	65.15	70.22	55.84	61.32	67.03	71.09	74.88
7	51.36	57.58	62.03	64.17	69.24	55.12	60.51	66.08	70.11	73.89
8	50.56	56.72	61.08	63.19	68.24	54.33	59.65	65.12	69.13	72.89
9	49.71	55.84	60.13	62.21	67.25	53.49	58.76	64.15	68.14	71.90
10	48.82	54.93	59.17	61.23	66.26	52.63	57.85	63.19	67.16	70.91
11	47.93	54.02	58.20	60.25	65.27	51.75	56.94	62.21	66.17	69.92
12	47.03	53.11	57.24	59.27	64.28	50.87	56.03	61.24	65.18	68.92
13	46.15	52.20	56.27	58.29	63.29	49.99	55.12	60.27	64.19	67.93
14	45.27	51.29	55.31	57.31	62.30	49.12	54.22	59.30	63.20	66.94
15	44.39	50.39	54.35	56.33	61.31	48.25	53.33	58.34	62.21	65.95
16	43.52	49.49	53.39	55.35	60.33	47.39	52.45	57.38	61.23	64.95
17	42.67	48.61	52.44	54.39	59.35	46.54	51.58	56.42	60.25	63.97
18	41.84	47.74	51.49	53.45	58.38	45.71	50.71	55.47	59.28	62.98
19	41.02	46.88	50.55	52.51	57.42	44.87	49.85	54.53	58.30	62.01
20	40.20	46.02	49.61	51.56	56.47	44.03	48.99	53.59	57.32	61.02
21	39.39	45.17	48.68	50.62	55.51	43.20	48.14	52.65	56.35	60.04
22	38.58	44.33	47.74	49.68	54.56	42.36	47.29	51.71	55.37	59.06
23	37.79	43.48	46.81	48.73	53.60	41.53	46.43	50.77	54.39	58.08
24	37.00	42.63	45.87	47.77	52.64	40.71	45.58	49.84	53.41	57.10
25	36.21	41.78	44.94	46.81	51.68	39.88	44.73	48.91	52.44	56.11
26	35.42	40.94	44.01	45.85	50.73	39.07	43.88	47.97	51.46	55.13
27	34.63	40.09	43.09	44.89	49.77	38.25	43.02	47.04	50.49	54.15
28	33.85	39.24	42.16	43.92	48.81	37.44	42.17	46.11	49.51	53.17
29	33.07	38.39	41.23	42.96	47.86	36.64	41.31	45.19	48.54	52.19
30	32.29	37.54	40.31	41.99	46.91	35.84	40.46	44.26	47.56	51.21
31	31.51	36.68	39.38	41.03	45.95	35.04	39.60	43.33	46.59	50.23
32	30.74	35.82	38.45	40.07	45.00	34.25	38.74	42.40	45.62	49.25
33	29.97	34.97	37.53	39.11	44.05	33.46	37.88	41.47	44.65	48.28
34	29.22	34.12	36.60	38.16	43.09	32.68	37.02	40.54	43.68	47.30
35	28.47	33.28	35.68	37.20	42.14	31.90	36.17	39.62	42.71	46.33
36	27.73	32.45	34.75	36.25	41.19	31.13	35.32	38.69	41.75	45.36
37	27.00	31.62	33.83	35.30	40.24	30.36	34.47	37.77	40.79	44.40
38	26.28	30.79	32.92	34.35	39.30	29.61	33.63	36.85	39.83	43.43
39	25.57	29.97	32.01	33.40	38.35	28.86	32.78	35.93	38.88	42.47
40	24.86	29.16	31.10	32.46	37.41	28.12	31.94	35.02	37.93	41.50
41	24.17	28.35	30.19	31.52	36.47	27.38	31.09	34.10	36.98	40.54
42	23.49	27.55	29.29	30.59	35.53	26.65	30.25	33.19	36.05	39.59

12.15: Life Expectancy by Age

England and Wales: Period expectation of life (years)…continued…

	Males					Females				
Age	**1900**	**1925**	**1950**	**1975**	**2000**	**1901**	**1925**	**1950**	**1975**	**2000**
43	22.81	26.75	28.40	29.67	34.60	25.93	29.41	32.29	35.12	38.64
44	22.13	25.96	27.52	28.76	33.66	25.21	28.57	31.38	34.19	37.69
45	21.46	25.17	26.64	27.86	32.73	24.49	27.74	30.49	33.27	36.75
46	20.80	24.40	25.78	26.96	31.81	23.77	26.92	29.60	32.35	35.81
47	20.15	23.63	24.94	26.08	30.89	23.06	26.11	28.72	31.44	34.87
48	19.50	22.86	24.10	25.21	29.99	22.35	25.30	27.85	30.54	33.94
49	18.87	22.10	23.27	24.34	29.08	21.65	24.50	26.99	29.65	33.01
50	18.24	21.34	22.46	23.50	28.19	20.95	23.70	26.13	28.77	32.09
51	17.63	20.58	21.65	22.67	27.31	20.26	22.91	25.27	27.89	31.18
52	17.02	19.83	20.85	21.85	26.43	19.58	22.12	24.43	27.03	30.26
53	16.42	19.09	20.07	21.04	25.54	18.91	21.35	23.59	26.17	29.36
54	15.83	18.36	19.30	20.24	24.68	18.24	20.58	22.75	25.31	28.46
55	15.25	17.65	18.55	19.46	23.81	17.59	19.82	21.93	24.46	27.56
56	14.67	16.95	17.81	18.68	22.95	16.94	19.08	21.11	23.61	26.68
57	14.11	16.27	17.08	17.92	22.12	16.30	18.35	20.30	22.79	25.80
58	13.55	15.59	16.37	17.18	21.29	15.67	17.63	19.50	21.96	24.93
59	13.02	14.93	15.68	16.45	20.46	15.05	16.91	18.71	21.14	24.06
60	12.49	14.27	15.01	15.74	19.66	14.45	16.20	17.94	20.33	23.20
61	11.98	13.63	14.36	15.04	18.87	13.87	15.49	17.17	19.53	22.35
62	11.48	13.00	13.72	14.37	18.10	13.29	14.80	16.42	18.74	21.51
63	10.99	12.38	13.10	13.71	17.33	12.73	14.12	15.68	17.96	20.67
64	10.51	11.79	12.50	13.07	16.57	12.18	13.46	14.96	17.19	19.84
65	10.03	11.21	11.91	12.44	15.83	11.64	12.82	14.25	16.45	19.04
66	9.57	10.66	11.33	11.84	15.11	11.13	12.20	13.55	15.71	18.23
67	9.12	10.13	10.77	11.26	14.40	10.62	11.60	12.87	14.98	17.45
68	8.69	9.62	10.23	10.70	13.71	10.13	11.01	12.20	14.26	16.67
69	8.27	9.11	9.70	10.15	13.04	9.65	10.43	11.56	13.56	15.91
70	7.86	8.62	9.19	9.63	12.39	9.19	9.87	10.93	12.87	15.16
71	7.47	8.13	8.69	9.13	11.76	8.74	9.33	10.33	12.20	14.43
72	7.10	7.66	8.21	8.64	11.14	8.30	8.80	9.75	11.55	13.72
73	6.74	7.20	7.75	8.18	10.56	7.89	8.29	9.19	10.91	13.03
74	6.39	6.77	7.30	7.75	10.00	7.48	7.81	8.65	10.30	12.35
75	6.06	6.37	6.88	7.35	9.47	7.10	7.36	8.14	9.72	11.70
76	5.72	6.00	6.47	6.96	8.95	6.73	6.92	7.66	9.15	11.06
77	5.40	5.66	6.09	6.60	8.44	6.38	6.52	7.20	8.60	10.45
78	5.08	5.34	5.72	6.23	7.96	6.04	6.13	6.77	8.08	9.85
79	4.78	5.04	5.37	5.88	7.50	5.72	5.77	6.36	7.58	9.26
80	4.50	4.75	5.03	5.56	7.06	5.41	5.42	5.97	7.10	8.70
81	4.23	4.46	4.70	5.23	6.64	5.10	5.10	5.61	6.66	8.17
82	3.97	4.18	4.39	4.92	6.18	4.80	4.80	5.27	6.23	7.61
83	3.72	3.91	4.10	4.63	5.79	4.52	4.50	4.94	5.84	7.11
84	3.50	3.67	3.83	4.38	5.43	4.26	4.22	4.64	5.47	6.64
85	3.29	3.47	3.59	4.12	5.10	4.03	3.97	4.35	5.13	6.20
86	3.10	3.29	3.38	3.88	4.79	3.82	3.73	4.08	4.80	5.79

12.15: Life Expectancy by Age

England and Wales: Period expectation of life (years)…continued…

	Males					Females				
Age	**1900**	**1925**	**1950**	**1975**	**2000**	**1901**	**1925**	**1950**	**1975**	**2000**
87	2.93	3.13	3.18	3.65	4.50	3.62	3.52	3.85	4.50	5.39
88	2.77	2.98	2.98	3.43	4.22	3.43	3.34	3.63	4.21	5.01
89	2.62	2.83	2.79	3.22	3.96	3.26	3.17	3.41	3.93	4.67
90	2.47	2.69	2.62	3.04	3.71	3.09	3.00	3.22	3.67	4.33
91	2.34	2.54	2.46	2.86	3.48	2.93	2.84	3.03	3.45	4.03
92	2.21	2.40	2.30	2.67	3.24	2.77	2.66	2.86	3.23	3.74
93	2.09	2.31	2.15	2.51	3.04	2.63	2.48	2.70	3.03	3.49
94	1.98	2.26	2.02	2.37	2.84	2.50	2.33	2.56	2.84	3.25
95	1.87	2.18	1.96	2.24	2.66	2.37	2.19	2.41	2.67	3.03
96	1.78	2.07	1.93	2.12	2.51	2.25	2.08	2.26	2.51	2.84
97	1.68	1.94	1.87	2.01	2.38	2.14	1.97	2.14	2.36	2.68
98	1.60	1.86	1.79	1.96	2.27	2.03	1.83	2.05	2.23	2.53
99	1.52	1.77	1.74	1.94	2.17	1.93	1.70	1.92	2.12	2.38
100	1.45	1.66	1.66	1.87	2.00	1.83	1.65	1.78	2.02	2.21

12.16: Methodological Algebra

This appendix provides the algebraic representation of the methodology used in the thesis (summarised in Equation 3.2 and 8.1). Hence, this section provides the detailed outline of the quantitative willingness to pay: mortality and morbidity methodology. This methodology was outlined in Chapter 3 and applied in Chapters 8 and 9 of the thesis.

12.16.1 Contains the methodological algebra for improvements in the death rate, or mortality.

12.16.2 Contains the methodological algebra for considering improvements in the quality of life of additional years of life expectancy.

12.16.3 Contains the methodological algebra for the combination of improved mortality and the improved morbidity associated with these additional life years.

Sources:
Mortality gain algebra is derived from:

- Nordhaus, "The Health of Nations: The Contribution of Improved Health to Living Standards"

Morbidity gain algebra is derived from:

- Cutler & Richardson, "The Value of Health: 1970-1990"

12.16.1: Methodological Algebra: Mortality

This approach had been refined by Nordhaus to provide a method of measuring the gain in real income from improved life expectancy, in the context of the life-cycle model of consumption. This approach represents part of the Hickson QALE methodology.

An individual is assumed to value consumption and health according to a lifetime utility function:

$$V[c_t;\theta,\rho,\mu_t]=\int_{\theta}^{\infty}u(c_t)e^{-\rho(t-\theta)}S[\mu_t]dt \qquad (1)$$

Where $V[c_t;\theta,\rho,\mu_t]$ is the value at time *t* of the consumption stream, now and in the future, faced be an individual of age θ; (c_t) is the stream of instantaneous utility or felicity consumption; ρ is the pure rate of individual time preference; $S[\mu_t]$ is the set of survival probabilities; and μ_t is the set of mortality rates. The key assumption here is that utility is a function of the expected value of consumption weighted by the probability of survival. It is also assumed that the survival function is exponential, and therefore equation (1) becomes:

$$V[c_t;\theta,\rho,\mu_t]=\int_{\theta}^{\infty}u(c_t)e^{-(\rho+\mu)(t-\theta)}dt \qquad (2)$$

This equation can be further simplified by assuming that the real interest rate faced by the individual is equal to the mortality adjusted rate of time preference $(\rho+\mu)$. Given these assumptions, an individual will choose a consumption annuity that yields constant consumption during the individual's lifetime, $c_t = c^*$. Integrating equation (2) yields a simpler outcome:

$$V[c_t;\theta,\rho,\mu_t]=\frac{u(c^*)}{(\rho+\mu)} \qquad (3)$$

Equation (3) shows that the total utility value of consumption, discounted by a discount rate that equals the sum of the force of impatience and the force of mortality.

12.16.1: Methodological Algebra: Mortality

An individual will often face a trade-off between health and wealth. At age θ, changes in consumption and health yield:

$$\left.\begin{aligned} \frac{dV}{dc*} &= \frac{u'(c*)}{(\rho+\mu)} \\ \frac{dV}{d\mu} &= \frac{u(c*)}{(\rho+\mu)^2} \end{aligned}\right\} \quad (4)$$

Hence, the trade=off between consumption and mortality is:

$$\frac{dc*}{d\mu} = \frac{-u(c*)}{[u'(c*)(\rho+\mu)]} \quad (5)$$

It is then possible to further simplify through making two normalisations. First, utility is defined so that one unit of utility is one extra unit of the consumption good, by setting $u'(c*) = 1$. Second, the pure rate of time preference is set equal to zero, such that when the utility of consumption is $u(c) = 0$, the individual is indifferent between life and death. This implies that there is zero utility after death. Given these assumptions, equation (5) can be reduced to:

$$\frac{dc*}{d\mu} = \frac{-u(c*)}{(\rho+\mu)} \quad (6)$$

Or without discounting:

$$\frac{dc*}{d\mu} = Tu(c*) \quad (7)$$

Where, T is life expectancy ($T=1/\mu$). The interpretation here is that a uniform change in mortality rates at every age will produce a welfare change equal to the number of years of life (T) times the goods value of life, given by u(c*).

12.16.2: Methodological Algebra: Morbidity

In this methodology health capital is defined as:

$$L\sum_{k=0}^{\infty}\frac{E_t[H_{t+k}]}{(1+r)^k} \quad (8)$$

Where H_t represents a person's quality of life in any year (scaled on a 0 to 1 basis, where 0 = death and 1 = perfect health) or the QALY. L represents the value of a year in perfect health and *r* is the real discount rate and *k* is the number of years of life.

After identifying health capital the next stage in the methodology is to consider the quality of life. The starting point for this (more difficult) measurement is the probability that a person is alive or dead in each year of the future. This can be achieved through considering life expectancy and life tables. These survival rates then need to be adjusted for the prevalence of disease. Quality of life weights also need to be attached to every condition considered in the methodology and for the variance of the QALY over time. Hence, combining estimates of the share of the population who are still alive, at *t+k*, the prevalence of people with particular conditions, where *d* is the range of conditions a person could have, and the quality of life for people with those conditions, quality of life can be estimated as:

$$H_{t+k} = \Pr[\text{ alive at } t+k] * (\sum_{d} \Pr = [\text{condition } d \text{ at } t+k] * [\text{QALY for } d \text{ at } t+k]) \quad (9)$$

12.16.3: Methodological Algebra: Hickson QALE

The two above methodologies consider measuring the value of mortality and morbidity, the thesis QALE methodology considers the value of mortality and morbidity in a single methodology that combined the two above approaches to measuring life and health. The result of combining the two above approaches is a methodology that considers the value of improvements in life expectancy whilst considering the quality and subsequent value of these additional years from a health (or QALY) perspective. This QALE (quality adjusted life expectancy) methodology is expressed as:

$$QALE = \frac{dc*}{d\mu + \lambda} = \frac{-u(c*)}{(\rho + [\mu + \lambda])} \tag{10}$$

Where, $u(c*)$ = the goods value of life and $c*$ = consumption. μ represents the set of mortality rates and $\lambda = (\sum_{d}$ Pr Condition D at t + k] * [QALY for D at t + k]), which is essentially the consideration for the health aspect of improved mortality rates, where *d* represents the range of possible health conditions. ρ = the pure rate of individual time preference. Finally, it should be noted that,

$$\frac{dc*}{d\mu + \lambda} > 0$$

because individuals are likely to forego some consumption in return for improved healthy life years.

13. Appendix: CD-Rom